

Spontaneous Uterine Artery Rupture in a Third-Trimester Pregnancy

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Case Summary

A 30-year-old Nigerian woman, gravida 3 para 1, presented to an urban US hospital at 30 weeks and 1 day of gestation with complaints of nausea, vomiting, and sudden-onset abdominal pain that started at rest. There was no history of vaginal bleeding, intercourse, straining, or abdominal trauma prior to onset of pain. Pain was worse with movement, and she described excruciating pain with car movements en route to the hospital.

She had initiated prenatal care one week preceding the onset of pain. A level II obstetric sonogram prior to presentation showed no abnormalities. Obstetric history included an uncomplicated pregnancy with normal spontaneous vaginal delivery at term and a first-trimester spontaneous abortion. There was no history of previous surgery or drug abuse. Family history was noncontributory. Medical history was notable for iron deficiency anemia.

The patient was hemodynamically stable. Her vitals showed heart rate 97 bpm, blood pressure 89/62 mm Hg, respiratory rate 16 bpm, and temperature 97.9°F.

Physical exam was remarkable for generalized abdominal tenderness without rebound tenderness or guarding. Fetal cardiotocography revealed a normal fetal heart tracing; however, contractions were noted to be occurring every 2 to 3 minutes. Pelvic examination revealed a closed and uneffaced cervix.



A placental abruption was immediately suspected. Bedside ultrasound revealed no abnormalities in the placenta.

Disseminated intravascular coagulation work-up was normal, including a normal fibrinogen. Complete blood count (CBC) showed anemia, with hematocrit of 27.4% and hemoglobin 9.7 g/dL.

A Missed Diagnosis...

The patient received terbutaline for preterm contractions. After a period of observation, the contractions lessened,

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but the patient started to complain of shoulder pain and was treated with acetaminophen. Prior to discharge, the cervix was rechecked and found to be unchanged. Unfortunately, the patient was discharged with the diagnosis of preterm contractions. She was instruct-

CBC was obtained. It revealed a decline in hemoglobin from 8.3 to 7.4 g/dL (normal 11.0-14.5) in a span of 6 hours. An obstetric sonogram showed no evidence of placental abruption; a 2.2x1.5x2.5-cm cyst was seen within the right adnexa, with a moderate amount of complex free fluid seen bilaterally.

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A CT scan of the abdomen/pelvis showed high-density ascites surrounding the liver and spleen and extending into the paracolic gutter.

The patient was tachycardic; however, blood pressure was stable. She was typed and crossed, and blood transfusion was started. An abdominal sonogram performed at bedside revealed moderate perihepatic free fluid. A surgical consultation was obtained; recommendation was to obtain a CT scan of the abdomen/pelvis, which showed high-density ascites surrounding the liver and spleen and extending into the paracolic gutter. After transfusion of 2 units of packed red blood cells, hemoglobin was 7.6 g/dL. Thrombocytopenia was also noted, with platelet count of 134 k/ μ L (normal 145-400).

ed to return to the hospital if contractions resumed or if there was vaginal bleeding, abdominal pain, or decreased fetal movements.

The patient noted minimal improvement of pain at home and developed symptoms of lower back pain and rectal pressure. She was evaluated by her obstetrician at 30 weeks and 2 days' gestation and was sent for further evaluation at the hospital.

On presentation to the hospital she appeared acutely distressed with pain. Her vitals were blood pressure 101/51, pulse 102, respiratory rate 20, and temperature 98.4°F.

Immediate resuscitation with crystalloids was initiated. CBC revealed a hematocrit of 23.2% and hemoglobin of 8.3 g/dL. She had generalized abdominal tenderness without rebound tenderness. Fetal cardiotocography showed a normal fetal heart tracing and contractions every 2 minutes. Cervical length by ultrasound was 2.3 cm without funneling. Urine toxicology was negative. Betamethasone was administered for fetal lung maturity. She was admitted for observation with a presumed diagnosis of preterm labor.

During observation, her pain worsened, and her pulse rate increased to 120.

The patient was taken for an exploratory laparotomy. A vertical midline incision was made. Approximately 2 L of hemoperitoneum was evacuated. A survey of the upper abdomen identified no source of bleeding. The gravid uterus prevented a thorough exploration of the lower abdomen; therefore, a cesarean delivery was performed. She was found to have laceration of left posterior uterine artery. The bleeding vessel was ligated using a #1 Vicryl suture.

Fetal cord gasses were pH 7.34, Pco₂ 36.4 mm Hg, Po₂ 143 mm Hg, Hco₃ 19 mEq/L, and base excess -6. The newborn was resuscitated by the neonatologist. He was initially apneic with no heart rate. He was masked and bagged for one minute, with a heart rate then of 80. He was intubated and epinephrine was administered, with resultant increase in heart rate to 170. Apgars at 1, 5, 10, and 15 minutes were 0, 1, 4, and 8, respectively.

The baby was transferred to the NICU, where he spent 6 weeks for complications related to prematurity: respiratory distress syndrome, necrotizing enterocolitis, and hyperbilirubinemia. He

was discharged home from the NICU in good health. The mother received a total of 7 units of packed red blood cells intra-op and post-op. She had an uneventful recovery.

Etiology

Spontaneous rupture of uterine vessels leading to hemoperitoneum during pregnancy is a rare and life-threatening condition. Steinberg et al have reported a maternal mortality rate as high as 49%.¹ The diagnosis of rupture of uterine vessels is rarely made by imaging modalities. Presenting symptoms of this condition are sudden onset of abdominal pain and signs of concealed bleeding.

Differential diagnoses include placental abruption, ectopic pregnancy, and uterine rupture, along with rare events such as rupture of the liver or spleen and rupture of a splenic artery aneurysm.

Placental perfusion is dependent on total uterine blood flow, which is mainly from the uterine and ovarian arteries. The uterine artery is a main branch of the internal iliac artery, while the ovarian artery is a direct branch of the aorta. The physiology and anatomy of the cardiovascular system change dramatically in pregnancy.

During pregnancy, there is marked hypertrophy of the blood supply to the uterus. Uterine veins also undergo remodeling by numerous factors that include reduced elastin content and adrenergic nerve density, which results in increased venous caliber and distensibility. More than 10% of cardiac output goes to the uterus at term, and veins in the pelvis are engorged. The uteroplacental blood flow increases progressively during pregnancy, ranging from approximately 450 to 650 mL/min near term.

The etiology of spontaneous rupture of uterine artery is unknown. Possible etiologies include arteriovenous malformations and uterine artery aneurysms, varicosities of the uterine vein, decidualization of endometriosis, increased venous pressure in the utero-ovarian vessels during muscular activ-

ity, and straining.^{1,2} The changes that occur to the cardiovascular system during pregnancy are sufficient to withstand pressure fluctuations. This has led to the belief that an inherent defect in the vessels predisposes it to rupture.³ Autoimmune vasculitis and vasculopathy have been associated with spontaneous rupture of other vessels.

Maternal mortality with spontaneous rupture of uterine artery was estimated as 49% in one of the largest published series conducted in 1950.¹ More recent data have shown a dramatic decline. Ginsburg et al reported a maternal mortality rate of 3.6% in cases reviewed between 1950 and 1985.³ This decline in mortality is secondary to advances in resuscitative efforts and anesthesia techniques.

A review of all cases of spontaneous hemoperitoneum in pregnancy over the past 20 years showed that 80% were of

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venous origin, 16% arterial origin, and 4% unknown.⁴ The bleeding site in 90% of cases was either the posterior side of the uterus or the parametrium.⁴

The rarity of spontaneous hemoperitoneum in pregnancy leads to misdiagnosis of the condition. As exemplified by this case, clinicians are hesitant to perform surgical procedures, especially when there is no clear-cut diagnosis. The diagnosis of spontaneous rupture of uterine artery is usually made after extensive explorative laparotomy ne-

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cessitating delivery of a preterm infant, as occurred in this case. Studies have shown that timely diagnosis can prevent adverse pregnancy outcome.⁵

Perinatal mortality was reported as 31% by Ginsburg et al.³ Presenting symptoms include sudden-onset abdominal pain and signs of hypovolemic shock without vaginal bleeding. A decreasing trend in hemoglobin is frequently a sign of concealed bleeding.³ Fetal distress is not a common sign.¹ In the face of an acute abdomen, little preoperative diagnostic studies are required. However, abdominal ultrasonography may reveal intra-abdominal fluid.

Comment and Summary

In order to make this diagnosis, a high index of suspicion is warranted. On review of the case, at initial presentation to the hospital, this patient possibly had intraperitoneal bleeding with diaphragmatic irritation, resulting in the complaint of shoulder pain prior to discharge. The contractions were possibly due to serosal irritation by blood.

Unfortunately, the patient was discharged home. On a subsequent visit, an ultrasound performed showing ascites with a downward trend in hematocrit should have prompted immediate surgical intervention. A lot of time was wasted

obtaining diagnostic imaging that added little information to the overall picture. Immediate surgical intervention may have resulted in less blood transfusion for the mother and perhaps less need for resuscitative efforts for the baby.

In summary, a high index of suspicion is warranted. A timely diagnosis of spontaneous rupture of uterine artery can decrease the mortality and morbidity associated with the condition.

The authors report no actual or potential conflicts of interest in relation to this article.

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