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CASE REPORT

Multidisciplinary management of placenta percreta complicated by embolic phenomena

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ABSTRACT
Hemorrhage and thrombosis are major causes of maternal mortality. This case discusses the management of a woman with placenta percreta complicated by intraoperative pulmonary embolism. A 39-year-old gravida 3 with two previous cesarean deliveries presented at 34 weeks of gestation with an antepartum hemorrhage. Magnetic resonance imaging confirmed placenta percreta. The multidisciplinary group including obstetricians, gynecological oncologists, interventional radiologists and anesthesiologists developed a delivery plan. Cesarean delivery was performed with internal iliac artery occlusion and embolization catheters in place. After the uterine incision our patient experienced acute hypotension and hypoxia associated with a drop in the end-tidal carbon dioxide and sinus tachycardia. She was resuscitated and the uterus closed with the placenta in situ. Postoperatively, uterine bleeding was arrested by immediate uterine artery embolization. With initiation of embolization, hypotension and hypoxia recurred. Oxygenation and hemodynamics slowly improved, the case continued and the patient was extubated uneventfully at the end of the procedure. Computed tomography revealed multiple pulmonary emboli. The patient was anticoagulated with low-molecular-weight heparin and returned six weeks later for hysterectomy. Placenta percreta with invasion into the bladder can be catastrophic if not recognized before delivery. The chronology of events suggests that this may have been amniotic fluid emboli. An intact placenta with abnormal architecture, such as placenta percreta, may increase the risk of amniotic fluid embolus. The clinical findings and co-existing filling defects on computed tomography may represent a spectrum of amniotic fluid embolism syndrome.
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Keywords: Placenta percreta; Pulmonary embolism; Amniotic fluid embolism

Introduction

Placenta percreta with invasion of the urinary bladder is a rare but potentially fatal condition.[1,2] In a 20-year analysis of abnormal placentation, Wu et al. found that the incidence of placenta accreta had increased to 1:533.[3] Prevention of fatal outcome requires attentive preparation. Multidisciplinary management involving obstetricians, interventional radiologists and anesthesiologists has reduced major obstetric hemorrhage and the need for major surgical intervention.[4] Recent evidence has shown that maternal outcome can be improved with the use of novel interventional radiology techniques followed by interval hysterectomy.[5–7]

In this case report we describe the successful multidisciplinary management of a case of placenta percreta complicated by intraoperative pulmonary embolism manifested as profound hypoxia and hypotension. This may have been amniotic fluid embolism (AFE). An in situ placenta with abnormal architecture, such as placenta percreta, may increase the risk of AFE.

Case report

A 39-year-old gravida 3 para 2 presented at 34 weeks of gestation with heavy vaginal bleeding. Until this time her pregnancy had been uncomplicated. She had no significant past medical history but she had previously undergone two uncomplicated low-transverse cesarean deliveries. At the time of presentation she was hemodynamically stable and the vaginal bleeding resolved without medical or surgical interventions. Ultrasound performed at that time identified placenta previa, and given her history of two previous cesarean sections, magnetic resonance imaging (MRI) was performed to exclude placenta percreta. This revealed a lobulated placenta invading the anterior uterine wall with possible

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invasion of the urinary bladder, findings consistent with placenta percreta (Fig. 1). A multidisciplinary group was established including obstetricians, gynecological oncologists, interventional radiologists and anesthesiologists to develop a delivery plan. As our gynecological oncologists would manage any potential urinary bladder investigations or resection, a urologist was not consulted at this stage. The plan was for a scheduled cesarean delivery at 36 weeks of gestation with temporary internal iliac artery occlusion and embolization catheters in place. The abnormal placenta would be left in situ and postoperatively uterine bleeding would be arrested by immediate uterine artery embolization. The patient was not concerned about subsequent fertility; therefore following outpatient treatment with methotrexate she would have a total abdominal hysterectomy 6-8 weeks after delivery.

The patient remained hospitalized until the time of delivery. She was advised to mobilize as little as possible during her in-hospital stay. As she was mobile and not confined to bed she did not receive venous thromboprophylaxis with heparin or compression stockings. She had no further vaginal bleeding. One day before the planned cesarean delivery, she went into spontaneous labor, which did not respond to tocolysis, prompting the surgery to be performed urgently.

The patient had two large-bore intravenous catheters placed and was given antacid prophylaxis before being taken to the interventional radiology suite. She was hemodynamically normal with a hemoglobin of 10.3 g/dL, INR 1.0, PTT 28.9 s, and four units of crossmatched blood available. The radiologist placed catheters in both common iliac arteries with balloon occlusion catheters advanced into each internal iliac artery

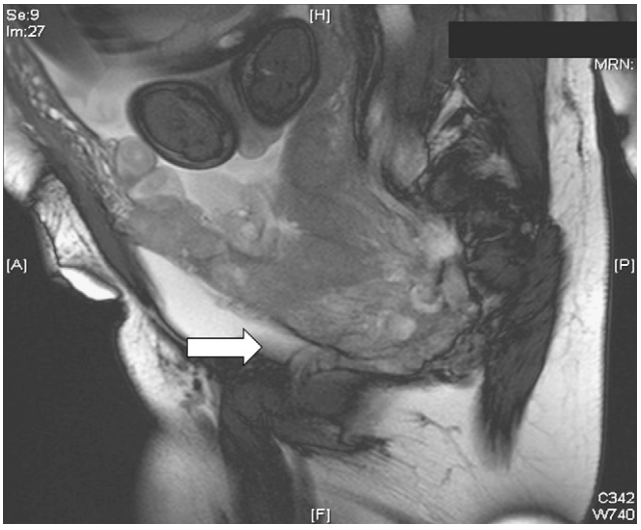

Fig. 1 MRI of the placenta invading the bladder dome (white arrow). The dynamic images from the MRI depict no independent movement between the placenta and bladder, i.e. they move as one.

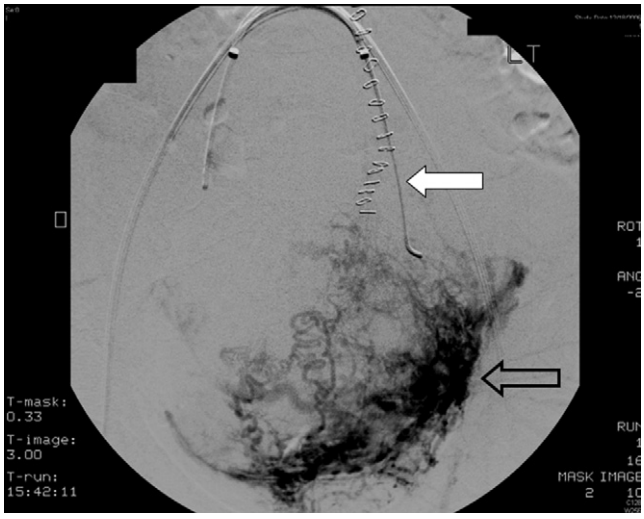

Fig. 2 Placement of arterial catheters in the proximal internal iliac arteries (white arrow) with occlusion/embolization catheters in the uterine arteries. The clear arrow depicts embolization of the left uterine artery.

positioned proximal to the uterine arteries (Fig. 2). A continuous arterial waveform was transduced from the common iliac catheter.

Once the catheters were successfully placed, the patient was taken to the operating theatre. Given our concerns for the length of the surgery and subsequent embolization in addition to the unknown amount of heparin she might receive as part of the interventional radiology, general anesthesia was thought to be the best option. Electrocardiogram, SpO_2, non-invasive blood pressure and bispectral index (BIS) were monitored. Additionally a direct arterial blood pressure was transduced from one of the common iliac arterial catheters. A level-1 Rapid Infuser was connected to a third 14-gauge intravenous catheter. A central venous line was not inserted. Sterile suction tubing was connected to a cell salvage device under the direction of a perfusionist. The patient was placed in the lithotomy position with left uterine displacement to facilitate cystoscopy. She had an uneventful rapid-sequence induction with propofol and succinylcholine and was easily intubated with cricoid pressure applied. Ventilation was maintained with volume-controlled ventilation (500 $mL \times 12$ breaths/min) and FiO_2 1. Anesthesia was maintained with a propofol infusion (50-125 $\mu g \cdot kg^{-1}min^{-1}$) and minimal amounts of sevoflurane to limit the amount of uterine relaxation following delivery. A single 100-μg dose of phenylephrine was required post-induction for mild hypotension.

Before the cesarean incision, placental invasion into the urinary bladder was confirmed by cystoscopy. Shortly after the fundal uterine incision, during delivery of the baby, the patient suffered an episode of acute hypotension (systolic pressure 74 mmHg) and hypoxia

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