

Case report: placenta increta leading to spontaneous uterine rupture in the second trimester

Abstract

Background: Uterine rupture caused by placenta accreta spectrum (PAS) is a rare, life-threatening condition that is rarely included in the differential diagnosis of acute abdomen during the second trimester of pregnancy.

Objective: To report an unusual case of spontaneous pre-labor uterine rupture at 26 weeks' gestation presenting as acute appendicitis clinical recognition and management of this rare pregnancy complication.

Clinical Case: A 32-year-old female at 26 weeks' gestation presented with non-specific abdominal pain. Laboratory and abdominal ultrasound findings were initially interpreted as acute appendicitis, prompting diagnostic laparoscopy. Intraoperative findings showed hemoperitoneum with no appendiceal or bowel pathology. Conversion to exploratory laparotomy revealed a 3 cm defect in the lower posterior uterine wall associated with placenta increta.

Conclusions: Spontaneous uterine rupture secondary to invasive placentation carries high maternal and fetal morbidity and mortality and can easily be misdiagnosed. It may occur at any gestational age, even in the absence of labor. Prompt diagnosis and immediate surgical intervention are critical to achieving favorable outcomes and preventing catastrophic events.

Keywords: uterine rupture, pregnancy, placenta accreta spectrum, placenta increta, acute abdomen

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Background

Placenta increta is a severe variant of abnormal placentation included in the placenta accreta spectrum (PAS), which is a life-threatening condition for both the mother and the fetus.^{1,2} The most severe complication associated with this condition is spontaneous uterine rupture, an obstetric catastrophe associated with high maternal and perinatal morbidity and mortality.³⁻⁵ Uterine rupture is defined as a partial or complete disruption of the uterine wall, which may result in the extrusion of the placenta or fetal components into the maternal abdominal cavity.⁸ It is a rare complication with an estimated incidence of 3 to 5.9 per 10,000 pregnancies. A high index of suspicion is required, especially in primigravid patients or prelabor presentations, which together account for only 1.9–6.4% of cases.⁶⁻⁹

Risk factors of uterine rupture include prior uterine surgery, multiparity, unmonitored use of uterotonics, placental abnormalities, instrumental delivery and limited healthcare access.⁸⁻¹¹ Clinical presentation of uterine rupture is highly variable and non-specific, frequently leading to diagnostic and therapeutic delays. Classic clinical presentation includes abdominal pain, vaginal bleeding, signs of acute abdomen such as hemoperitoneum, hemodynamic collapse, and non-reassuring fetal status (such as fetal tachycardia, recurrent decelerations, severe bradycardia, or intrauterine fetal demise).¹² Common differential diagnoses include acute appendicitis, preterm labor, ovarian torsion, diverticulitis, and urinary tract infections.^{13,14}

Ultrasound protocols such as Focused Assessment with Sonography for Obstetrics (FASO), adapted from trauma FAST protocols, can rapidly identify intra-abdominal free fluid, a positive finding demands immediate diagnostic laparoscopy or laparotomy based on hemodynamic stability.¹⁵ Management includes surgical

intervention, fluid resuscitation for hypovolemic shock, with the surgical approach guided by defect size, hemodynamic status, patient's reproductive goals.⁸ Emergency hysterectomy is reserved for instability, irreparable defect, or fulfilled childbearing desire.¹⁶ Maternal complications include hemorrhage and multiorgan failure,^{2,9} while fetal complications include NICU admission, hypoxic-ischemic injury and death.^{17,18} Given its low prevalence and high mortality, we present a case of spontaneous uterine rupture secondary to placenta increta at 26 weeks' gestation, initially misdiagnosed as acute appendicitis, in which prompt surgical exploration achieved favorable outcomes.

Case presentation

A 32-year-old gravida 2, presented at 26 weeks gestation to the emergency department with sudden-onset abdominal pain. Her surgical history included a lower-segment cesarean delivery three years prior due to transverse fetal lie. Her current pregnancy was well-controlled; a routine 23-week anatomical ultrasound showed appropriate fetal growth, normal amniotic fluid index, and a posterior corporal placenta without structural abnormalities.

Upon arrival to the emergency department, vital signs showed blood pressure 120/80 mm Hg, heart rate 85 bpm and oxygen saturation 95%. Physical examination revealed abdominal hyperesthesia, hyperbaralgesia, deep tenderness in the hypogastrium and right iliac fossa, and positive rebound tenderness (Bumberg's sign). Cervical examination showed a long, closed cervix. Fetal heart rate monitoring showed a reassuring baseline of 140 bpm without decelerations. Laboratory findings included: hemoglobin 12 g/dL and leukocytosis of 11,100/mm³ with 1,800/mm³ band forms.

Abdominal ultrasound confirmed a single live fetus appropriate for 26 weeks, a posterior placenta without retroplacental hematoma and normal amniotic fluid. However, free fluid was identified in the perihepatic and perisplenic spaces, alongside an aperistaltic, dilated bowel loop suggestive of acute appendicitis. General surgery was consulted, and a provisional diagnosis of acute appendicitis was made. The patient underwent diagnostic laparoscopy with general surgery where approximately 800 cc of hemoperitoneum was evacuated. Intra-abdominal inspection revealed no bowel or appendix pathology, but focal hemorrhagic infiltration of the posterior uterine wall was noted (Figure 1). Given the hemoperitoneum and uterine wall findings, the decision to convert to an exploratory laparotomy was made. Upon uterine exteriorization, a 3 cm full-thickness uterine wall defect was identified in the lower posterior segment, 2 cm superior to the uterosacral ligaments. Placental tissue suspicious for invasive accrete spectrum was visible through the defect. A classical (corporal) cesarean delivery was performed, followed by a supracervical hysterectomy with bilateral adnexal preservation due to intraoperative hemodynamic instability. Total estimated blood loss was 2,500 mL. A live male newborn weighing 930 grams was delivered with Apgar scores 6 and 7 at 1 and 5 minutes, respectively (Ballard maturity rating: 27 weeks). The newborn required immediate endotracheal intubation due to bradycardia and poor respiratory effort.



Figure 1 Illustration of posterior uterine wall rupture.

Postoperatively, the mother was monitored in the intensive care unit for 24 hours, remaining hemodynamically stable with adequate urine output and normal kidney function. She was discharged on postoperative day four with a hemoglobin of 8.8 g/dL. Oral iron and vitamin supplementation were initiated, restoring her hemoglobin to 12.6 g/dL at 6 weeks post-discharge (Figure 2).

Histopathological study confirmed a 750 g subtotal hysterectomy specimen featuring hypertrophic myometrium with marked vascular ectasia (uterine atony) and placenta increta involving the uterine isthmus (Figure 3 & 4). Placenta displayed second-trimester chorionic villi with intervillous fibrin deposition involving 10% of the placental disc. The newborn remained in the neonatal intensive care unit on mechanical ventilation for one month, followed by step-down to intermediate care, and was discharged at three months of life. The infant required low-flow nasal cannula oxygen therapy until nine months of age due to bronchopulmonary dysplasia.

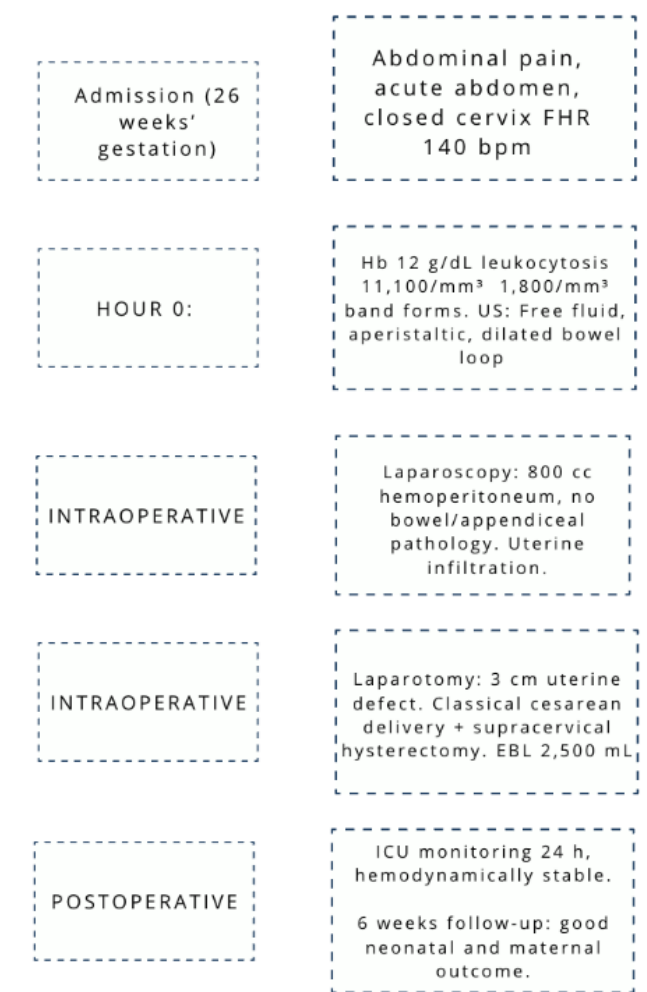


Figure 2 Chronology of symptoms, diagnosis, and interventions.

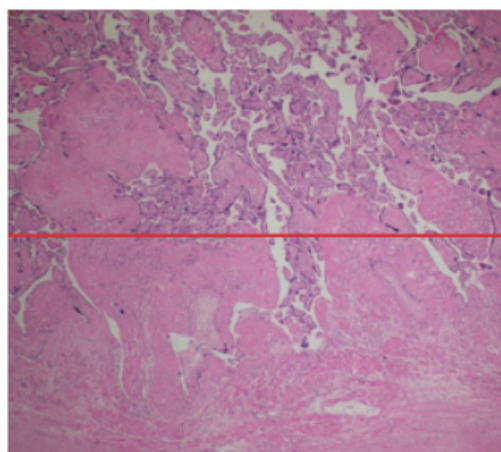


Figure 3 Placenta accreta spectrum (H&E, 4x): Section demonstrating chorionic villi extending through myometrium without intervening decidua.

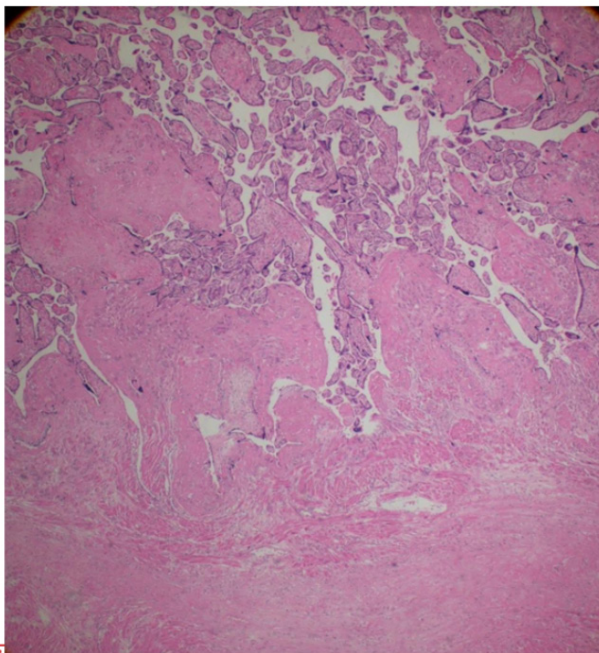


Figure 4 Myometrium infiltrated by chorionic villi.

Discussion

Placenta accreta spectrum (PAS) presenting as an acute abdomen is an exceptionally rare clinical entity, with very few documented cases causing pre-labor uterine rupture during the second trimester. However, as global cesarean delivery rates continue to rise, the incidence of abnormal placentation is increasing. The signs and symptoms of mid-trimester uterine rupture are non-specific, frequently mimicking non-obstetric surgical emergencies and leading to critical diagnostic delays.⁸ In this case, the patient's clinical presentation and initial imaging pointed toward acute appendicitis, prompting initial laparoscopic intervention. However, the intraoperative findings reported, such as uterine wall infiltration and hemoperitoneum prompted immediate conversion to laparotomy, exposing the total posterior defect.

Maternal and fetal outcomes in uterine rupture depend heavily on gestational age, early diagnostic and intervention [SN1.1]. Fetal morbidity in these cases includes severe perinatal hypoxia, low Apgar scores, prolonged intensive care unit admission and neonatal demise. Maternal morbidity includes massive hemorrhage, hypovolemic shock, visceral injury, and multi-organ failure.⁸

Unlike several previously reported mid-trimester ruptures that resulted in catastrophic fetal loss,^{19,20} more recent cases illustrate how quickly these presentations can worsen. A 2025 case report presented a placenta increta case causing uterine rupture at 22 weeks' gestation in a patient presenting with hemodynamic instability and fetal bradycardia.⁷ Similarly, a 2026 case report described uterine rupture at 28 weeks' gestation in a uterus with multiple scars that resulted in fetal demise.¹

Early surgical intervention and specialized neonatal care contributed to a successful outcome for both mother and newborn in our case. Surgical management of uterine rupture secondary to PAS must be individualized. In hemodynamically stable patients with low-transverse defects and a desire for future fertility, uterine repair may be

considered.^{8,16,21} In the presence of adherent placenta, extensive tissue destruction, or maternal instability, subtotal or total hysterectomy is the procedure of choice. In our case, subtotal hysterectomy was made to achieve rapid surgical hemostasis and minimize operative time given her intraoperative instability.

Conclusion

This case exposes the risk of abnormal placentation and spontaneous uterine rupture in patients with a history of prior cesarean delivery. Uterine rupture is a catastrophic obstetric emergency that can occur at any gestational age, even in the absence of active labor. Maintaining a high index of suspicion in pregnant patients presenting with acute abdominal pain - particularly those with prior uterine surgery - is essential for rapid diagnosis, emergent surgical intervention, and the prevention of fatal outcomes for both mother and child.

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None.

Conflicts of interest

The authors declare that there are no conflicts of interest.

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