



CASE REPORT

Anesthetic Management of Atonic Postpartum Hemorrhage with Hemorrhagic Shock and Impending Cardiac Arrest for Emergency Peripartum Hysterectomy

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ABSTRACT

Postpartum hemorrhagic complication is a critical situation for an anesthesiologist. This situation requires timely and skilful anesthetic management. A massive postpartum bleeding leading to severe hypovolemic shock may result in life-threatening cardiopulmonary arrest. The treatment of postpartum hemorrhage (PPH) has two components: First, resuscitation and control of bleeding and second, identification and management of underlying cause. Here is a case report of a 20-year-old with atonic PPH resulting in hypovolemic shock and impending cardiac arrest and successful anesthetic management for emergency peripartum hysterectomy to save the life of the patient.

Keywords: Cardiac arrest, Hypovolemic shock, Hysterectomy, Postpartum hemorrhage, Rapid sequence induction.

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INTRODUCTION

Postpartum hemorrhage (PPH) is defined as blood loss of more than 500 mL following vaginal delivery or more than 1000 mL following cesarean delivery. A loss of these amounts within 24 hours of delivery is termed early or primary PPH, whereas such losses are termed late or secondary PPH if they occur 24 hours after delivery.¹ The usual presentation of PPH is heavy vaginal bleeding that can quickly lead to signs and symptoms of hypovolemic shock. This rapid blood loss reflects the combination of high uterine blood flow and the most common cause of PPH, i.e., uterine atony.² Hypovolemia may lead to the scenario of a cardiac arrest, which is feared in the labor and delivery suite; yet, the incidence is 1 in 30,000 pregnancies.³

Circulatory arrest is the cessation of normal circulation of the blood due to failure of the heart to contract effectively. Resuscitation and management of obstetric hemorrhage and, possibly, hypovolemic shock and identification and management of the underlying cause(s) of the hemorrhage must be done.⁴

CASE REPORT

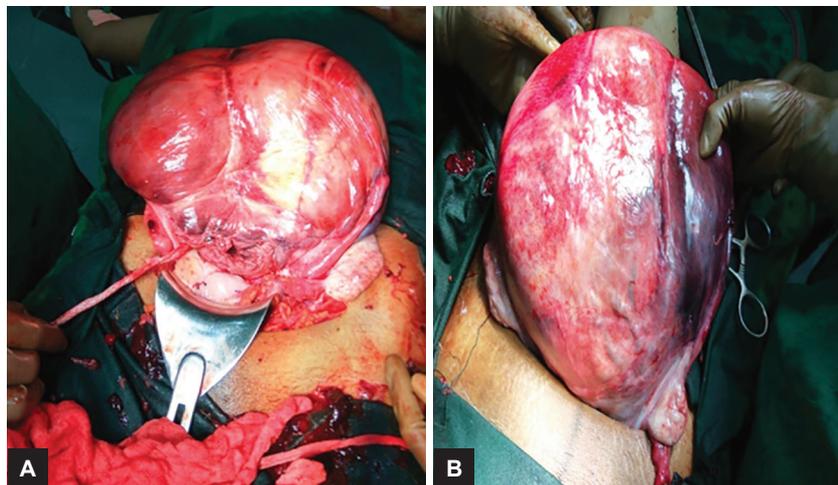
A 20-year-old female, with obstetric history of Gravida 1 Para 1 Live birth 1, presented with full-term pregnancy in labor. She was taken up for emergency cesarean section in view of oligohydramnios. Injection oxytocin 20 units IV and injection methergine was given intraoperatively. At 7.15 AM in the postoperative room, her uterus was intermittently relaxing and there was bleeding per vagina. Medical management of PPH was done using injection oxytocin 20 IU IV infusion. Injection carboprost 250 mg 3 doses IM were repeated over 15 minutes and tablet misoprostol 1200 mg per vagina. At about 7.55 AM, PPH was not controlled and she developed drowsiness, tachycardia, hypotension, and tachypnea, and the gynecologist planned for emergency peripartum hysterectomy.

At 8.05 AM, preanesthetic evaluation was done, patient was drowsy, not responsive, with severe pallor and thready pulse; heart rate (HR) was 160/minute; blood pressure (BP) 60 mm Hg systolic, SPO₂ was 95%; and respiratory rate (RR) was 32/minute. Complete hemogram and coagulation tests were sent on urgent basis. Blood was sent for grouping and cross matching. Patient was shifted immediately to the operation theater, two large-bore 18G IV lines were secured, and monitors attached. Patient was premedicated with injection glycopyrrolate 0.2 mg IV. She was preoxygenated for 3 minutes with 100% O₂. Rapid sequence induction (RSI) was carried out. Injection pentazocine 30 mg IV, injection midazolam 1 mg IV, injection thiopentone 150 mg IV, injection ketamine 50 mg, and injection succinylcholine 100 mg IV were given. Airway was secured with no. 7 cuffed endotracheal tube. Anesthesia was maintained with O₂, intermittent vecuronium, and intermittent positive pressure ventilation. Ringer lactate and normal saline were infused. One unit of whole blood transfusion was started. Five minutes after transfusing blood, pulse was

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Figs 1A and B: Flabby uterus

feeble and BP picked up to 80/46 mm Hg. Heart rate came down to 132/minute. O₂ Saturation improved to 100%. After 2 units of blood transfusion, hemodynamic parameters were stable with HR – 120/minute, BP – 130/90 mm Hg, SPO₂ – 100%; urine output was about 40 to 50 mL per hour. Blood loss was around 2300 mL, which was replaced with 5 units of ringer lactate, 1 unit of normal saline, 1 unit of voluven, 2 units of whole blood, and 4 units of fresh frozen plasma. Uterus was completely flabby and diagnosis of atonic PPH was made and surgeons went ahead with obstetric hysterectomy (Figs 1A and B). She had spontaneous breathing efforts and was reversed with injection neostigmine 3 mg and injection glycopyrrolate 0.4 mg. After monitoring for 5 minutes on table for adequate efforts, patient was extubated after thorough oral suctioning. Patient was drowsy, maintained saturation with O₂ face mask at 4 L/minute, and hemodynamically stable.

She was shifted to intensive care unit for further monitoring. Postoperatively, central line was put for fluid management and central venous pressure monitoring. She was monitored for 2 hours. Her vital parameters on shifting were HR – 106/minute, BP – 140/90 mm Hg, SPO₂ – 100%, RR – 18/minute.

DISCUSSION

The usual presentation of PPH is one of heavy vaginal bleeding that can quickly lead to signs and symptoms of hypovolemic shock. This rapid blood loss reflects the combination of high uterine blood flow and the most common cause of PPH, i.e., uterine atony.² Rapid recognition and diagnosis of PPH are essential to successful management. Resuscitative measures and the diagnosis and treatment of the underlying cause must occur quickly before sequelae of severe hypovolemia and coagulopathy develop. The major factor in the adverse

outcomes associated with severe hemorrhage is a delay in initiating appropriate management. In our case, after cesarean section, patient developed PPH, which in the initial stage was treated medically. In view of failure of these measures to control persistent bleeding and deteriorating condition of patient, decision for emergency hysterectomy was taken. Considering the hypovolemic shock, we decided to take the patient under general anesthesia in such a way that the drugs and techniques used to anesthetize the patient were optimally safe.³ The pharmacological properties required of an intravenous induction agent that satisfies the aims of RSI, therefore, includes rapid onset and few adverse hemodynamic effects. Major cause of hypovolemia, being persistent bleeding from atonic uterus, was treated by crystalloids, colloids, and blood to preserve perfusion of vital organs.

CONCLUSION

This case emphasizes that timely intervention with efficient and coordinated team efforts results in successful outcome in a critical case of atonic PPH undergoing emergency hysterectomy.

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