International Journal of Science and Research (IJSR)
ISSN: 2319-7064
ResearchGate Impact Factor (2018): 0.28 | SJIF (2019): 7.583

A Case Report of Takotsubo Cardiomyopathy in Pregnancy

Dr. Gaganpreet Kaur[1], Dr. Hafizur Rahman[2], Dr. S. Haque[3], Dr. Prerna Chettri[4]

[1, 2] Department of Obstetrics and Gynecology, Sikkim Manipal Institute of Medical Sciences, Gangtok, India

[3, 4]Department of Cardiology, Sikkim Manipal Institute of Medical Sciences, Gangtok, India

Abstract: **Background**: ***Takotsubo cardiomyopathy (TCM) occurs rarely in pregnancy. It leads to left ventricular dysfunction with apical ballooning. It may occur in women of childbearing age in the antepartum, intrapartum or postpartum period. With medical management there is resolution of cardiac dysfunction within weeks. Case presentation: A 26-year-old female in her second pregnancy presented at 38 weeks of gestation in early labor. She had a history of cesarean section 6 years ago and underwent cesarean section with bilateral tubal ligation. Postoperatively she developed palpitations with fluctuating heart rate and workup showed a stress induced cardiomyopathy. She had full recovery of cardiac function by 6 weeks postpartum after medical management. Conclusion: Antenatal women who present with cardiac symptoms should be evaluated for TCM cardiomyopathy after myocardial infarction has been ruled out. It is managed with a multidisciplinary approach. Cardiac dysfunction generally recovers within 4 weeks although some patients may need long term management of cardiac failure***

Keywords: Takotsubo cardiomyopathy, Preeclampsia, Pregnancy

1. Background

Takotsubo cardiomyopathy (TCM) also known as stress induced cardiomyopathy is defined as new onset left ventricular (LV) dysfunction with cardiac wall motion abnormalities without any significant coronary artery disease [1, 2, 3]. Cardiac dysfunction recovers spontaneously in most patients within days or weeks [4, 5]. The common triggers of TCM are emotional or psychological stressors. It occurs more frequently in patients with pre-existing psychiatric conditions [6]. However, no identifiable stressors may be found in 20% of patients. [1, 7]. In majority of cases, TCM in pregnancy presents in the peripartum period [8, 9, 10]. Hence, it is difficult to differentiate TCM from peripartum cardiomyopathy [5].In this article, we present a case of a 26-year-old female who underwent cesarean section and subsequently developed TCM during her postoperative stay. We further review the current literature on stress-induced cardiomyopathy in pregnancy.

2. Case Report

A 26-year-old Indian female, gravida 2 para 1, was admitted with early labor at 38 weeks of gestation after complaints of pain abdomen. She perceived normal fetal movements with no leaking or bleeding per vaginum. She had a pulse rate at 70 beats/minute, blood pressure of 120/82 mmHg, afebrile at 36.7°C. Her respiratory rate was 18 breaths per minute and peripheral oxygen saturation of 99% on room air. She was having 3 mild contractions every 10 minutes lasting for 20 sec each. There was no scar tenderness. On per vaginum examination, her bishops score was poor. She was not willing for a vaginal trial. On admission, her hemoglobin was 12.3 g/dl and the platelet count was 160,000/ml. The renal, liver functions and coagulation profile were normal. She was planned for caesarean section.

Six years back, she had a caesarean delivery at term for failed induction. The post-operative course was uneventful in her previous pregnancy. In the present pregnancy her initial antenatal period was uneventful. Her haemoglobin was 13.0 g/dl and all viral markers were negative. Her blood group was AB with positive Rhesus factor. She had 4 ultrasounds which showed normal growth with no fetal anomalies or abnormal doppler changes. She underwent emergency caesarean under spinal anesthesia. Her surgery lasted for 90minutes. There were adhesions between the urinary bladder and uterus, which were separated by sharp dissection. Previous scar site on the uterus was intact. A baby boy was delivered which cried immediately after birth. Uterus was closed in layers and bilateral tubal ligation was done. Peri-operatively her vitals were normal. Post operatively, she was given injectable antibiotics, analgesics, prokinetics. She received intravenous fluid at the rate of 100 ml/hour. After 6 hours of surgery, she complained of substernal chest pain and palpitation. She was conscious and oriented to time, place and person. Her pulse rate was 110-120 beats per minute. Her BP was 130/96 mm Hg. Her SpO2 on room air was 98%. Her respiratory rate was 26 per minute. An urgent ECG was done and cardiology opinion was sought. Electrocardiogram (ECG) revealed a sinus rhythm with tachycardia, ST inversion in leads I,II,III, aVF, V2-V6, suggestive of acute anterior wall ischemia. A transthoracic echocardiogram showed cardiac wall motion abnormalities of mid and distal interventricular septum, apex and anterior wall hypokinesia. The left ventricle (LV) was dilated with an ejection fraction of 42%. An assay of cardiac enzymes showed elevated Troponin I levels of 0.15 ug/L (reference range 0.01-0.023 ug/L) and Troponin T levels of 0.14ug/L (reference range 0.01-0.017ug/L).

Tab Ecospirin 75mg was added along with Metoprolol succinate extended release tablet 25mg once a day. The LMWH (low molecular weight heparin) was started 12 h post-surgery for one week. Left cardiac catheterization was subsequently done on day 3, approximately 18 h after the last dose of LMWH and showed left ventricular apical ballooning with no evidence of atherosclerosis in the

coronaries. Postpartum, she remained normotensive. She was discharged on day 7 of her surgery on metoprolol 25mg once a day. Tab Ecospirin and LMWH were stopped after 7 days of surgery. The echocardiogram at 6 weeks post-delivery revealed normal cardiac chambers with no regional wall motion abnormalities and a left ventricle ejection fraction of 65%.

3. Discussion

Takotsubo cardiomyopathy (TCM) is a rare life-threatening event that can affect pregnant women. It should be treated as a cardiac emergency. It is a recently recognised entity that mimics acute myocardial infarction. It causes profound, reversible left ventricular dysfunction and occurs after sudden emotional or psychological stress. This disorder mainly affects elderly female patients (mean age 67 years). It is a diagnosis of exclusion as acute myocardial infarction, peripartum cardiomyopathy, acute myocarditis, and dilated cardiomyopathy have a similar clinical picture and need to be ruled out . TCM typically presents with acute retrosternal chest pain, palpitations and diaphoresis mimicking myocardial infarction [5]. Affected patients may have symptoms of left heart failure like paroxysmal nocturnal dyspnea, orthopnea and dyspnea [1]. Electrocardiogram (ECG), cardiac biomarkers, echocardiography and left heart angiography are required in diagnostic evaluation of TCM. The ECG abnormalities include ST segment elevation in the anterior and precordial leads with some having ST segment depression [11, 12]. A few patients may have QT interval prolongation, T wave inversion and abnormal Q waves [11]. The majority of patients will have raised cardiac troponin levels with normal to mildly elevated creatinine kinase. The brain natriuretic peptide (BNP) may be elevated in up to 83% of cases suggesting ventricular strain but to a lesser extent than that seen in acute myocarditis [12]. Transthoracic echocardiography may reveal a large area of LV regional wall motion akinesia extending beyond the territory of a single coronary artery [13]. There is apical ballooning of the LV with normal basal contractile function. The LV ejection fraction may be reduced ranging from 20 to 49% [14]. There may be mitral regurgitation with or without a systolic anterior motion of the anterior leaflet [13].

A diagnostic score has been proposed to diagnose TCM. It has two major and three minor items [15]. Major items are reversible left ventricular apical ballooning and electrocardiographic changes mimicking acute myocardial syndrome. Minor items are emotional stress, moderate elevation of cardiac enzymes, and chest pain [16]. All major and minor items were present in our patient.

Despite the increasing awareness of TCM, the pathological mechanisms remain unclear. Some findings suggest an exaggeration of sympathetic system [17]. During the acute phase, plasma catecholamine levels are higher in TCM patients as compared to patients with acute myocardial infarction. Catecholamine overload may cause or worsen an intraventricular pressure gradient resulting in myocardial ischemia [18]. This pressure gradient reverses with β-antagonist treatment [18-20]. Hemodynamic support with noradrenaline infusion is debatable as Takotsubo syndrome is related to exaggerated sympathetic activity and noradrenaline infusion could worsen symptoms [21]. Reports of the onset of Takotsubo syndrome during peri-operative state is well documented as it is stressful phase [22-26]. Serious complications like left ventricle wall rupture, arrhythmias with repolarisation dispersion, long Q-T interval, torsades de pointe, conduction abnormalities with third degree atrioventricular block have been reported [27, 28, 29]. Fatal outcomes in patients with TCM have been observed during the initial phase of the disease [30]. However, the prognosis is good in patients who survive the initial heart failure without complication, and recurrence is unusual [30, 31, 32, 33].The pathogenesis, prevalence and best therapeutic approach of Takotsubo syndrome remains unclear but β-antagonists are the treatment of choice during the acute phase. Specialised investigations in a coronary care unit are needed to diagnose and manage Takotsubo cardiomyopathy.

Most patients recover with normal cardiac function within 4 to 8 weeks [5, 12]. Our patient had resolution of heart failure symptoms with full recovery by 6 weeks post-delivery. These patients need symptomatic treatment and cardiac optimization with diuretics, angiotensin-converting enzyme inhibitors and beta blockers. As beta-blockers are associated with fetal growth restriction, there is need to schedule ultrasounds for fetal growth every 4 weeks.

The decision on the timing and mode of delivery should be guided by obstetrical indication. It should involve a multidisciplinary team of cardiologists, obstetricians, neonatologists and psychologists.

Recurrence of TCM in premenopausal women is very rare [34]. Close follow up upon discharge is needed as some patients may deteriorate and develop adverse cardiac and cerebrovascular events [1].

4. Conclusion

The pathogenesis and prevalence of Takotsubo syndrome remain elusive, despite increasing awareness and documented reports. The best therapeutic modality has yet to be established, but β-blockers are the drug of choice during the acute phase. There should be an index of suspicion when a patient presents with acute onset of chest pain, electrocardiographic changes mimicking acute myocardial infarction, acute heart failure or ventricular arrhythmias during the induction of general anaesthesia, and more frequently during the whole peri-operative period. Management in a coronary care unit is essential.

5. Abbreviations

ECG: Electrocardiogram
LMWH: Low Molecular Weight Heparin
LV: Left ventricle
LVOT: Left Ventricular Outflow Tract
TCM: Takotsubo cardiomyopathy

6. Future Scope

By analysing more number of cases of TCM in pregnancy, there will be a better understanding of clinical features and management. The rarity of the disease limits this prospect. More data is needed to understand this entity thoroughly.

References

1)

[1] Templin C, Ghadri JR, Diekmann J, Napp LC, Bataiosu DR, Jaguszewski M, et al. Clinical features and outcomes of Takotsubo (stress) cardiomyopathy. N Engl J Med. 2015;373(10):929–38.Article Google

[2] Minatoguchi M, Itakura A, Takagi E, Nishibayashi M, Kikuchi M, Ishihara O. Takotsubo cardiomyopathy after cesarean: a case report and published work review of pregnancy-related cases. J ObstetGynaecol Res. 2014;40(6):1534–9.View ArticleGoogle Scholar

[3] Salmoirago-Blotcher E, Dunsiger S, Swales HH, Aurigemma GP, Ockene I, Rosman L, et al. Reproductive history of women with Takotsubo cardiomyopathy. Am J Cardiol. 2016;118(12):1922–8.View ArticleGoogle Scholar

[4] Pelliccia F, Kaski JC, Crea F, Camici PG. Pathophysiology of Takotsubo syndrome. Circulation. 2017;135(24):2426–41.View ArticleGoogle Scholar

[5] Yaqub Y, Jenkins LA, Nugent KM, Chokesuwattanaskul W. Postpartum depression and apical ballooning syndrome (takotsubo syndrome). J ObstetGynaecol Can. 2009;31(8):736–9.View ArticleGoogle Scholar

[6] Nayeri A, Rafla-Yuan E, Farber-Eger E, Blair M, Ziaeian B, Cadeiras M, et al. Pre-existing psychiatric illness is associated with increased risk of recurrent Takotsubo cardiomyopathy. Psychosomatics. 2017.Google Scholar

[7] Corrigan FE 3rd, Kimmel MC, Jayaram G. Four cases of takotsubo cardiomyopathy linked with exacerbations of psychiatric illness. InnovClinNeurosci. 2011;8(7):50–3.PubMedPubMed CentralGoogle Scholar

[8] Virani SS, Khan AN, Mendoza CE, Ferreira AC, de Marchena E. Takotsubo cardiomyopathy, or broken-heart syndrome. Tex Heart Inst J. 2007;34(1):76–9.PubMedPubMed CentralGoogle Scholar

[9] Ruiz S, Martinez-Marin M, Luque P, Nassar N, Oros D. Takotsubo cardiomyopathy after cesarean section: a case report and literature review. J ObstetGynaecol Res. 2017;43(2):392–6.View ArticleGoogle Scholar

[10] Suzuki T, Nemoto C, Ikegami Y, Yokokawa T, Tsukada Y, Abe Y, et al. Development of takotsubo cardiomyopathy with severe pulmonary edema before a cesarean section. J Anesth. 2014;28(1):121–4.View ArticleGoogle Scholar

[11] Otani Y, Tokunaga K, Kawauchi S, Inoue S, Watanabe K, Kiriyama H, et al. Cerebral infarction arising from Takotsubo cardiomyopathy: case report and literature review. NMC Case Rep J. 2016;3(4):119–23.

[12] Rozema T, Klein LR. Takotsubo cardiomyopathy: a case report and literature review. Cardiol Young. 2016;26(2):406–9.

[13] Gupta S, Gupta MM. Takotsubo syndrome. Indian Heart J. 2018;70(1):165–74.

[14] Roshanzamir S, Showkathali R. Takotsubo cardiomyopathy a short review. Curr Cardiol Rev. 2013;9(3):191–6.

[15] Abe Y, Kondo M. Apical ballooning of the left ventricule: a distinct entity? Heart 2003; 89: 974– 6.

[16] Lipiecki J, Durel N, Decalf V, et al. Ballonisation apicale transitoire du ventricule gauche ou syndrome du tako-tsubo. A propos de 10 nouveaux cas. Archives des Maladies du Coeur et des Vaisseaux 2005; 98: 275– 80.

[17] Wittstein IS, Thieman DR, Lima JA, et al. Neurohumoral features of myocardial stunning due to sudden emotional stress. New England Journal of Medicine 2005; 352: 539– 48

[18] Villareal RP, Achari A, Wiansky S, et al. Anteroapical stunning and left ventricular outflow obstruction. Mayo Clinic Proceedings 2001; 76: 79– 83.

[19] Kyuma M, Tsuchihashi K, Shinshi Y, et al. Effect of intravenous propanolol on left ventricular apical ballooning without coronary artery stenosis (ampulla cardiomyopathy): three cases. Circulation Journal 2002; 66: 1181– 4.

[20] Penas-Lado M, Barriales-Villa R, Goicolea J. Transient left ventricular apical ballooning and outflow tract obstruction. Journal of the American College of Cardiology 2003; 42: 1143– 4

[21] Ohtsuka T, Hamada M, Kodama K, et al. Neurogenic stunned myocardium. Circulation 2000; 101: 2122– 4.

[22] Takigawa T, Tokioka H, Chikai T, et al. A case of undiagnosed 'takotsubo' cardiomyopathy during anesthesia. Masui 2003; 52: 1104– 6.

[23] Takayama N, Iwase Y, Ohtsu S, et al. 'Takotsubo' cardiomyopathy developed in the postoperative period in a patient with amyotrophic lateral sclerosis. Masui 2004; 53: 403– 6.

[24] Mizutani K, Okada M. A case of intraoperative repeated coronary artery spasm with ST-depression. Masui 2002; 51: 1114– 6.

[25] Jensen JB, Malouf JF. Takotsubo cardiomyopathy following cholecystectomy: a poorly recognized cause of acute reversible left ventricular dysfunction. International Journal of Cardiology 2006; 106: 390– 1.

[26] Gavish D, Rozenman Y, Hafner R, et al. Takotsubo cardiomyopathy after general anesthesia for eye surgery. Anesthesiology 2006; 105: 621– 3.

[27] Denney SD, Lakkireddy DR, Khan IA. Long QT syndrome and torsade de pointes in transient left ventricular apical ballooning syndrome. International Journal of Cardiology 2005; 100: 499– 501

[28] Marboeuf P, Ennezat PV, Gonin X, et al. Syndrome de tako-tsubo et obstruction intraventriculaire gauche: mécanisme de pérennisation? A propos d'un cas. Archives des Maladies du Coeur et des Vaisseaux 2006; 99: 69– 72.

[29] Akashi YJ, Tejima T, Sakurada H, et al. Left ventricular rupture associated with Takotsubo cardiomyopathy. Mayo Clinic Proceedings 2004; 79: 821– 4.

[30] Tsuchihashi K, Ueshima K, Uchida T, et al. Transient left ventricular apical ballooning without coronary artery stenosis: a novel heart syndrome mimicking acute myocardial infarction. Journal of the American College of Cardiology 2001; 38: 11– 8.

[31] Lentschener C, Vignaux O, Spaulding C, et al. Early postoperative tako-tsubo-like left ventricular dysfunction: transient left ventricular apical ballooning syndrome. Anesthesia and Analgesia 2006; 103: 580–2.
[32] Bybee KA, Prasad A, Barsness GW, et al. Clinical characteristics and thrombolysis in myocardial infarction frame counts in women with transient left ventricular apical ballooning syndrome. American Journal of Cardiology 2004; 94: 343– 6
[33] Akashi YJ, Musha H, Kida K, et al. Reversible ventricular dysfunction takotsubo cardiomyopathy. European Journal of Heart Failure 2005; 7: 1171– 6.
[34] Hefner J, Csef H, Frantz S, Glatter N, Warrings B. Recurrent Tako-Tsubo cardiomyopathy (TCM) in a pre-menopausal woman: late sequelae of a traumatic event? BMC Cardiovasc Disord. 2015;15:3.