Intra-aortic Balloon Pump for Cesarean Hysterectomy and Massive Hemorrhage in a Parturient with Placenta Accreta and Pulmonary Embolus

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Abstract

During cesarean hysterectomy for a placenta accreta, a 36-year-old parturient underwent a massive resuscitation for profound bleeding and also suffered a pulmonary embolus leading to cardiac arrest. Chest compressions and epinephrine were required for resucitation. When surgery was complete, she was taken to the intensive care unit on an epinephrine infusion and inhaled nitric oxide but was brought back to the operating room after 3 h for surgical exploration. Echocardiography revealed a poorly contracting left ventricle, and an intra-aortic balloon pump was inserted. She gradually recovered full function and was discharged home after 35 days.

Keywords: Cesarean hysterectomy, intra-ortic balloon pump, obstetric hemorrhage, placenta accreta, pulmonary embolus

INTRODUCTION

Abnormal placental attachment (e.g., placenta accreta) can result in severe postpartum hemorrhage.[1] Morbidity or mortality may be related to hemorrhage, cesarean hysterectomy, surgical and postoperative complications;[2] however, intraoperative pulmonary embolus during abnormal placentation surgery has not been previously reported.

We present the anesthetic management of a patient with placenta accreta undergoing cesarean hysterectomy who experienced a pulmonary embolus during a massive postpartum hemorrhage. The report highlights the importance of transesophageal echocardiography (TEE) for the diagnosis and intra-aortic balloon pump (IABP) for management. The patient gave written permission for publication of this case.

CASE REPORT

A 36-year-old, gravida 5 para 2 parturient with a singleton pregnancy was scheduled for a cesarean delivery with possible hysterectomy at 34 3/7 weeks. She had been diagnosed with a complete placenta previa and focal accreta at 29 weeks gestation. She had three prior cesarean deliveries with the second one resulting in nonsurviving conjoined twins. She had been in the hospital for 2 weeks before her cesarean delivery due to a large volume bleed (estimated blood loss of 500 ml) that resulted in hypotension and required transfusion of 2 units of packed red blood cells (PRBC). She weighed 76 kg and was 157 cm tall. Her airway exam was Mallampati class II, with normal mouth opening, full range of motion of her neck and thyromental distance >6 cm. Admission hematocrit was 31.6 g/dL, platelet count was 139×10^9/L, and international normalized ratio (INR) was 1.1.

Intravenous (IV) access was obtained (2×16G peripheral IV cannulae). Combined spinal-epidural anesthesia was performed with a 17-G Touhy needle and Gertie Marx 26-gauge needle at the L3/4 interspace. The patient was given a spinal dose of 0.75% bupivacaine 12 mg, fentanyl 10 mcg, and preservative-free morphine 200 mcg intrathecally, and a Springwound 19-G epidural was inserted. The patient obtained a T4 sensory block to cold and sharp sensation bilaterally.

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Access this article online

Quick Response Code:

Website:
www.jmuonline.org

DOI:
10.4103/JMU.JMU_84_18

How to cite this article: Riley ET, Akbar K, Carvalho B. Intra-aortic balloon pump for cesarean hysterectomy and massive hemorrhage in a parturient with placenta accreta and pulmonary embolus. J Med Ultrasound 2019;27:104-6.

Cesarean delivery was performed through a midline skin incision followed by classical uterine incision with the neonatal Apgar scores were 2 and 8 at 1 and 5 min, respectively. Following delivery, general anesthesia was induced, and a supracervical hysterectomy was performed. There was significant bleeding from her sacral plexus, more specifically, the left ovarian vein. A general surgeon was consulted secondary to bleeding and increased hemodynamic instability.

A TEE probe was inserted during the hemorrhage resuscitation to help assess volume status and guide administration of blood, blood products, and fluid management. Soon after transthoracic echocardiography (TTE) insertion, an echodense freely-mobile mass consistent with a large clot swirling in the right atrium was observed. The clot then entered the right ventricle and exited through the outflow tract. Shortly afterward, the patient's end-tidal carbon dioxide suddenly declined, and she went into pulseless electrical activity requiring three rounds of cardiopulmonary resuscitation with chest compressions. She was given multiple doses of epinephrine, sodium bicarbonate, and calcium chloride as indicated. After return of spontaneous circulation, the TTE showed severe global hypokinesis of the right ventricle and left ventricle. Subsequent TEE examination several hours later showed the improved function of an now underfilled left ventricle, severe septal hypokinesis to akinesis with preserved right ventricle apical function consistent with McConnell's sign. Though poorly visualized, there was an echo density in the right pulmonary artery consistent with a large embolus.

The patient was started on inhaled nitric oxide at 15 ppm due to TEE signs showing increased right ventricular pressure, and an epinephrine infusion to maintain blood pressure was initiated. The patient continued to bleed from the surgical site as well as from a liver laceration sustained during the chest compressions, and a 10.5 L estimated blood loss was recorded during her surgery. Intraoperatively she received 48 units of PRBC's, 40 units of fresh frozen plasma, 13 units of platelets, 8 units of cryoprecipitate, 8 L of crystalloid, and 7 mg of factor VII. Hematological parameters after massive volume resuscitation were: hemoglobin 13.4 g/dL, platelet count 193×10^9/L, prothrombin time 13.8 sec, partial prothrombin time 37.1 sec, INR 1.1, and fibrinogen 263 mg/dL.

3 h after going to the intensive care unit she was brought back to the operating room for surgical exploration, abdominal washout, and re-packing. The partial pressure of oxygen in blood remained in the low 40's and epinephrine was required to keep her systolic blood pressure over 90 mmHg. An IABP was placed due to severe cardiac dysfunction, and resulted in a dramatic improvement of her hemodynamics and oxygenation. She was taken back to the intensive care unit on an IABP, inhaled nitric oxide and an epinephrine infusion. 3 days later her IABP was removed after she had stabilized hemodynamically. She continued to improve and was subsequently extubated on postoperative day 7. A bilateral upper extremity venous duplex showed evidence of occlusion in one of the two right brachial veins and noncompression of the cephalic veins bilaterally. She was started on therapeutic anticoagulation (heparin drip with goal of keeping partial thromboplastin time between 55 and 75 s). She required a cystotomy repair on day 30 and was discharged home 35 days after her cesarean hysterectomy without significant morbidity and intact neurological function.

DISCUSSION

Abnormal placental attachment incidence has increased significantly over the past decade, driven by a rise in the rates of cesarean delivery.[3] Placenta accreta is often associated with severe postpartum hemorrhage as occurred in this case. This surgical case presented unique management challenges due to a pulmonary embolus occurring during a massive blood transfusion for obstetric hemorrhage.

Maternal cardiac arrest is estimated to occur in 1:12,000 admissions for delivery.[4] Obstetric hemorrhage is the leading cause of maternal cardiac arrest,[4] and in the setting of massive bleeding and resuscitation for a placenta accreta, would be the presumed primary differential diagnosis for the arrest. Fortunately, a TEE was placed during the hemorrhage to assess volume status and aid resuscitation. Without the TEE *in situ* when the pulmonary embolus occurred, the diagnosis would have been presumed to be due to hypovolemia. The TTE allowed for appropriate management for the cardiac derangement following the pulmonary embolus. Focusing primarily on hemorrhage-related hypovolemia would have worsened the outcome from this case of pulmonary embolus-induced cardiac arrest.

There is an increased risk of pulmonary embolus in pregnancy, and venous thromboembolism with pulmonary embolism is a leading cause of maternal death.[5] In our case, prolonged bed rest and reluctance to prescribe anticoagulants in the setting of placental accreta with the potential risk of massive hemorrhage, likely contributed to her having a pulmonary embolus. Pulmonary emboli occurring after cesarean deliveries are often treated with anticoagulation and embolectomy.[6-8] Thrombolytic therapy is indicated in patients with large pulmonary emboli. However, in concurrent or recent postpartum hemorrhage it may be best to forgo thrombolytic therapy. Bilger *et al*. opted for a temporary filter in the inferior vena cava, and thrombolytic therapy after a patient developed an inferior vena cava thrombosis 6 days after a delivery complicated by a postpartum hemorrhage.[9]

With this case, the embolus occurred intraoperatively and we did not feel that thrombolytics or anticoagulants were safe or indicated in a woman having a massive postpartum hemorrhage. We felt the best course of action beyond maintaining adequate circulating volume was to support cardiac function with epinephrine and help reduce right sided-pressure and improve right ventricular function with pulmonary vasodilation from inhaled nitric oxide. Although

nitric oxide has not definitively been shown to be effective for pulmonary embolus, there is some evidence to suggest it may have a potential role.[10]

To our knowledge, this is the first reported case describing the use of an IABP following a massive hemorrhage and pulmonary embolus in a parturient. A previous case report described the use of IABP to support a pregnant patient in the second trimester after a pulmonary embolus and subsequent embolectomy.[11] The patient recovered and delivered a healthy infant. Other reported cases of IABP use during pregnancy were for the management of primary cardiac lesions.[12-15] In our case, the IABP led to significant hemodynamic improvements and successful recovery.

CONCLUSION

The case highlights the importance of TTE for the management of massive hemorrhage during abnormal placentation surgery. The potential for a venous thromboembolic event should be considered even in the setting of massive hemorrhage, and TTE can help determine the etiolgy and assist the management of a cardiac arrest. The IABP was instrumental in improving her hemodynamics and should be contemplated as a therapeutic option in the setting of cardiac dysfunction not responsive to inotropes.

Declaration of patient consent

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

Financial support and sponsorship

This case report was funded internally by the Department of Anesthesia, Stanford University School of Medicine.

Conflicts of interest

There are no conflicts of interest.

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