Primary Cesarean Delivery Results in Emergency Hysterectomy due to Placenta Accreta: A Case Study

Jaclyn Humphrey, CRNA, MSN

Placenta accreta is a major cause of obstetric hemorrhage, a situation that remains the most significant cause of maternal morbidity and mortality worldwide. It is generally recognized that a previous cesarean delivery increases the risk of placenta accreta. However, the risk also increases with previous intrauterine procedures. In 2010, The Joint Commission released a sentinel event alert regarding the prevention of maternal death, which recommended the adoption of protocols to treat postpartum hemorrhage. This case study demonstrates the success of quickly initiating protocol interventions necessary to prevent disseminated intravascular coagulation and maternal mortality, while reviewing current literature on risk identification, management, and treatment of obstetric hemorrhage resulting from placenta accreta.

Keywords: Emergency hysterectomy, disseminated intravascular coagulation, obstetric hemorrhage, placenta accreta.

Obstetric hemorrhage remains the leading cause of maternal morbidity and mortality worldwide. Massive obstetric hemorrhage is responsible for 25% to 35% of the estimated 358,000 maternal deaths worldwide each year with more than 80% occurring post partum. Almost half (45%) of postpartum hemorrhage (PPH) cases lead to death within 24 hours. A condition that often leads to PPH is placenta accreta. Placenta accreta is a general term that indicates when the placenta is abnormally attached to the myometrium; it often occurs because of placenta previa and/or previous cesarean delivery.

As the rate of cesarean deliveries increases in the United States, so does the incidence of placenta accreta. It has been reported that placenta accreta has become as frequent as 1 in 533 pregnancies in 2002, increasing from previous reports of 1 in 4,027 in 1970 and 1973. It has been reported that maternal morbidity occurs in up to 60% and mortality in up to 7% of women with placenta accreta. Ultrasonography with Doppler flow mapping and magnetic resonance imaging (MRI) may suggest placenta accreta prenatally. However, a definitive diagnosis of placenta accreta can be obtained only post partum.

The first step in the management of unexpected hemorrhage is the stabilization of the mother's hemodynamic status, which requires large-bore intravenous (IV) access, invasive monitoring, and both aggressive fluid and transfusion therapy. In 2010, The Joint Commission released a sentinel event alert regarding the prevention of maternal death, which recommended the adoption of protocols to treat PPH. This case study demonstrates the successful treatment of PPH and prevention of disseminated intravascular coagulation (DIC) by using such an algorithm.

Case Summary

A 35-year-old woman, gravida 3, para 0, presented to the labor and delivery unit, with spontaneously ruptured membranes and a breech presentation, for a primary cesarean delivery at 39 weeks' gestation. The patient complained of leakage of clear fluid since approximately 2 am. She was experiencing mild contractions on arrival. The patient reported a surgical history of uterine dilation and curettage after 2 spontaneous abortions, and an operative hysteroscopy for resection of possible small septum and intracavitary scar tissue. After the last intervention, the patient reported that she needed an intrauterine balloon for several days. The patient also reported a history of postoperative nausea and vomiting, but claimed no other remarkable medical history or allergies; she was receiving no medications besides prenatal vitamins.

The pregnancy was complicated by advanced maternal age, pregnancy-induced hypertension, and a marginal anterior placenta previa that resolved at 32 weeks' gestation. She had a Mallampati score of 3, and an 18-gauge peripheral IV catheter was inserted in her left forearm. Preoperative vital signs were as follows: blood pressure of 136/71 mm Hg, heart rate (HR) of 112/min, respiratory rate (RR) of 18/min, oxygen saturation measured by pulse oximetry (SpO2) of 100% on room air, and temperature of 36.7°C. Fetal HR was in the range of 130/min. Hematocrit (Hct) was 34%, and the starting platelet count was 177,000/μL.

The patient had eaten toast at 2:30 AM, so the operation was deferred for 6 hours, but increasing contractions and worsening discomfort prompted the decision to move ahead with the cesarean delivery at 8 AM. The patient was dilated to 4 cm, 100% effaced, and –2 station. The patient...
was transported to the operating room, where standard physiologic monitors were applied. A blood pressure cuff was placed on the right upper extremity, set to cycle every 2 minutes. A continuous pulse oximeter and 3-lead electrocardiography monitors were also applied. The primary cesarean delivery was accomplished with spinal anesthesia placed at the L2-3 level, while the patient was in a sitting position, with a 25-gauge pencil-point spinal needle. The spinal placement was uncomplicated and dosed with 0.75% bupivacaine, 1.6 mL, plus fentanyl, 10 μg. The patient was immediately placed in the supine position with a right hip roll. A T4-S5 sensory level was achieved. Immediately before spinal anesthesia was performed, the patient’s blood pressure was 150/75 mm Hg. Her blood pressure was pretreated with phenylephrine, 100 μg, and her postspinal blood pressure was 140/70 mm Hg.

A face mask with 10 L of oxygen flows was applied to the patient. At this time, she received cefazolin, 2 g, and ondansetron, 4 mg. A urinary catheter was placed by the operating room (OR) nurse. Intravenous boluses of ephedrine, 10 mg, and phenylephrine, 200 μg total for the case, were given, with resulting blood pressures of 115/60 to 120/60 mm Hg. Uterine incision was made at 8:48 AM, and a male neonate was delivered at 8:50 AM. Apgar scores were 9/9 at 1 and 5 minutes.

The surgeon noted an irregularity in placental attachment that was later described as a “sticky” 2 × 2 cm area in the anterior and medial part of the uterus. The area of placenta required removal with a sharp curette, which was followed with an oversewing of that area of the endometrium to achieve hemostasis. Oxytocin, 20 U, was administered in 1 L of lactated Ringer’s (LR) solution, and firm uterine tone was noted. When excellent hemostasis was visually confirmed by the surgeon, ketorolac, 30 mg, was administered according to the surgeon’s request. Before the patient left for the postanesthesia care unit (PACU), the uterus was expressed, with minimal bleeding noted. The patient was transferred to the PACU at 9:45 AM with a recorded estimated blood loss (EBL) of 800 mL, fluid intake of 3,000 mL of LR, and urine output of 400 mL. Vital signs in the PACU were blood pressure of 121/73 mm Hg, HR of 93/min, RR of 18/min, SpO2 100% on room air, and a temperature of 37°C.

Shortly after postoperative vital signs were measured, the uterus was expressed again. This time, a very large (approximately 800-mL) clot resulted. The surgeon was notified immediately and subsequently ordered oxytocin, 30 mg, which was administered by the anesthesia provider in 1 L of LR. The surgeon then administered misoprostol, 1,000 μg, rectally. While the patient was continuously monitored in the PACU, a mild amount of bright red vaginal bleeding accompanied by mild abdominal pain was noted. The patient agreed to the insertion of an intrauterine balloon to tamponade the bleeding. However, the attempt made at the patient’s bedside was unsuccessful. The patient’s vital signs remained stable throughout this time.

At 10:50 AM the patient was returned to the OR, where standard monitors were applied. Light sedation was used to facilitate the placement of the intrauterine balloon under ultrasound guidance. The patient received 40 mg of propofol, 2 mg of midazolam, and 200 μg of fentanyl throughout the procedure and was comfortable. A second 18-gauge IV was placed in the right antecubital vein. The patient’s vital signs remained stable and did not require the use of vasopressors. Two liters of LR was administered with oxytocin, 40 U. The documented EBL was 100 mL for the duration of the intrauterine balloon placement, and urine output was 200 mL for the procedure. Vital signs in the PACU at noon were blood pressure of 117/71 mm Hg, HR of 110/min, RR of 18/min, SpO2 of 100% on room air, and temperature of 37°C. The patient was awake, alert, and resting comfortably. At this time, a complete blood cell count, coagulation panel, and blood type and screen/cross were obtained.

Twenty minutes later, at 12:20 PM, the patient was noted to be hypotensive, at 60/40 mm Hg, and reporting dizziness. A 1-L fluid bolus was administered and 2 U of packed red blood cells (PRBCs) ordered. The SpO2 reading dropped to 83%. At this time, a face mask was placed, with 8 L of oxygen bringing the SpO2 reading up to 97%. The patient responded well to the interventions, and her blood pressure rose to 110/70 mm Hg. Within 30 minutes, the patient’s vital signs stabilized, and she was weaned back to room air, maintaining an SpO2 of 100%. She remained stable for approximately 2 more hours. She reporting feeling improved, and she attempted breastfeeding. There was minimal further vaginal bleeding noted at this time.

At 2 PM, the patient experienced a recurrence of substantial perfuse bleeding from the vagina, with a sudden output of 850 mL of bright red blood in the intrauterine balloon tubing. Her blood pressure began trending downward, accompanied by abdominal pain rated 10 on a 10-point scale. The patient received 500 mL of hetastarch (Hespan), and received 2 U of PRBC to treat a Hct value of 25.4%. The initial plan was to attempt a uterine artery embolization in the interventional radiology suite. However, within minutes of the decision, the patient became acutely unstable and was transferred quickly to the OR at 3:30 PM for an exploratory laparotomy. She underwent bilateral uterine artery ligation, abdominal supracervical hysterectomy, and cystoscopy.

The patient arrived in the OR pale and diaphoretic. As the surgical team was preparing the patient, general anesthesia was administered IV with midazolam, 2 mg; fentanyl, 100 μg; propofol, 100 mg; and succinylcholine, 100 mg. The trachea was intubated with a 6.5-mm cuffed endotracheal tube using a Macintosh 3 laryngoscope blade with a grade 2 view. An orogastric tube was...
placed and the stomach contents drained. Anesthesia was maintained with 0.5 minimum alveolar concentration of sevoflurane supplemented with nitrous oxygen. A trauma-1-infuser was used to rapidly infuse 4 U of PRBC and 2 U of platelets through a hot IV catheter line. A right radial arterial line was placed and blood samples were drawn; of note were these values: Hct, 25.3%; platelets, 95,000/μL, and D-dimer, 10,092 ng/mL D-dimer units. Another unit each of PRBC and platelets were administered, and another set of blood samples drawn. The Hct concentration returned to 26%, and another unit of PRBC was given, for a total of 8 U of PRBC with 3 U of platelets throughout the day. Crystalloid use was minimalized for flushing medications and blood products. Total crystalloids for the case equaled 1,000 mL. Hetastarch, 500 mL, was also given to maintain blood pressure, as were IV boluses of phenylephrine, 150 μg total.

All attempts were made to preserve the uterus, but the bleeding could not be controlled and a hysterectomy was performed. Blood loss was estimated to be 2,000 mL during the procedure, leading to a total EBL for the day of 4 to 5 L. At 6:30 PM, the patient was transferred to a propofol IV drip, 50 μg/kg/min, for transportation to the surgical intensive care unit (SICU) after closure of the surgical wound. She also received calcium gluconate, 1 g, IV to correct her most recent calcium level, 2.7 mg/dL, before arriving in the SICU. Her postoperative vital signs were blood pressure of 144/92 mm Hg, HR 92/min, RR 12/min, 100% SpO2 on 100% oxygen, and temperature of 36.5°C. Overnight her Hct dropped to 20%, and was treated with 2 additional units of PRBC. A decision was made to keep the patient intubated overnight. She was extubated without complication at 7 AM the following day. The patient was alert and fully oriented with no deficits. Her vital signs were stable, and laboratory values were normalizing: Hct, 28.7%; platelets, 109,000/μL; and calcium, 7.5 mg/dL. The patient was discharged home 4 days later in stable condition.

**Discussion**

Placenta accreta is a potentially life-threatening obstetric condition that often leads to massive hemorrhage for which the anesthetist needs to be prepared. There are 3 forms of placenta accreta, distinguishable by the depth of placental penetration into the uterus. Placenta accreta, when not used as a generalized term, indicates that chorionic villi of the placenta strongly attach to the myometrium (middle layer of the uterine wall containing mostly smooth muscle) but do not penetrate it. Placenta increta occurs when the chorionic villi invade the myometrium. Placenta percreta, the rarest yet most severe of the conditions, involves the chorionic villi penetrating the entire uterine wall and often results in the placenta attaching to other organs in the abdomen.

Although the development of placenta accreta is not fully understood, it is hypothesized that an underdevelopment of the uterine lining, excessive invasion of the trophoblastic cells (layer of embryo that initially adheres and implants to the uterine wall), or a combination of both are involved as a result of previous instrumentation in the uterus. Major hemorrhage is common during attempts to remove the placenta after delivery. In this case, pathology reports of the patient’s uterine tissues identified, more specifically, the presence of placenta increta.

Women at greatest risk of placenta accreta are those who have had a previous cesarean delivery causing myometrial damage, and women with either an anterior or posterior placenta previa overlying the uterine scar. The risk increases in women with both placenta previa and a previous cesarean delivery, and also as the number of repeated cesarean deliveries increases (Table 1). According to the Society for Maternal-Fetal Medicine, additional risk factors for placenta accreta include increasing maternal age, multiparity, other prior uterine surgery, prior uterine curettage, uterine irradiation, endometrial ablation, Asherman syndrome, uterine leiomyomata, uterine abnormalities, hypertensive disorders of pregnancy, and smoking. However, it is still unknown to what extent each condition actually increases the risk of placenta accreta. Interestingly, the patient did not have a previous cesarean delivery, but she did have an extensive history of uterine procedures, which may have led to damage and scarring of uterine tissue. Also, obstetric and gynecologic records indicated the patient had an anterior placenta previa that resolved at 32 weeks’ gestation.

Diagnosis of placenta accreta before delivery is ideal, but there are no diagnostic techniques at this time that can definitively rule in or rule out the condition. Placenta accreta is usually affirmed post partum using specimens from the hysterectomy. That being said, placenta accreta can be suspected using our knowledge of risk factors combined with the findings of ultrasonography, Doppler ultrasonography, and MRI. Ultrasonographic features suggestive of placenta accreta are irregularly shaped placenta lacunae (vascular spaces) in the placenta giving a “Swiss cheese” appearance, lack of a myometrial zone (also

**Table 1. Risk of Placenta Accreta After Previous Cesarean Delivery**

<table>
<thead>
<tr>
<th>No. of previous cesarean deliveries</th>
<th>Risk of placenta accreta (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>1.9</td>
</tr>
<tr>
<td>1</td>
<td>15.6</td>
</tr>
<tr>
<td>2</td>
<td>23.5</td>
</tr>
<tr>
<td>3</td>
<td>29.4</td>
</tr>
<tr>
<td>4</td>
<td>33.3</td>
</tr>
<tr>
<td>5</td>
<td>50.0</td>
</tr>
</tbody>
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(From Walfish et al2 by permission of Oxford University Press.)
known as a hypoechoic border) between the placenta and myometrium, blood vessels or placental tissue bridging the uterine-placental margin, retroplacental myometrial thickness of less than 1 mm, and increased vasculature seen on color Doppler sonography.\(^5,7\) However, there are disagreements as to which sonographic and Doppler findings are conclusive of placenta accreta, and the number of the patients included in these diagnostic studies is small.\(^5,7\) Currently there is a lack of evidence suggesting that MRI improves pregnancy management or outcomes in patients with sonographically suspected placenta accreta.\(^5,7\) Magnetic resonance imaging may be helpful if ultrasonography is inconclusive or if there is question of placenta increta and percreta specifically.\(^5,7\) As for laboratory markers, there may be a direct relationship between placenta accreta and maternal levels of α-fetoprotein and human chorionic gonadotropin.\(^5,6,8\) However, neither of these markers has been evaluated for screening or diagnostic purposes.\(^9\)

If placenta accreta is suspected, management often begins with planning delivery at a medical center with an experienced multidisciplinary care team that has the resources to handle emergent hemorrhage. Delivering at such a facility resulted in a nearly 80% risk reduction among those women with suspected placenta accreta.\(^6,9\) The multidisciplinary team should include a gynecologic surgeon proficient in pelvic surgery, an anesthetist, a blood bank team, a urologist in case bladder resection or repair is required, an intensivist for postpartum care, and an interventional radiologist should artery catheterizations be used.\(^6,7\) Prophylactic antibiotics are also indicated, and the regimen should be repeated every 2 or 3 hours of surgery or after 1,500 mL of EBL.\(^5,7\)

Recommendations for the safest delivery for a woman with suspected placenta accreta is a planned preterm (34-35 weeks) cesarean hysterectomy with the placenta left in situ because attempts at removing the placenta are associated with substantial hemorrhage.\(^3,7\) Conservative treatments to preserve fertility report higher risks of postoperative complications such as severe hemorrhage, DIC, and infection resistant to antimicrobial therapy that may require laparotomy and hysterectomy.\(^3,6,8\) These women continue to be at risk for weeks or even months after delivery.\(^6\) If placenta accreta is unexpected before cesarean delivery, as in this case study, the placement of deep myometrial sutures in that area may achieve hemostasis if a focal area of partial placenta is identified on removal of the placenta.\(^2,3,5\)

Despite the planned course of delivery, it is essential that the anesthetist be prepared for the possibility of substantial maternal hemorrhage, a potential for emergency hysterectomy, and the prevention of DIC. The best anesthetic method for women with placenta accreta is highly debated, and goes back to individualizing the anesthetic based on a review of pertinent history, physical examination findings, laboratory results, and imaging data.\(^6\) However, the American Society of Anesthesiologists Task Force on Obstetric Anesthesia advised that general anesthesia might be the most appropriate method because of the potential blood loss and subsequent interventions necessary to maintain hemodynamic stability.\(^9\) It is universally agreed that anesthetic considerations should include large-bore venous access to allow rapid crystalloid and blood produce infusion, availability of high flow-rate infusion devices, hemodynamic monitoring capabilities (central venous and peripheral arterial access), and devices to prevent thromboembolism.\(^3\) Special attention should also be given to padding and positioning the patient to prevent nerve compression, as well as aggressive prevention and treatment of hypothermia.\(^2,5,6,8\)

A life-threatening consequence of PPH that the anesthetist wants to prevent is the development of DIC. Postpartum DIC presents earlier than DIC resulting from surgical or traumatic hemorrhage, often leading to maternal death within 24 hours.\(^2,3,10\) Disseminated intravascular coagulation is a disruption of hemostasis involving a massive activation of the clotting cascade.\(^11\) This results in widespread thrombosis, depletion of platelets and coagulation factors, and excessive thrombolysis.\(^12\) Once DIC has begun, control of coagulation activation and thrombin generation presents a major challenge to successful treatment. The result is often multiorgan failure, continued hemorrhage, and death. Between 1998 and 2009, the prevalence of pregnancy-related DIC increased by 35.9% (from 9.2 to 12.5 cases per 10,000 delivery hospitalizations) and accounted for approximately one-fourth of maternal deaths during that period.\(^13\)

Fundamentally, the principal course of action to avoid DIC is to quickly recognize and control bleeding, yet quantifying the actual amount of blood lost by the patient proves challenging. There is some disparity regarding the definition of severe PPH with values ranging from more than 500 mL after vaginal delivery (1,000 mL after cesarean delivery)\(^2\) to actual or expected blood loss greater than 1,500 mL.\(^8\) Al-Nuaim et al\(^12\) suggest that because blood loss after delivery is difficult to measure, PPH may be defined as a reduction in Hct concentration requiring blood transfusion. Shields and colleagues\(^8\) recommend assessing blood loss by weighing every laparoscopic sponge, the bedware if needed, and all the fluid in collection systems. From this total, any nonblood fluid (amniotic fluid) in the collection system before delivery of the placenta should be subtracted from the EBLs. After delivery, bedding should be changed to eliminate the risk of amniotic fluid contamination from that point forward.\(^8\) This method has been shown to improve the accuracy of blood loss, which greatly assists in the ability to diagnose postpartum hemorrhage.\(^8\) Vital signs as well as other clinical signs such as weakness, sweating, and syncope should also be taken into consideration along
with objective data. However, as seen in this case study, stable vital signs do not rule out continued bleeding.

Another key element to quickly diagnose PPH is to be on alert for patients with multiple risk factors. The protocol developed by Shields and colleagues categorizes women into low-, medium-, and high-risk groups based on medical history and laboratory values (Table 2). Based on the risk assessment, multidisciplinary teams (obstetrician, anesthesia, blood bank, etc) should be alerted for potential obstetric hemorrhage to make necessary preparations such as having cross-matched blood available for high-risk parturients. It is important to determine as early as possible if the blood bank has available stores to meet emergent hemorrhage needs, as well as allow additional time to secure products needed for patients with rare blood types or antibodies. Based on the risk assessment provided by Shields et al, the patient in this case study would have fallen into the medium-risk group for obstetric hemorrhage because of her prior uterine surgeries. Placenta accreta was not diagnosed previously, and her placenta previa resolved.

The protocol expands the diagnosis of hemorrhage into 3 stages so that bleeding can be quickly identified and appropriate interventions taken. The protocol also allows the team to quickly recognize failed interventions, which allows for rapid progression to the next intervention in a successive manner before substantial blood loss occurs. Stage 0 is designated for a normal course of delivery with minimal blood loss. Stage 1 is defined as bleeding greater than expected for a normal vaginal delivery (500 mL) or cesarean delivery (1,000 mL). Along with blood loss, patients are advanced to stage 1 if abnormal vital signs or patient symptoms such as a sustained HR rate above 100/min, blood pressure below 85/45 mm Hg, shortness of breath, confusion, or agitation are noted.

Stage 2 is indicated for bleeding that does not respond to conservative treatment, which includes oxytocin, uterine massage, methylergonovine (Methergine, 0.2 mg IM), carboprost tromethamine (Hemabate, 250 μg IM), and misoprostol (800 to 1,000 μg rectally). At stage 2, multidisciplinary teams are notified and advised to assist in care. An anesthesia provider and the obstetrician are asked to the bedside while vital signs are taken every 5 minutes and oxygen is administered to the patient. At this time, a second IV access should be obtained, a urinary catheter should be inserted if not already present, and bloodwork should be sent to the laboratory. Additional interventions to cease bleeding are suggested such as an intrauterine balloon, uterine artery ligation, and interventional radiology. Placement of an intrauterine balloon (balloon tamponade) was attempted in this case study at bedside but failed, which prompted a second return to the OR. Although intrauterine balloon tamponade has not yet been proved to control ongoing PPH any better than other methods, it is usually attempted first because it can be rapidly deployed, is less invasive than surgery, and lacks major complications. The balloon is inserted into the uterine cavity and filled with sterile water or saline until the uterus is firm to abdominal palpation. The purpose of the balloon is to perform a “tamponade test,” which has a positive result if little or no bleeding is observed from the vagina or catheter attached to the intrauterine balloon. Ongoing bleeding (actual blood loss or EBL exceeding 1,500 mL) would require surgical exploration, as noted by stage 3 of the protocol.

Because the patient’s vital signs stabilized for a short time after the intrauterine balloon placement but bleeding was still noted, plans were made to take the patient to the interventional radiology suite in hopes of performing a uterine artery embolization. The uterine arteries branch from the anterior trunk of the internal iliac arteries and provide the primary blood supply to the uterus. Embolization of these vessels requires fluoroscopic guidance, the expertise of an interventional radiologist, and a stable patient. Success rates for embolization are reported as high as 85% to 95%, and preserve both the uterus and fertility. However, the patient quickly decompensated before arriving at interventional radiology and was therefore rapidly transferred to the OR for an

<table>
<thead>
<tr>
<th>Low risk (clot-to-hold in blood bank)</th>
<th>Medium risk (type and screen)</th>
<th>High risk (cross-matched and blood bank alert)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unscarred uterus</td>
<td>Prior uterine surgery</td>
<td>Placenta previa/accreta</td>
</tr>
<tr>
<td>No history of postpartum hemorrhage</td>
<td>History of postpartum hemorrhage</td>
<td>Hematocrit &lt; 30% with other risk factor</td>
</tr>
<tr>
<td>≤ 4 previous vaginal deliveries</td>
<td>≥ 4 previous vaginal deliveries</td>
<td>Bleeding on admission</td>
</tr>
<tr>
<td>Singleton pregnancy</td>
<td>Multiple gestation</td>
<td>Coagulation defect</td>
</tr>
<tr>
<td></td>
<td>Large uterine fibroids</td>
<td>Platelets &lt; 100,000/μL</td>
</tr>
<tr>
<td></td>
<td>Chorioamnionitis</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Magnesium sulfate use</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Positive antibodies on antepartum screen</td>
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</table>

Table 2. Risk Assessment at Admission
(Reprinted from Shields et al with permission from Elsevier.)
exploratory laparotomy, bilateral uterine artery ligation, abdominal supracervical hysterectomy, and cystoscopy.

General anesthesia was quickly induced, and the patient’s trachea was intubated. Because the patient experienced substantial blood loss up to this point (estimated between 2 and 3 L), the administration of blood products was a priority. There is no current consensus regarding the optimal ratio of blood products during transfusion of a hemorrhaging patient, but recent study results from patients with combat-related trauma requiring massive transfusion suggest that a high plasma to PRBC (1:1.4) ratio significantly improves survival.2,5 Shields and colleagues8 administered PRBC to plasma in ratios of 3:2 for the first 6 U of PRBC and 4 U of plasma, and then the ratio was increased to 1:1 based also on evidence from combat-related hemorrhage studies. The protocol treats toward laboratory goals of Hct greater than 24%, international normalized ratio less than 1.4, platelets above 50,000/μL, and fibrinogen more than 100,000 mg/dL. The use of intraoperative cell-salvage technology (eg, Cell Saver) in PPH has been debated because of the theoretical concern of possibly causing amniotic fluid embolism syndrome.2,5 Although there are available filters that eliminate this risk, fetal red blood cells may remain in the final product (range, 0.13%-4.35%) and risk alloimmunization.5 Intraoperative cell-salvage technology has been used during cesarean deliveries with minimal complications,2 but it is important to acknowledge the need for fresh frozen plasma, cryoprecipitate, and/or platelets while using cell-salvage autotransfusion during hemorrhage because a proportion of the coagulation factors and platelets are removed in the reconstitution process.5

The obstetric and gynecologic surgical team attempted to preserve the uterus and fertility of the patient, yet understood if alternative interventions failed, hysterectomy should not be delayed. In most cases of uterine rupture or placenta accreta, early hysterectomy is recommended.3,5-8 However, in selective cases regarding hemorrhage in the presence of uterine atony, it may be reasonable to consider treatment with recombinant activated factor VII (rFVIIa) before attempting a hysterectomy.3 Recombinant activated factor VII is a synthetic vitamin K–dependent glycoprotein that activates the extrinsic pathway of the coagulation cascade resulting in the increased generation of thrombin and a stable fibrin plug limited to the site of tissue injury, which makes it particularly useful in the obstetric setting.2,14 Because of its mechanism of action, rFVIIa works best after the administration of blood products and in the presence of adequate numbers of circulating platelets and adequate fibrinogen concentration.14 It is also important to correct acidosis for optimal function of rFVIIa.14 Its use is not without side effects, however. A large cohort study published in The New England Journal of Medicine found that “treatment with high doses of rFVIIa significantly increased the risk of arterial but not venous thromboembolic events, but mostly among elderly” patients. Conversely, a smaller study of only obstetric hemorrhage cases did not find an increased risk of thromboembolic events.15 Therefore, recombinant activated factor VII is recommended for treatment of refractory hemorrhage when other “traditional well tested options” have been exhausted.2,16 See Table 3 for the medications and most commonly used doses to treat obstetric hemorrhage.

### Conclusion

Placenta accreta is known to be a major source of obstetric hemorrhage, a situation that remains the most noteworthy cause of maternal morbidity and mortality worldwide. As the rate of cesarean deliveries and intrauterine procedures increases in the United States, so does the incidence of placenta accreta. Anesthesiologists play an important role in the multidisciplinary team with their knowledge in fluid management, transfusion therapy, and critical care to prevent perioperative and postoperative complications of hemorrhage.

This case study stresses the importance of acting quickly to successfully control PPH resulting from pla-

### Table 3. Medications and Most Commonly Used Doses to Treat Obstetric Hemorrhage2,8,17

<table>
<thead>
<tr>
<th>Medication</th>
<th>Most common dose and mode of administration</th>
<th>Cautions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oxytocin</td>
<td>10-40 U in 1 L of saline, IV rapid infusion</td>
<td>May produce antidiuretic effect and produce water intoxication, cerebral edema, and convulsions if used with aggressive IV hydration</td>
</tr>
<tr>
<td>Methylergonovine (Methergine)</td>
<td>0.2 mg IM; repeat every 5 minutes as needed</td>
<td>Avoid in patients with hypertension</td>
</tr>
<tr>
<td>Prostaglandin F2-α, carprofen tromethamine (Hemabate)</td>
<td>0.25 mg IM or intramyometrial; repeat every 15 minutes as needed</td>
<td>Avoid in patients with asthma or hypertension</td>
</tr>
<tr>
<td>Prostaglandin E1 suppositories: misoprostol (Cytotec)</td>
<td>800-1,000 μg rectally or sublingually</td>
<td>None</td>
</tr>
</tbody>
</table>
Placenta accreta. Patients with placenta accreta require close monitoring despite stable vital signs because continued bleeding may be undetectable. As demonstrated, a protocol is beneficial in that it may alert medical staff to potential hemorrhage situations, allow for available resources to rapidly treat persistent bleeding, and prevent DIC and maternal mortality. It is strongly recommended that such a protocol be established in each institution that performs obstetric care, as well as opportunities to practice the hemorrhage scenarios on obstetric floors to ensure a high state of readiness across the multidisciplinary teams. Finally, the author recommends the pursuit of further research in the diagnosis and management of placenta accreta and obstetric hemorrhage in hopes of preventing unnecessary maternal death in the future.

REFERENCES

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