Uterine rupture in a primigravid patient and anesthetic implications: A case report

Alexander A. Litwin, CRNA, MSNA
Lake Mary, Florida

Uterine rupture is an unexpected, relatively uncommon occurrence in the general obstetric population, but it is a potentially devastating complication. Uterine rupture of the unscarred uterus is extremely rare. Awareness of the risk factors as well as the signs and symptoms of uterine rupture are essential for an early diagnosis and prompt treatment.

The patient is a 38-year-old female, gravida 3, para 0, at 38 weeks' gestation undergoing an elective labor induction. The induction of labor and epidural analgesia progress relatively uneventfully. Following approximately 1.5 hours of "pushing," a viable male infant was delivered. Newborn Apgar scores were 6 at 1 minute and 9 at 5 minutes. An hour after delivery the patient began complaining of syncope; at this time bleeding was greater than expected and the obstetrician decided a dilatation and curettage for retained placenta was necessary. A dilatation and curettage was negative, and an ultrasound of the abdomen revealed the presence of significant blood clots, laparotomy was performed, and uterine rupture was identified. The patient developed disseminated intravascular coagulation, uterine bleeding continued, and the patient ultimately required a hysterectomy.

Key words: **Epidural analgesia, obstetrical anesthesia, uterine rupture.**

Rupture of the gravid uterus is an unexpected, relatively uncommon occurrence in the general obstetric population, but it is a potentially devastating complication that must be diagnosed promptly.[1,2] The incidence of uterine rupture may vary appreciably among institutions with a reported range of 0.3% to 1.7 % for women with a history of a uterine scar, and 0.03% to 0.08% among delivering women with an unscarred uterus.[1-5] Previous cesarean section is recognized as the primary risk factor for uterine rupture in the United States and rupture of intact, unscarred uterus is rare.[1,2,4,6,7]

Early diagnosis and swift treatment of uterine rupture is essential for optimal maternal and fetal outcomes. When a patient chooses a trial of labor after the previous cesarean section, the obstetrical staff is keenly aware of the risk for uterine rupture at the scar site. Uterine rupture of the unscarred uterus is extremely rare and may not be in the immediate differential diagnosis, being confused with appendicitis, intestinal obstruction, or abruptio placenta.[6] Maternal mortality rates are reported as 0% to 2%.[1,3] Death is usually secondary to delayed diagnosis, inadequate blood transfusion, or delayed laparotomy.[6] The incidence of fetal mortality with rupture of the uterus varies from 0% to 25%.[1,3,5] Awareness of the risk factors, as well as the signs and symptoms of uterine rupture, are essential for an early, accurate diagnosis and prompt treatment.

Case presentation

The patient is a 38-year-old female, gravida 3, para 0, at 38 weeks' gestation undergoing an elective induction for oligohydraminos and decreased fetal movement. Pertinent history includes uterine fibroids, polycystic ovarian disease, and infertility. Surgical history includes laparoscopic laser vaporization and 2 dilatation and curettage procedures. The patient was admitted at 2:45 PM, and prostin gel, 5 mg, was applied to facilitate dilatation. As per hospital policy regarding healthy parturients with uncomplicated pregnancies, no laboratory studies were ordered. Induction with pitocin was initiated at 8:30 AM the following morning, and a labor epidural was placed at noon when the patient achieved a cervical dilatation of 4 cm. Labor analgesia was provided with a bolus of a standard solution of ropivacaine, 0.2 %, and maintained with the same solution plus 2 µg of fentanyl per mL at 10 mL per hour.

The labor induction and epidural analgesia progressed relatively uneventfully except for the patient experiencing intermittent nausea, vomiting, and occasional episodes of a headache. Vital signs remained within preinduction and preanalgesia range. The obstetrician ruptured the membranes at 7:30 PM with return of clear amniotic fluid. At 8:30 PM the patient complained of upper abdominal pain, which continued between contractions. At 10:15 PM following approximately 1.5 hours of "pushing," a viable male infant was delivered vaginally with vacuum assistance. Newborn Apgar scores were 6 at 1 minute and 9 at 5 minutes. Immediate maternal postdelivery vital signs were blood pressure, 105/75, and heart rate, 79.

Estimated blood loss was noted by the obstetrician as 500 mL for the vaginal delivery. As per institutional policy for removing epidural catheters at a convenient opportunity, the epidural catheter was removed 30 minutes after delivery in consideration of uneventful delivery and stable vital signs.

At 45 minutes postdelivery, the patient began complaining of syncope; maternal vital signs were blood pressure, 87/56, and heart rate, 112. The patient was placed from a head-up 30-degree position to a Trendelenberg position, and at 60 minutes postdelivery the blood pressure was 97/66 and heart rate was 104. Intravenous fluids (lactated Ringer's solution) was increased to maximum flow at the initial decrease in blood pressure to provide a 500 mL bolus over 15 minutes. At 11:20 PM large clots were noted with the vaginal bleeding and methylergonovine (Methergine), 0.2 mg, was administered intravenously. Intravenous lactated Ringer's solution continued to infuse at a wide-open rate. By 11:50 PM, "free flow bleeding with clots" was noted, and a second dose of methylergonovine, 0.2 mg, was administered intravenously. Additional estimated blood loss was now noted to be 1,000 mL. At 11:45 PM, the obstetrician, concerned that the bleeding was secondary to retained placenta, decided a dilatation and curettage was necessary. Vaginal bleeding continued and hematocrit was reported as 22.3%. In preparing the patient for the operating room, 2 units of packed red blood cells were started, and an indwelling urinary catheter was placed with frank blood noted in the urine.

At 12:20 AM, the patient was transferred to the operating room for a dilatation and curettage, possible laparotomy, or possible hysterectomy. The patient underwent a rapid sequence induction with etomidate (Amidate) and succinylcholine, an atraumatic intubation was performed, and general anesthesia was maintained with oxygen and 0.75% to 1.0% end-tidal sevoflurane. Following induction of anesthesia, a radial arterial line was placed, a right internal jugular triple-lumen catheter inserted, and initial fluid volume resuscitation was initiated with lactated Ringer's solution and normal saline until blood products were available. Initial vital signs were blood pressure, 80 to 100 systolic; 45 to 60 diastolic; and heart rate, 100 to 120. A dilatation and curettage was performed with no blood clots or retained placenta identified. Ultrasound of the abdomen revealed significant blood clots, which could not be identified as intra- or extrauterine. A laparotomy was performed, and a uterine rupture was identified with 800 mL of blood evacuated from the peritoneal cavity. The diagnosis of disseminated intravascular coagulation was presumed due to the continual oozing at the surgical site and gross hematuria (with no evidence of bladder rupture). The diagnosis was later confirmed with laboratory studies. The initial platelet count was 54,000; the prothrombin time, 52 seconds (control 8-11.2); partial prothrombin time, 87.9 seconds (control 27-36.6); international normalized ratio 4.89 (control 0.8-1.2); and fibrin split products, 640 (control < 5). The surgeon was unable to control continued bleeding with uterine artery clamping, and the patient ultimately required a hysterectomy. Fluid resuscitation continued with a total of 6 units of packed red blood cells, 10 units of fresh frozen plasma, 1 10-pack of pooled platelets, and 1 unit of crioprecipitate.

At the completion of surgery, the patient was transported to the intensive care unit, intubated, and maintained on ventilatory support because of the potential for development of adult respiratory distress syndrome due to the fluid volume resuscitation. Vital signs had returned to preinduction levels. The patient was awake and alert shortly after admission to the intensive care unit, was maintained intubated and on ventilatory support for several days secondary to pulmonary edema, was extubated on the fourth postoperative day, and was discharged from the hospital 3 days later. The newborn male infant was discharged from the hospital 2 days after delivery to the care of eager grandparents.

Discussion

Although uterine rupture is a rare occurrence, knowing the risk factors, signs, and symptoms are important for early detection and treatment. The most common cause for uterine rupture is separation of a previous cesarean section scar.[6,7] Other causes for uterine rupture include operations or manipulations (such as curettage, perforation, or myomectomy), indirect or blunt trauma (eg, seat belt trauma), penetrating wounds, congenital uterine abnormalities, placenta percreta, and uterine tumors.[7-9] The primary risk factor for uterine rupture is previous cesarean section. Shipp et al identified a 3-fold increased risk of uterine rupture during a trial of labor after cesarean delivery in patients having a short interdelivery interval of 9 to 18 months.[10] Additional risk factors for uterine rupture are grand multiparity, breech version, fetal macrosomia, evidence of dysfunctional labor, fetopelvic disproportion, excessive suprafundal pressure, prolonged labor with excessive pitocin use, and precipitous labor.[2,6-8]

The use of uterotonic agents has been associated with an increased risk of uterine rupture. Yap et al found 71.4% of women who experience uterine rupture had labor induced or augmented with pros-

taglandin E-2 or oxytocin. Induction of labor with oxytocin appeared to confer a 4.6-fold increased risk of uterine rupture compared with no oxytocin use among women with previous cesarean section who attempted a trial of labor.[4] Leung et al found that dysfunctional labor increased the risk of uterine rupture with dysfunctional labor noted in 44% of women with uterine rupture and in 10% of the control patients. Types of dysfunctional labor noted were protracted active phase of labor, arrest of dilatation, prolonged latent phase of labor, and arrest of descent.[8] Hamilton et al noted 5 indicators of dystocia studied were found more frequently than expected in the uterine rupture group. The 5 indicators involved cervical dilatation at time of admission, arrest of dilatation for greater than 2 hours, arrest of dilatation for greater than 4 hours, 2-hour arrest of dilatation associated with admission dilatation, and final examination of dilatation. The author determined that unusually slow progress of cervical dilatation is more common than expected among women with uterine rupture. They also concluded that women with a previous cesarean section delivery might be less able to tolerate nonprogressive labor.[3]

Signs and symptoms of uterine rupture include vaginal bleeding, severe uterine or lower abdominal pain, shoulder pain from subdiaphragmatic irritation by blood, disappearance of fetal heart tones, severe maternal hypotension, and maternal shock.[4] The clinical picture of uterine rupture is variable and depends on the time of occurrence, cause, degree and extent of rupture, amount of bleeding, and the general condition of the patient. Oxorn divides uterine rupture into several groups: silent or quiet, violent, and uterine rupture with delayed diagnosis. A silent or quiet rupture presents without initial dramatic signs and symptoms often with only a rise in maternal heart rate, pallor, and slight vaginal bleeding. This usual variety of rupture develops over several hours characterized by abdominal pain, rapid maternal heart rate, pallor, tenderness on palpation, and absence of fetal heart. If not diagnosed, hypotension and shock may occur. A violent rupture is apparent almost immediately, characterized by a sharp pain following a hard uterine contraction, the presenting fetal part is no longer at the pelvic rim, and fetal movement and heart rate cease. Signs and symptoms of shock appear suddenly and complete cardiovascular collapse may occur.[11] Uterine rupture with delayed diagnosis is a condition that is not evident until the patient is in a process of gradual deterioration. Diagnosis may evolve from evaluation of unexplained anemia, the development of a palpable hematoma in the broad ligament, or the patient goes into shock. Sometimes the diagnosis is made only at autopsy.[11]

Historically, there has been concern that epidural analgesia may mask the pain of uterine rupture thereby delaying the diagnosis of uterine rupture. However, pain, uterine tenderness, and maternal tachycardia have a low sensitivity as diagnostic signs and symptoms of uterine rupture. Fetal heart rate anomaly (tachycardia and late, variable, or prolonged deceleration) is usually the first sign of uterine rupture with maternal pain associated with tachycardia and hypotension from blood loss following changes in fetal heart rate.[1,2,4] When epidural analgesia is implemented for labor, the pain associated with uterine rupture may break through a previously adequate epidural block and is described as sharp unremitting upper abdominal pains not related to contractions.[9,12]

Obstetrical management options for uterine rupture include uterine repair, uterine artery ligation, and hysterectomy.[12,13] With spontaneous uterine rupture or frank rupture during a trial of labor after a previous cesarean delivery, a hysterectomy is frequently required.[12] In select cases, suturing of the rupture may be performed when the uterine musculature can be reestablished to assure a reasonable degree of success and safety for a future pregnancy.[11] Clamping of the uterine rupture site involving the lower segment of the uterus may lead to inadvertent clamping of the ureters, bladder, or both.[1] Clamping of the uterine arteries and veins may assist in controlling bleeding. Ligation of the internal iliac arteries at times reduces the hemorrhage appreciably, but may not completely control bleeding and may delay performance of the definitive treatment, hysterectomy.[11,12] Anesthetic management for a patient with uterine rupture is usually general anesthesia.[12,13] Invasive hemodynamic monitoring may be appropriate in the acutely hypovolemic patient with aggressive fluid volume resuscitation, maintenance of urine output, and the administration of blood and blood component replacement.[12,13]

Summary

The exact time of uterine rupture in the case reported is difficult to identify. After delivery the patient remained hemodynamically stable for over an hour. Syncope and hypotension led to a search for the differential diagnosis. Vaginal bleeding secondary to retained placenta was the primary diagnosis until signs of disseminated intravascular coagulation developed, the dilatation and curettage was negative, and the ultrasound identified extrauterine blood clots. Uterine rupture in a healthy primigravid patient is extremely rare and of low probability in the initial differential diagnosis of vaginal bleeding with syncope and hypotension. In the case presented, aggressive resuscitation and

treatment aided in overcoming the delay in the diagnosis and definitive treatment for uterine rupture.

Being aware of the risk factors and signs and symptoms for uterine rupture, anesthesia providers can increase the early recognition of uterine rupture and be prepared for the aggressive therapy necessary for successful treatment.

REFERENCES

1. Leung AS, Leung EK, Paul RH. Uterine rupture after previous cesarean delivery: maternal and fetal consequences. *Am J Obstet Gynecol.* 1993;169:945-950.
2. Phelan JP, Korst LM, Settles DK. Uterine activity patterns in uterine rupture: a case-control study. *Obstetrics Gynecol.* 1998;92;394-397.
3. Hamilton EF, Bujold E, McNamara H, Gauthier R, Platt AW. Dystocia among women with symptomatic uterine rupture. *Am J Obstet Gynecol.* 2001;184:620-624.
4. Yap OW, Kim ES, Laros RK Jr. Maternal and neonatal outcomes after uterine rupture in labor. *Am J Obstet Gynecol.* 2001;184:1576-1581.
5. Bujold E, Gauthier RJ. Neonatal morbidity associated with uterine rupture: what are the risk factors? *Am J Obstet Gynecol.* 2002;186: 311-314.
6. Miller DA, Goodwin TM, Gherman RB, Paul RH. Intrapartum rupture of the unscarred uterus. *Obstet Gynecol.* 1997;89:671-673.
7. Diaz SD, Jones JE, Serykakov M, Mann WJ. Uterine rupture and dehiscence: ten-year review and case-control study. *South Med J.* 2002;95:431-435.
8. Leung SA, Farmer RM, Leung EK, Medearis AL, Paul RH. Risk factors associated with uterine rupture during trial of labor after cesarean delivery: a case-control study. *Am J Obstet Gynecol.* 1993; 168:1358-1363.
9. Cunningham FG, Gant NF, Leveno KJ, et al. *Williams Obstetrics.* 21st ed. New York, NY: McGraw Hill; 2001:646-652.
10. Shipp TD, Zelop CM, Repke JT, Cohen A, Lieberman E. Interdelivery interval and risk of symptomatic uterine rupture. *Obstet Gynecol.* 2001;97:175-177.
11. Oxorn H. *Human Labor and Birth.* 5th ed. Norwalk, Conn: Appleton-Century-Crofts; 1986:541-546.
12. Dewan DM, Hood DD. *Practical Obstetric Anesthesia.* Philadelphia, Pa: WB Saunders; 1997:170-181.
13. Hughes SC, Levinson G., Rosen MA. *Shnider and Levinson's Anesthesia for Obstetrics.* 4th ed. Philadelphia, Pa: Lippincott Williams & Wilkins; 2002:365-367.

AUTHOR

Alexander A. Litwin, CRNA, MSNA, is a staff nurse anesthetist with JLR Medical Group, Maitland, Fla, providing anesthesia care for the Florida Hospital Healthcare System in Orlando, Fla.