CLINICAL VIGNETTE

Peripartum Cardiomyopathy with Hypoxic Respiratory Failure in a Patient with Cesarean Delivery

Peter Drocton, MD and Sachin Gupta, MD

A 29-year-old G4P0303 female at 36 weeks and 4 days gestation was seen in the Emergency Department for acute dyspnea, subjective fever, and abdominal cramping for 2 days. She noted that all family members in the household had tested positive for the SARS-CoV-2 virus approximately one month prior, though she tested negative at the time. She had tried over-the-counter cold remedies without relief and sought medical care. Past medical history was notable for preeclampsia with seizures during three previous pregnancies, with preterm delivery via cesarean section each time. She reported alcohol and methamphetamine use during the current pregnancy. Initially, she reported last use was 6-8 weeks prior to admission, though upon further questioning admitted to use in the last seven days. She reported minimal prenatal care, stating that she only discovered she was pregnant five weeks prior to onset of current symptoms.

On physical exam, she was in mild distress with a normal temperature of 36.8°C, heart rate in the 140s, blood pressure 141/106 and respiratory rate progressively increasing into the upper 20-30/min. Room air oxygen saturation was 86%, which improved to 95% on 10L nonrebreather face mask. She was transitioned to BiPAP and then CPAP. EKG showed sinus tachycardia at 134 and troponin-1 was elevated to 0.057 times three, and BNP 455pg/mL. Additional labs included WBC 16K/cumm; platelets 461K/cumm, Hgb 10.5 g/dL, mildly elevated potassium; and urine toxicology screen positive for methamphetamines. Chest x-ray showed evidence of volume overload and bilateral pleural effusions, while a CT angiography chest was negative for pulmonary embolism. SARS-CoV-2 PCR testing was negative. She was initially admitted to the monitored unit but was later transferred to the ICU. The following morning, echocardiogram showed severely reduced left ventricular systolic function with an EF 15-20%; significant regional wall motion abnormalities with diffuse hypokinesis; and estimated PAP 61mmHg.

The differential diagnosis for the patient's new-onset acute hypoxic respiratory failure with decompensated heart failure included peripartum cardiomyopathy exacerbated by pre-eclampsia, current methamphetamine use, and possible viral coinfection, or more likely, some combination of the above. Cardiology, pulmonology, anesthesiology, and maternal-fetal medicine services were all consulted to assist in developing a care plan. Diuresis with furosemide was begun and a nitroglycerin infusion was titrated to manage blood pressure. The patient's oxygen requirement progressively improved, and she was transitioned to nasal canula at 3L with SpO2 99%. Of note, there were no signs of fetal distress beyond mild tachycardia. Given history of pre-eclampsia, maternal hypertension and prior cesarean section in the setting of maternal cardiopulmonary compromise indicated imminent delivery.

Delivery of the fetus via repeat cesarean section was performed on hospital day 3. Magnesium sulfate had been started on hospital day 2 and was continued through the perioperative period for neuroprotection in the setting of preeclampsia. A pulmonary artery catheter (PAC) was placed before surgery showed elevated right ventricle pressures and a PCWP 20mm Hg. A radial arterial line was also placed. Epidural anesthesia was initiated without complications, and epinephrine 2%-lidocaine 1:200,000 (28cc total) was dosed to achieve bilateral T4 block level. Hemodynamic parameters initially remained unchanged. Nitroglycerin had been weaned off prior to surgery, and as the sympathectomy from the epidural progressed, a norepinephrine drip was instituted and titrated to maintain MAP 65mmHg. Occasional bolus doses of phenylephrine were also given for blood pressure support. Repeat cesarean section was performed without complication, and the patient tolerated all phases of the surgery well, from delivery of the neonate and placenta through to skin closure. Oxytocin was infused at approximately 25U per hour, and carboprost 250mcg IM was given once. Estimated blood loss was approximately 700cc, with 1700cc of crystalloid given during the case. She was maintained on oxygen via face mask at 6L/min during and immediately after surgery, and maternal vital signs remained otherwise stable. Several hours post-delivery, the patient complained of shortness of breath and was temporarily restarted on CPAP. She was again given furosemide and bumetanide, with approximately 2L of fluid diuresed over the next 16 hours. On hospital day 4, her PAC showed mildly elevated right and left filling pressures as well as pulmonary hypertension and the patient was further diuresed 2L. The patient was gradually transitioned to room air over the next two days, with oral medications initiated for blood pressure control. She continued to improve and was discharged on hospital day 9.

Discussion

The physiologic changes that occur during pregnancy and after delivery can result in clinical decompensation of parturients with cardiomyopathies who may have very limited cardiac reserves. This patient had many factors contributing to her cardiomyopathy, including her pregnancy, recent and continued methamphetamine use, preeclampsia, and the possibility of residual effects related to Covid-19 disease, though diagnostic testing could not conclusively prove this to be a factor. Prior to delivery, the increased metabolic demands of the pregnant state are met by increases in blood volume, and myocardial function, with decreases in systemic vascular resistance (SVR) initially which then begins to rise in the mid-late third trimester.[1] During the surgery, blood loss can contribute to decreased intravascular volume and cardiac output. The use of either neuraxial or general anesthesia also results in a profound decrease in SVR. Immediately after delivery, cardiac output can rise by as much as 80% due to relief of IVC compression by the fetus and a potentially rapid autotransfusion from the placenta.[2] Over the next 12 to 24 hours post-partum SVR continues to increase with the loss of the low resistance placenta, the reversal of the effects of anesthesia and mobilization of dependent edema and interstitial fluid.[1]

Anesthetic agents act to blunt the physiologic increased metabolic demands and hemodynamic stress that normally accompanies labor and delivery and allowed for surgical intervention. The goals of management include avoidance of excessive anesthetic-induced myocardial depression and maintenance of normovolemia, while minimizing the inherent sympathetic stimulation associated with labor and surgery.[3] Ensuring that the anesthesia provider can maintain euvolemia during in this rapidly evolving environment is especially challenging given our patient's co-morbidities and why invasive arterial and pulmonary arterial pressure monitoring was pursued. The use of pulmonary artery catheters (PAC) has been the established, gold standard for assessing these important values.[4-6] Siu et al additionally have developed a risk score to identify predictors associated with unfavorable cardiac events in parturients with heart disease. Our patient had a 75% risk for a peripartum cardiac event,[7] due to her reduced LVEF and NYHA class IV status.

Although some would argue a transesophageal echocardiography (TEE) would have provided an equal or better assessment of volume status there were several reasons against its use in this case. The primary anesthetic for the procedure was neuraxial anesthesia given its maternal and fetal benefits, and when done via epidural it is easily titratable and has beneficial effects on preload and afterload in the setting of decreased LVEF. To have placed and interpreted the TEE would have required deep sedation with effects on the neonate in the immediate neonatal period, as well as have precluded the mother from participating in her delivery. Further, as many of the changes in SVR and fluid status occur in the 12-24 hours postoperatively,[2] a TEE would not have been tolerated by the patient unless sedation was maintained for this extended duration. The PAC remained in place throughout the immediate post-delivery period for this patient, and proved invaluable in guiding treatment.

REFERENCES

1. **Stergiopoulos K, Shiang E, Bench T**. Pregnancy in patients with pre-existing cardiomyopathies. *J Am Coll Cardiol*. 2011 Jul 19;58(4):337-50. doi: 10.1016/j.jacc.2011.04.014. PMID: 21757110.
2. **Robson SC, Dunlop W, Boys RJ, Hunter S**. Cardiac output during labour. *Br Med J (Clin Res Ed)*. 1987 Nov 7;295(6607):1169-72. doi: 10.1136/bmj.295.6607.1169. PMID: 3120929; PMCID: PMC1248244.
3. **Howlett JG, McKelvie RS, Costigan J, Ducharme A, Estrella-Holder E, Ezekowitz JA, Giannetti N, Haddad H, Heckman GA, Herd AM, Isaac D, Kouz S, Leblanc K, Liu P, Mann E, Moe GW, O'Meara E, Rajda M, Siu S, Stolee P, Swiggum E, Zeiroth S; Canadian Cardiovascular Society**. The 2010 Canadian Cardiovascular Society guidelines for the diagnosis and management of heart failure update: Heart failure in ethnic minority populations, heart failure and pregnancy, disease management, and quality improvement/assurance programs. *Can J Cardiol*. 2010 Apr;26(4):185-202. doi: 10.1016/s0828-282x(10)70367-6. PMID: 20386768; PMCID: PMC2886538.
4. **Cotton DB, Benedetti TJ**. Use of the Swan-Ganz catheter in obstetrics and gynecology. *Obstet Gynecol*. 1980 Nov;56(5):641-5. PMID: 7432736.
5. **Hjertberg R, Belfrage P, Hägnevik K**. Hemodynamic measurements with Swan-Ganz catheter in women with severe proteinuric gestational hypertension (pre-eclampsia). *Acta Obstet Gynecol Scand*. 1991;70(3):193-8. doi: 10.3109/00016349109006209. PMID: 1927295.
6. **Chohan U, Afshan G, Mone A**. Anaesthesia for caesarean section in patients with cardiac disease. *J Pak Med Assoc*. 2006 Jan;56(1):32-8. PMID: 16454133.
7. **Siu SC, Sermer M, Harrison DA, Grigoriadis E, Liu G, Sorensen S, Smallhorn JF, Farine D, Amankwah KS, Spears JC, Colman JM**. Risk and predictors for pregnancy-related complications in women with heart disease. *Circulation*. 1997 Nov 4;96(9):2789-94. doi: 10.1161/01.cir.96.9.2789. PMID: 9386139.