Dural Puncture Epidural Anesthesia for Cesarean Section in a Patient with Pre-Eclampsia complicated by Gerbode Defect and Pulmonary Hypertension.

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INTRODUCTION

Cardiovascular conditions are the leading cause of maternal mortality in the United States [1, 2]. Of all types of heart disease affecting pregnant women, pulmonary hypertension (PH) has the highest risk of maternal death or heart failure, despite having the lowest incidence [3]. A 2017 retrospective review of 49 cases in North America reports overall mortality of 8.3% [4], and additional studies show these patients are a high-risk group for poor maternal and fetal outcomes [5]. Because of these risks of high maternal and fetal morbidity and mortality, multiple sources including the World Health Organization (WHO), have issued practice guidelines stating that pregnancy is contraindicated in women with PH [6]. Despite clinical recommendations against pregnancy in those with preexisting PH, cases have been increasing within the United States [2], necessitating an increased understanding of the perioperative management of these patients.

CASE REPORT

A 34-year-old G3P0020 female at 33w4d with a history of morbid obesity with a BMI of 58.3, VSD closure at age 2 complicated by 3rd-degree heart block requiring a pacemaker, with subsequent upgrade to transvenous system at age 12, Atrial flutter, Moderate/severe tricuspid regurgitation, severe Pulmonary HTN, HTN, DM2, HLD, anxiety, is admitted for preeclampsia with severe features. On arrival, she was in atrial flutter with systolic blood pressures of 200 over diastolic of 80s and proteinuria and therefore it was decided by the obstetric team to undergo an elective cesarean section. A preoperative echocardiogram was obtained at the request of the anesthesia team showing a normal LV EF and a left to right shunt between the LV outflow tract to right atrium, consistent with Gerbode defect.

Preoperatively an arterial line was placed. Afterward, a dural puncture epidural was chosen to be the primary anesthetic for dural puncture and slow titration of epidural lidocaine. 14mL of 2% lidocaine with epinephrine was administered slowly over 15 minutes with bolus dosing of norepinephrine for subsequent hypotension. After achieving a T4 anesthetic level a continuous norepinephrine infusion was started at 2 mcg/min. Cesarean was completed without difficulty. Post-operatively patient was managed by cardiology for HTN and volume overload. She required a pacemaker generator exchange and cardioversion for atrial fibrillation and was discharged on post-op day 12. The newborn was brought to the NICU for respiratory support but was transferred to the maternity ward on POD #2 without any significant medical conditions.

Figure 1

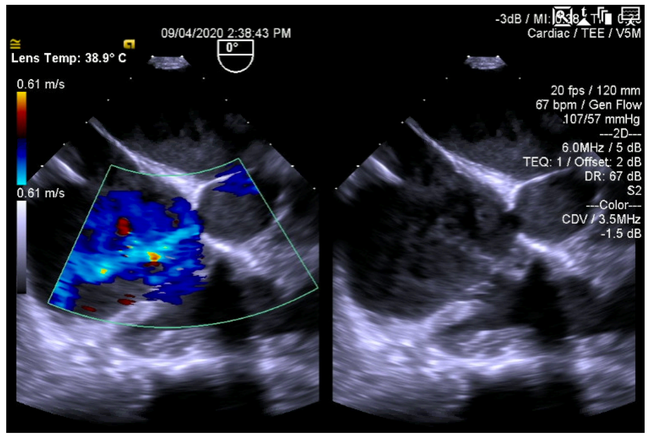

Figure 1: TTE results demonstrating patient's Gerbode Defect, with communication between the LV and RA, contributing to patient's right heart strain

DISCUSSION

Pulmonary hypertension poses significant risks for pregnancy. Increased pulmonary vascular resistance contributes to RV strain and hypertrophy, and when combined with the increased cardiac output of pregnancy can lead to RV dysfunction and potential failure. This is worsened by the loss of normal compensatory mechanisms in pregnancy to reduce PVR, such as dilation of the pulmonary vasculature and RV eccentric hypertrophy[7,8]. Likely, the etiology of the patient's severe PH was her congenital Gerbode defect, a rare communication between the LV and RA that eventually led to Eisenmenger's syndrome.

Delivery via elective cesarean section at 33 weeks was chosen to minimize pregnancy-related increased blood volume and pulmonary arterial pressures, as well as to avoid decreases in preload associated with the Valsalva maneuver during labor [9].

General anesthesia poses several risks to this population including direct myocardial depression by volatile anesthetic agents, decreased venous return associated with positive pressure ventilation, and the large sympathetic response to direct laryngoscopy that can further increase pulmonary arterial pressure[10]. By using DPE as the primary anesthetic agent, we were able to slowly titrate anesthetic to minimize hypotension or rapid decreases in SVR[12]. This also allowed us to minimize the risk of conversion to GA[11] by having a way to continually titrate local anesthetic no matter the duration of the cesarean section. Periods of hypotension were treated with norepinephrine, whose action on α1- and β1-adrenoceptors provides SVR and inotropic support. Continuous fluid infusion and fluid boluses were avoided to prevent hypervolemia and further RV wall tension.

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