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Case Report

Postpartum Hemorrhage With Cardiorespiratory Collapse Transported From a Rural Hospital

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Postpartum hemorrhage (PPH) is a highly morbid condition requiring prompt recognition, rapid hemorrhage control, and intensive supportive care. This case report details collaboration between a rural transferring hospital, a long-distance fixed wing academic critical care air medical transport team, and a tertiary receiving hospital. It reviews in-flight resuscitation of the hemodynamically unstable PPH patient.

Case Report

A 33-year-old woman (gravida 3, para 3) with a history of gestational diabetes, hypertension, and a previous cesarean section gave birth to a healthy newborn via cesarean section after a prolonged labor. She experienced PPH and an amniotic fluid embolism. She was intubated, her uterus was repaired, and an intrauterine tamponade balloon was placed. She remained hemodynamically unstable, requiring methylergonovine and misoprostol administration. A massive transfusion protocol and vasopressors were also initiated.

The flight team was activated and found the patient saturating well, with a heart rate of 144 beats/min and blood pressure of 86/52 mm Hg. Arterial blood gas revealed the following: pH = 7.12, pCO_2 = 39, pO_2 = 278, HCO_3 = 12.3, and Base Excess = −15.7. Two units of packed red blood cells (PRBCs) and 2 units of fresh frozen plasma had been given; norepinephrine (20 μg/min), epinephrine (0.05 μg/kg/min), and vasopressin (0.04 U/min) were infusing.

During the 3.5-hour transport, the patient remained hemodynamically unstable with her blood pressure dropping to 57/34 mm Hg. Two units of PRBCs were transfused, and the epinephrine drip was up-titrated. Initial in-flight Venous Blood Gas revealed the following: pH = 6.979, pCO_2 = 48, pO_2 = 26, HCO_3 = 11.3 and Base Excess = −19. The flight team always carries 2 units of O-negative PRBCs and 2 units of low-titer A-positive liquid plasma provided by the regional trauma center, all of which were transfused. Her minute ventilation was also increased to help compensate for profound lactic acidemia. Subsequently, the team was able to down-titrate both vasopressors, the repeat Venous Blood Gas improved, and the patient was successfully transferred.

Upon arrival to tertiary care, the patient was emergently taken for dilation, curettage, intrauterine balloon catheter replacement, and vaginal packing. She later underwent bilateral uterine artery embolization. An additional 7 units of PRBCs, 3 units of fresh frozen plasma, and 1 unit of platelets were transfused. She stabilized in the hospital and was discharged on postpartum day 5 with a final hematocrit of 23.8.

Discussion

PPH is defined as either a cumulative blood loss of ≥ 1,000 mL or blood loss accompanied by signs and symptoms of hypovolemia within 24 hours since birth.[1] It complicates 1% to 5% of deliveries and is the leading cause of maternal mortality worldwide.[2-4] Additionally, sequalae of PPH may include shock, adult respiratory distress syndrome, disseminated intravascular coagulation, acute renal failure, loss of fertility, and pituitary necrosis (Sheehan syndrome).[1] Uterine atony is the most common etiology of PPH, occurring in 70% to 80% of cases.[5,6] The patient described in this case report had several risk factors for PPH including possible fetal macrosomia related to gestational diabetes mellitus, hypertension, and previous cesarean section. Previous cesarean section places a patient at higher risk for both placenta previa and placenta accreta syndrome; both of which are additional risk factors for PPH.[7,8] Prompt recognition and treatment of PPH are critical to decreasing the associated morbidity and mortality.

Given that uterine atony is the most common etiology of PPH, bimanual massage and uterotonic agents are considered the first-line treatment. An escalating series of uterotonic agents is generally recommended with oxytocin, a common starting agent, followed rapidly by carboprost tromethamine (Hemabate, Pfizer, New York, New York/US) and/or methylergonovine. If these agents fail to stop the bleeding, an intrauterine tamponade balloon can be placed. Other

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causes of PPH should be considered, including examination for genital tract lacerations, retained products of conception, uterine inversion, and coagulation defects.

Obstetric trauma such as cervical, vaginal, perineal, or uterine artery lacerations occurring during cesarean section can all lead to life-threatening PPH. Laceration repair to achieve hemostasis is the primary management; however, interventional radiology embolization or laparotomy/hysterectomy in more severe cases may be required depending on the bleeding source (laceration vs. vascular injury) and resource availability.[1]

Retained products of conception are another etiology that can lead to PPH. It is recommended to inspect the placenta fully with each delivery to guard against this etiology, but even with full inspection, additional products of conception could remain. If the patient has persistent PPH and uterine atony and obstetric trauma has been ruled out, an ultrasound can help determine whether retained products of conception are contributing to the hemorrhage. Prompt removal of retained products along with continued supportive care can lead to cessation of the hemorrhage.

Uterine inversion involves the displacement of the uterus below the level of the cervix. It is rare but can lead to significant hemorrhage and cardiovascular collapse. Treatment is centered on manual replacement of the uterus. Muscle relaxation may be required to successfully replace the uterus, and once replaced, the hemorrhage generally has been known to discontinue.[1]

Acute coagulopathy can complicate and exacerbate PPH. The American College of Obstetricians and Gynecologists delineates 2 specific etiologies of acute coagulopathy, beyond simple massive blood loss, that can be implicated. First, placental abruption leading to uterine atony should be considered. In 1 study, placental abruption accounted for 17% of patients with PPH who required massive transfusion.[9] Second, amniotic fluid embolism is another rare cause of PPH and was likely the etiology of the patient presented here. An amniotic fluid embolism can be inferred whenever the triad of hemodynamic instability, respiratory failure, and disseminated intravascular coagulation is present.[10]

In this case, it was presumed that the patient experienced an amniotic fluid embolism in the setting of her PPH given her hemodynamic instability and respiratory failure. Amniotic fluid embolism is a rare event, with 1 study suggesting a rate of 7.7 per 100,000 births (95% confidence interval [CI], 6.7-8.7) with a case fatality rate of 21.6% (95% CI, 15.5%-27.6%).[11] Statistically significant risk factors identified in that study included maternal age greater than 35 years (odds ratio [OR] = 2.2; 95% CI, 1.5-2.1), placenta previa (OR = 30.4; 95% CI, 15.4-60.1), and cesarean delivery (OR = 5.7; 95% CI, 3.7-8.7).[11] Amniotic fluid embolism classically presents with rapid and abrupt cardiorespiratory failure or arrest, disseminated intravascular coagulation (elevated D-dimer, low fibrinogen, and thrombocytopenia), and noncardiogenic pulmonary edema. Treatment is based on supportive care with early mechanical ventilation, massive transfusion, vasopressor support, and continued treatment of other etiologies of PPH as detailed previously.

In this patient's case, she was rapidly treated with aggressive supportive care but ultimately required critical care air transport to a tertiary hospital for uterine artery embolization before she stabilized, allowing vasopressor support to be weaned, extubation, and, ultimately, discharge.

Conclusion

This unique case of a hemodynamically unstable patient experiencing profound PPH and likely an amniotic fluid embolism leading to cardiorespiratory failure shows the value of highly trained and appropriately resourced critical care flight teams, in this case 2 critical care nurses, with prolonged transports. Although not specifically highlighted, a recent trial of prehospital blood transfusion by air medical transport teams showed a mortality benefit for trauma patients in hemorrhagic shock; it was clear that this patient stabilized in no small part because of the blood product transfusions given in flight.[12] As described here, air medical crews involved in prolonged transports of hemodynamically unstable patients should be prepared and resourced to manage and maintain invasive airways, resuscitate with blood products as needed, and tailor their resuscitation with point-of-care laboratory results and frequent patient reassessments.

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