Undiagnosed peripartum cardiomyopathy: Anesthesiologist's nightmare!

Madam,

Peripartum cardiomyopathy (PPCM) is a rare heart condition with high mortality rate and poses a challenge to the anesthesiologist.[1,2] Symptoms like breathlessness, chest discomfort, and pedal edema can present in the late antenatal period in any parturient making the diagnosis of PPCM difficult. A 37 years' normal patient G2P2L1, with history of previous cesarean (CS), was planned for CS for breech presentation. Her previous pregnancy and antenatal period of this pregnancy were uneventful. Subarachnoid block (SAB) was administered at L3-4 level (0.5% hyperbaric, bupivacaine 10 mg, and fentanyl 25 mcg). Five minutes following SAB, the patient became drowsy, developed bradycardia (HR-32/min), and unrecordable noninvasive blood pressures. Atropine 0.6 mg was administered twice followed by 1 mg adrenaline for persistent bradycardia. Though the hemodynamics stabilized (HR-110/min, BP-140/80 mmHg), the patient remained drowsy; hence, general anesthesia (GA) was administered and a healthy female child was delivered within 10 minutes of intubation. Further intraoperative course was uneventful and the patient was extubated at the end of surgery.

Although she appeared well immediately after surgery, 10 hours later she developed breathlessness with desaturation (SpO_2- 90%) and bilateral lung crepitations. Postoperatively she had received 1.5 L of crystalloids with 10 units' oxytocin in each 500 ml. A presumptive diagnosis of pulmonary edema was done and morphine 6 mg and furosemide 40 mg were administered. Noninvasive ventilation with continuous positive airway pressure 10 cm H_2O was instituted with improvement in saturations to 98%. Electrocardiography (ECG) showed sinus tachycardia and echocardiograph revealed severe left ventricular (LV) dysfunction (ejection fraction: 35%), global hypokinesia, mild mitral regurgitation, and LV hypertrophy. Potential causes for pulmonary edema were stress (typical or variant type) cardiomyopathy and PPCM. As the cardiac enzymes were normal and ECG did not show new ST/T changes, diagnosis was narrowed to PPCM and treatment with tablet ramipril 2.5 mg BD and furosemide 20 mg BD was started. She responded well to treatment and was discharged on fifth postoperative day.

A diagnosis of PPCM can be done in the context of development of cardiac failure in the last month of pregnancy or within 5 months postpartum, absence of identifiable cause of cardiac failure, absence of heart disease, and presence of LV systolic dysfunction on ECH.[1] Etiology of PPCM includes myocarditis, abnormal immune response to pregnancy, maladaptive response to the hemodynamic stresses of pregnancy, stress-activated cytokines, and prolonged tocolysis.[2] Clinical spectrum of stress (typical/atypical) and PPCM are overlapping; however, to differentiate between them biomarkers like modified prolactin and CRP can be used. It was differentiated from stress-induced Takotsubo cardiomyopathy by absence of LV apical ballooning and ST/T changes and normal cardiac enzymes.[3] Atypical stress cardiomyopathy can be ruled out by doing CRP, which was raised in our patient.

There is a delay in the diagnosis of PPCM due to the overlap of symptoms and signs of normal late pregnancy to heart failure. Quantification of the severity of symptoms (orthopnea, dyspnea, unexplained cough, lower extremity swelling, excessive weight gain during last month of pregnancy, palpitations) has been validated on a metric score by Fett JD with each symptom given a score from 0 to 2. A score greater than 4 points mandates need of additional investigations like ECG, B-type natriuretic peptide, thus aiding in early diagnosis.[4] Retrospectively, the index patient had a score of 5. We could have done early detection of impending heart failure using this score presurgery. In our case, SAB may have led to hemodynamic compromise.[5] There is no consensus about the choice of anesthesia for CS in PPCM patient. Both GA and regional anesthesia (RA) have been used.[6] GA allows control of ventilation and easier management of pulmonary edema. Combined spinal epidural can be used, in which low-dose spinal provides dense blockade and epidural can be utilized to extend the block if required. SAB should be avoided as it causes intense sympathetic blockade leading to cardiac compromise. Recently, single shot low-dose spinal has been successfully used in PPCM patients.[7]

Early detection of PPCM by judging severity of symptoms using validated scale by Fett JD can aid in early diagnosis.[4] A high index of suspicion for PPCM is required in any pregnant patient having increased breathlessness toward end of pregnancy/postpartum or presenting with intraoperative severe cardiovascular collapse and clinical features of heart failure in the postoperative period. ECG is advisable in such patients, as PPCM has grave prognosis with high chances of recurrence in subsequent pregnancies.[8]

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

Anuradha Borle, Devalina Goswami[1], Tanvi Meshram[1], Manpreet Kaur[1], Shiv Akshat[1]

Department of Anesthesiology, Washington University School of Medicine, St Louis, MO, USA, [1]Department of Anaesthesiology, Pain Medicine and Critical Care, AIIMS, New Delhi, India

Address for correspondence: Dr. Anuradha Borle, Department of Anesthesiology, Washington University School of Medicine, St Louis, MO, USA.
E-mail: borle.a@wustl.edu

References

1. Ramachandran R, Rewari V, Trikha A. Anaesthetic management of patients with peripartum cardiomyopathy. J Obst Anaesth Crit Care 2011;1:5.
2. Pearson GD, Veille JC, Rahimtoola S, Hsia J, Oakley CM, Hosenpud JD, *et al*. Peripartum cardiomyopathy: National Heart, Lung, and Blood Institute and Office of Rare Diseases (National Institutes of Health) workshop recommendations and review. JAMA 2000;283:1183-8.
3. Kurowski V, Kaiser A, von Hof K, Killermann DP, Mayer B, Hartmann F, *et al*. Apical and midventricular transient left ventricular dysfunction syndrome (tako-tsubo cardiomyopathy): frequency, mechanisms, and prognosis. Chest 2007;132:809-16.
4. Fett JD. Validation of a self-test for early diagnosis of heart failure in peripartum cardiomyopathy. Crit Pathw Cardiol 2011;10:44-5.
5. Bajwa SK, Bajwa SJ, Sood A. Cardiac arrest in a case of undiagnosed dilated cardiomyopathy patient presenting for emergency cesarean section. Anesth Essays Res 2010;4:115-8.
6. Sumikura H, Hamada T, Idei M. [Anesthetic management of parturients with peripartum cardiomyopathy]. Masui 2014;63:31-8.
7. Gupta K, Gupta SP, Jose S, Balachander H. Low dose spinal anesthesia for peripartum cardiomyopathy. J Anaesth Clin Pharmacol 2011;27:567-8.
8. Sliwa K, Hilfiker-Kleiner D, Petrie MC, Mebazaa A, Pieske B, Buchmann E. Current state of knowledge on aetiology, diagnosis,

management, and therapy of peripartum cardiomyopathy: A position statement from the Heart Failure Association of the European Society of Cardiology Working Group on peripartum cardiomyopathy. Eur J Heart Fail 2010;12:767-78.

Access this article online

Quick Response Code:

Website: www.joacp.org

DOI: 10.4103/joacp.JOACP_221_17

How to cite this article: Borle A, Goswami D, Meshram T, Kaur M, Akshat S. Undiagnosed peripartum cardiomyopathy: Anesthesiologist's nightmare! J Anaesthesiol Clin Pharmacol 2019;35:566-8.