A case of peripartum cardiomyopathy in acute heart failure for emergency surgery

Sir,

Peripartum cardiomyopathy (PPCM) remains a nightmare to anaesthesiologists. Here, we report a case of PPCM in acute heart failure posted for emergency lower segment caesarean section.

A thirty eight year old, morbidly obese multipara with 35 weeks gestation and gestational hypertension, presented with sudden onset of breathlessness. She did not have any significant past medical history. On examination, she was tachypnoeic, tachycardiac, hypertensive, and hypoxaemic. Auscultation revealed bilateral crepitations. She was started on bilevel positive airway pressure (BiPAP) support. Routine laboratory parameters were within normal limits. Electrocardiogram showed sinus tachycardia. Echocardiogram revealed global left ventricular hypokinesia, moderate left ventricle dysfunction, and an ejection fraction of 35%.

Frusemide and nitroglycerine infusions were started. After cardiology consultation, a provisional diagnosis of peripartum cardiomyopathy was made. Emergency termination of pregnancy was decided. Under high-risk informed consent, she was taken up for anaesthesia. Our anaesthetic plan was general anaesthesia with modified rapid sequence induction. She was positioned with pillows beneath her upper chest in a ramped fashion in view of the anticipated difficult intubation. Under local anaesthesia, an arterial line and large bore venous access were placed. Careful administration of fluids was done. Patient was induced with titrated boluses of thiopentone and intubated with succinylcholine. BiPAP was maintained till the time of laryngoscopy. To suppress the haemodynamic response of laryngoscopy, Inj. metoprolol 3 mg was given. Even though intubation was completed in 15 seconds, patient desaturated to a peripheral oxygen saturation (SpO_2) level of 55% which improved on positive pressure ventilation. Baby was delivered in less than 5 minutes. Early cord clamping was done to limit autotransfusion. Inj. oxytocin 20 IU was added to 500 mL normal saline and administered slowly. Analgesia was supplemented with fentanyl and paracetamol. Intraoperatively, haemodynamics remained stable. Patient was continued on frusemide and nitroglycerine infusion and shifted on ventilator support. She was extubated 6 hours later, but her cardiac function deteriorated and she was started on dobutamine support and restarted on BiPAP. Patient was shifted out of intensive care unit on fourth postoperative day and discharged on tenth postoperative day.

The revised criteria for diagnosis include heart failure, secondary to left ventricular systolic dysfunction with a left ventricular ejection fraction (LVEF) <45%, occurrence towards the end of pregnancy or in the months following delivery and no other identifiable cause of heart failure. Majority are diagnosed in the first month after delivery. Its pathophysiology is still incompletely understood and is thought to be involving angiogenic, metabolic, hormonal, and oxidative stress factors. Pre-eclampsia and hypertension are strongly associated with PPCM.[1] The symptoms mimic those of normal pregnancy and the diagnosis is often missed. It is a diagnosis of exclusion. Levels of brain natriuretic peptide (BNP) and N-terminal pro-BNP are significantly elevated. Management is based on the principles of management of systolic heart failure due to other causes with caution taken to avoid drugs which cause foetal harm. Anticoagulation is recommended for all PPCM patients with an LVEF <35%.[2]

The maternal outcome is variable with death occurring in 1.4% to 28%.[3] Venous thromboembolism and arrhythmias are common complications. These patients are at a risk of sudden cardiac death even after recovery. Ejection fraction at presentation best predicts recovery and ejection fraction <30% is associated with lower rates of recovery.[3]

Both general and regional anaesthesia have been used in the management of these patients.[4,5] Our anaesthetic goals should be to maintain cardiac contractility and preload and prevent increase in afterload. Volume overload should be closely watched for and prevented. Neuraxial anaesthesia has the advantage of reducing afterload and avoiding the sympathetic response to intubation. Since our patient was in acute heart failure and unable to lie supine, we decided to administer general anaesthesia. Oxytocin can reduce and methergine can increase afterload significantly.[5] Postoperatively these patients need intensive care monitoring.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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