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Case Report

Acute onset of left heart failure immediately following cesarean delivery: A peripartum cardiomyopathy case presentation

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Preface

Peripartum cardiomyopathy (PPCM) is a rare disease of unknown cause in which a left ventricle dysfunction occurs during the last trimester of pregnancy and the first five months postpartum, and it, reported to be 1 over 1300 to 1 over 4000 of life births [1]. PPCM is known to be one of the most common causes of severe complications in pregnancy [2]. It may occur in childbearing women of any age but it is commonly after the age of thirty. The clinical course of PPCM is variable, from rapidly progressive to end stage heart failure to complete recovery of the left ventricle function. The most frequent initial presentation are symptoms appropriate with the New York Heart Association (NYHA) functional class 3 or 4 as marked limitation of physical activity and unable to carry on any physical activity without discomfort and symptoms of heart failure at rest, or may present with sever ventricular arrhythmia [3]. Risk factors for the development of PPCM are multiparicity, preeclampsia and elderly pregnancy. Recently, there are advances in understanding the pathophysiology of PPCM, a role of unbalanced oxidative stress and generation of a cardio toxic prolactin sub fragment is suspected [4]. A high index of suspicion is required for the diagnosis as, short of breathing and ankle swellings are common in the peripartum period. In this study we present a case report where a primigravida bearing twins developed sever left ventricle failure and pulmonary edema due to PPCM suddenly and immediately after delivery.

Case presentation

Forty two years old primigravida bearing twins (37 weeks plus 4 days) was admitted to our institute for elective cesarean delivery. Her past history was negative for chronic illness and no history of allergy to any medication and previous anesthesia except, at 24 weeks of pregnancy she has been admitted to the hospital because of right flank pain due to a right hydronephrosis. She was admitted to the high risk pregnancy unit for treatment and observation. In the recent admission her physical examination revealed a blood pressure of 156/97; heart rate of 101/min, body temperature of 36.6 C and body weight was 95 Kg, ankle swilling and facial edema. Due to the elevated blood pressure, a preeclampsia was suspected and a full profile of blood and urine analysis was carried out, the blood numbers were normal including renal and liver function, Hemoglobin of 10gr/dl and the PT- INR of 0.96 and an APTT of 24.8 seconds and normal urinalysis. The patient arrived to the operating room the, day after admission for the planned cesarean section without any sign of distress. Standard monitoring of ECG, Non Invasive Blood Pressure and Pulse Oximetry were applied, the blood pressure was high as 152/90 mm Hg, heart rate of 106/min and O_2 saturation of 97% on room air. In sitting position and after draping the skin of her back with Chlorhexidine 0.5%, spinal anesthesia was conducted at the level of L3-L4 vertebral space using, a 27G pencil point needle with hyperbaric Bupivacaine 0.5% 10 mg plus fentanyl 0.02 mg. Surgery started 10 minutes after completing the anesthesia. The blood pressure dropped to 124/72 mm Hg and, remained within the normal range in the subsequent time till the end of the cesarean section. After lying on bed the parturient started coughing and told us that she has episodes of cough in the last month with no fever and she didn't visit the family physician. During the operation, blood pressure, heart rate and O_2 saturation all remained within the normal range. On arrival to the Post Anesthesia Care Unite (PACU) the patient's vital signs were B.P 108/70 mm Hg, heart rate 101/min and O_2 saturation 97% on an oxygen mask. Twenty minutes later the parturient started suffering from dyspnea and, desaturation of lower than 90% on FiO_2 of 0.4. A reservoir mask was connected without any improvement, the patient situation worsened; became dyspneic with rapid and shallow breaths. Auscultation revealed, diminished air entrance in the bases of both lungs and signs of pulmonary congestion. Manual ventilation by self-inflating bag started and endotracheal intubation done, a profuse fluid came out through the tracheal tube. A single dose intravenous furosemide 40 mg and, sedation with Morphine 10 mg started. A chest x-ray revealing pulmonary edema, and electrocardiogram show up, sinus tachycardia without signs of myocardial ischemia. Transthoracic Echocardiography (TTE) found global left ventricle hypo kinesis with Ejection Fraction (EF) of 40% and left ventricle diameter of 4.3 cm, normal right ventricle contraction and mild mitral and tricuspid regurgitation. The patient was transferred to the general intensive care unit mechanically ventilated under sedation with FiO2 of 0.8. Treatment with continuous intravenous furosemide, Angiotensin Converting Enzyme Inhibitor (ACEI) captopril and, beta sympathetic agonist blocker started according to the cardiologist consultation. In the second day after, an elevation of the Troponin T appeared and, revision of the TTE by the cardiologists revealed an overestimation of the

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ejection fraction the day before, then possible ischemia in the territory of the left anterior descending coronary artery was suspected, coronary angiography was done the same day and revealed normal coronaries. On the third day new TTE done and it was better than the previous one with an estimated EF OF 45%. A chest X-ray was taken showing decreased lung congestion. On the fourth day, after being supported for 72 hours, the patient was taken off the mechanical ventilation and was transferred to the ward in the fourth day, with recommendations to continue treatment with beta agonist blocker and ACEI. By the seventh day patient and the new-borne twins were discharged back home, with a recommendations to be in close follow up with the cardiology clinic.

Discussion

The peripartum cardiomyopathy affect the gravid woman at any age, it manifested with symptoms of heart failure, secondary to left ventricle systolic dysfunction toward the end of pregnancy as well as during the early period after delivery. The exact cause of the disease is unknown, but the common causes of left ventricle failure should be excluded. Many symptoms of cardiac disease occur during normal pregnancy as, dyspnea, dizziness, orthopnea and decreased exercise capacity. Early and rapid diagnosis needs a high index of suspicion. Our patient is from a rural area and began coughing two weeks before the scheduled cesarean section but yet she did not visit her family physician to address the symptoms. We highly suspect that the disease began in the third trimester of pregnancy and the patient became symptomatic while lying on her back or at nighttime. Many PPCM patients present with heart failure without any previous signs or symptoms to alert the clinicians that cardiomyopathy is going to develop. In 19% of the cases the symptoms may present in the last gestational month [5]. Peripheral edema may occur in one third of healthy gravid women as demonstrated in the present case, whereby during the admission process the physical examination did not show any prominent sign of heart failure in spite the chest auscultation. In our case the sudden onset of pulmonary edema and acute hypoxic respiratory failure required immediate intervention with tracheal intubation and positive ventilation, to ensure the adequate oxygen content and delivery to the tissues. The PPCM with no fulminant onset may take more than 7 days to determine the diagnosis [6]. Laboratory studies of the case showed an increase of the Creatine Phosphokinase (CPK), Troponin T and C - reactive protein (CRP). The CPK can be elevated after normal or cesarean delivery, especially from the uterus and skeletal muscles [7]. Troponin T elevation was temporary and returned to normal range within three days, while the coronary angiography was negative to myocardial ischemia. The CRP elevated to maximum level on the third day after delivery. Investigation on large cohort of PPCM, patients demonstrated that this cardiomyopathy is associated with a pro-inflammatory response, as evident with elevated plasma level of CRP, Interleukin-6 and tumor necrosis factor [8,9]. Our patient has been treated with the routine and conventional treatment of heart failure and pulmonary edema, with beta receptor antagonist Bisoprolol, angiotensin converting enzyme inhibitor captopril and furosemide with a clear improvement in the cardiovascular and respiratory function as mentioned before. Several case reports of PPCM have showed that the addition of the Dopamine agonist Bromocriptin to the standard therapy may be beneficial in these cases [10]. Bromocriptin has been used in postpartum to stop lactation; however this has been associated with several reports of myocardial infarction [11]. Anticoagulation therapy is strongly recommended in PPCM patient receiving Bromocriptin but before, recommending this as routine therapy, a further large studies need to be conducted.

The presented case describes an undiagnosed PPCM that occurred during the third trimester of gestation and the clinical presentation as pulmonary edema and acute left ventricle failure appeared in the day of delivery. On a practical level, we conclude that further strict education of the gravid women should be conducted in the rural area of the developing countries. We as anesthesiologists should be aware to detect minimal signs of cardiovascular symptoms during pregnancy and peripartum period by focusing in the perioperative patient assessment particularly anamnesis.

References

1. Yancy CW, Jessup M, Bozkurt B (2013) ACCF/AHA guidelines for the management of heart failure: a report of the American College of Cardiology Foundation/American Heart Association Task Force in Practice Guidelines. *J Am Coll Cardiology* 62: 147-293.
2. Reimold SC, Rutherford JD (2001) Peripartum cardiomyopathy. *N Engl J Med* 344: 1629-1630. [Crossref]
3. Diao M, Diop IB, Kane A, Camara S, Kane A, et al. (2004) [Electrocardiographic recording of long duration (Holter) of 24 hours during idiopathic cardiomyopathy of the peripartum]. *Arch Mal Coeur Vaiss* 97: 25-30. [Crossref]
4. Selle T, Renger I, Labidi S, Bultmann I, Hilfiker-Kleiner D (2009) Reviewing peripartum cardiomyopathy: current state of knowledge. *Future Cardiol* 5: 175-189. [Crossref]
5. Elkayam U, Akhter MW, Singh H, Khan S, Bitar F et al. (2005) Pregnancy- associated Cardiomyopathy: Clinical characteristics and a comparison between early and late presentation. *Circulation* 111: 2050-2055. [Crossref]
6. Goland S, Modi K, Bitar F, Janmohamed M, Mirocha JM, et al. (2009) Clinical profile and predictors of complications in peripartum cardiomyopathy. *J Card Fail* 15: 645-650.[Crossref]
7. Chemnitz G, Nevermann L, Schmidt E, Schmidt FW, Lobers J (1979) Creatine kinase (EC-No.2.7.3.2) and creatine kinase isoenzymes during pregnancy and labor and in the cord blood. *Clin Biochem* 12: 277-281. [Crossref]
8. Walenta K, Schwarz V, Schirmer SH, Kindermann I, Friedrich EB, et al. (2012) Circulating microparticles as indicators of peripartum cardiomyopathy. *Eur Heart* J 33: 1469-1479.[Crossref]
9. Hilfiker-Kleiner D, Struman I, Hoch M, Podewski E, Sliwa K (2012) 16-kDa prolactin and bromocriptine in postpartum cardiomyopathy. *Curr Heart Fail Rep* 9: 174-182. [Crossref]
10. Habedank D, Kühnle Y, Elgeti T, Dudenhausen JW, Haverkamp W, et al. (2008) Recovery from peripartum cardiomyopathy after treatment with bromocriptine. *Eur J Heart Fail* 10: 1149-1151. [Crossref]
11. Hopp L, Haider B, Iffy L (1996) Myocardial infarction postpartum in patients taking bromocriptine for the prevention of breast engorgement. *Int J Cardiol* 57: 227-232. [Crossref]