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Precipitous delivery complicated by uterine artery laceration and uterine rupture in an unscarred uterus: A case report

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<i>Keywords:</i> Precipitous labor and delivery Uterine rupture Uterine artery laceration Postpartum hemorrhage	Precipitous delivery is associated with rapid cervical dilation and fetal descent. Complications of precipitous delivery can include vascular trauma, uterine rupture, and uterine artery laceration. Uterine artery laceration is a rare complication that can lead to significant postpartum hemorrhage and injury. Careful evaluation for trauma and aggressive resuscitation are critical to prevent maternal morbidity and mortality. This is a case report of a 39-year-old woman, gravida 2 para 1, at 39 weeks of gestation who delivered after induction of labor due to chronic hypertension. Her labor course was precipitous and complicated by uterine rupture and uterine artery laceration with postpartum hemorrhage that required massive transfusion, exploratory laparotomy with a supracervical hysterectomy, and interventional radiology for uterine and cervical artery embolization. This seems to be the first published case report of precipitous delivery associated with uterine artery laceration and uterine rupture. Thorough evaluation after precipitous delivery is critical to decrease maternal morbidity and mortality secondary to uterine artery injury.

1. Introduction

The onset of labor occurs with effective and consistent contractions every three to five minutes [1]. The expectation of labor results in the continued progression of cervical dilation, delivery of the fetus, and delivery of the placenta, otherwise known as the three stages of labor [1].

Precipitous labor and delivery is an abnormally rapid process, defined as fetal delivery within three hours after labor onset with regular contractions [2]. Precipitous labor is rare, with an incidence of 0.1–3% in the United States [2]. Risk factors for precipitous labor include multiparity, chronic hypertension, prostaglandin E-2 induction, low birth weight (<2500 g), and assisted reproductive treatments [2–5]. Despite its low incidence, precipitous labor can lead to life-threatening complications, thus increasing maternal and fetal morbidity and mortality [6]. Complications may include genital tract lacerations, uterine rupture, postpartum hemorrhage (PPH), placental abruption, and prolonged length of hospital stay [2,6,7]. Robust uterine contractions from precipitous labor may damage the uterus and birth canal, leading to

uterine rupture. However, uterine rupture is more common among women with a history of a cesarean delivery [4]. Uterine rupture in women who do not have a history of cesarean section is exceedingly rare, with an incidence of 3.3 out of 100,000 deliveries [8]. In addition, PPH is associated with precipitous labor and harbors significant morbidity and mortality. Spontaneous uterine artery rupture has been connected with PPH, although also rare. Uterine artery rupture with delayed presentation after normal vaginal delivery is extremely rare, as only two cases have been reported thus far [9]. In addition, uterine artery rupture on the left is reported to be more common, as in this case, possibly due to the dextrorotation of the uterus [9]. Uterine artery rupture with precipitous labor has not been described in the literature.

To date, limited data is available on the risk factors, complications, and management of precipitous labor [10]. This case report concerns a patient whose precipitous spontaneous vaginal delivery was complicated by significant genital tract lacerations with uterine artery laceration and retroperitoneal bleeding. She was successfully treated with a supracervical hysterectomy and uterine artery embolization. The aim of this report is to emphasize the need for careful genital tract evaluation

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and low thresholds for aggressive surgical and/or minimally invasive interventions in patients with precipitous delivery complicated by PPH.

2. Case Presentation

A 39-year-old woman, gravida 2 para 1, was admitted at 39 weeks of gestation for induction of labor due to chronic hypertension, managed conservatively without antihypertensives. Obstetrical history included an uncomplicated full-term vaginal delivery three years prior. She had no prior surgical history.

She presented for induction at 39 weeks with an unfavorable Bishop score of 1. Cervical ripening was achieved with 2 doses of misoprostol 50 μ g buccally. She received neuraxial anesthesia, an epidural, for pain management. Her cervical exam rapidly changed from 2 cm to fully dilated with fetal expulsion within 70 min. She delivered a 3100 g female infant, with Apgar scores at 1 and 5 min of 8 and 9, respectively. The placenta delivered intact five minutes after fetal expulsion.

Post-delivery evaluation revealed a second-degree perineal laceration, bilateral sulcal lacerations, and bilateral cervical lacerations. The patient was evaluated in the operating room for optimal visualization. A 4 cm right and a 3 cm left vaginal sulcal laceration, a 3 cm cervical laceration at 10 o'clock, a 3-4 cm cervical laceration at 2 o'clock, and a second-degree perineal laceration were repaired. Vaginal and cervical tissue remained friable, and vaginal packing was placed for tamponade. The patient experienced an initial blood loss of 1000 mL immediately after delivery, PPH was identified, and massive transfusion protocol was initiated. She received 20 units of oxytocin, 250 mg of hemabate, 1 g tranexamic acid, 2 units of packed red blood cells in the immediate postpartum period. She remained hemodynamically stable and was transferred to the recovery room.

In the recovery room, the patient continued to have heavy bleeding through vaginal packing and had unstable vital signs (marked tachycardia and hypotension). She was immediately taken back to the operating room for an exploratory laparotomy. Intraoperatively, the uterus was noted to have a large anterior uterine rupture with a hematoma extending over the anterior lower uterine segment, bladder wall, left parametria and left pelvic sidewall extending into the retroperitoneal space (Figs. 1 and 2). A left uterine artery rupture was noted. A supracervical hysterectomy and left salpingo-oophorectomy were performed to treat both the uterine rupture and left uterine artery laceration. The large retroperitoneal hematoma remained stable at approximately 12 cm. Abdominal and vaginal packing were placed for continued tamponade. She underwent radiologic evaluation to assess for additional vascular compromise and need for interventional radiology procedures. A computed tomography angiogram of the abdomen and pelvis with



Fig. 1. Large retroperitoneal hematoma. A large retroperitoneal hematoma extends anteriorly on the patient's left.



Fig. 2. Anterior uterine rupture.

An anterior uterine rupture is identified to the left of the gloved left index finger.

intravenous contrast revealed active extravasation from multiple locations into a large 10.1 cm \times 12.0 cm pelvic hematoma. An additional incidental finding of atrophic right kidney with enlargement of left kidney was also noted. A pelvic angiogram revealed active bleeding from the left uterine artery stump. The left internal iliac artery, left uterine artery stump, anterior division of right internal iliac artery, and right cervical artery were embolized with hemostatic agents, Gelfoam and occlusion coils. Complete hemostasis and hemodynamic stability were achieved.

Throughout the course of these events, the patient lost an additional 2000 mL of blood, for a total blood loss of 3000 mL. She was resuscitated with an additional 7 units of packed red blood cells, 3 units fresh frozen plasma, 2 units cryoprecipitate, 2 units of platelets, 4 L crystalloids, and 1 g calcium chloride. The patient was transferred to the surgical intensive care unit for close monitoring and recovery. On postoperative day 1, she returned to the operating room for abdominal wall closure. On postpartum day 3, she experienced acute kidney injury and super-imposed preeclampsia with severe features that resolved with surveillance and medical management. The patient was discharged home on postpartum day 9 in stable condition with close outpatient monitoring of hypertension.

3. Discussion

Precipitous labor is theorized to initiate from a combination of abnormally low resistance in the birth canal, abnormal forceful uterine contractions, and/or lack of painful labor sensations or awareness of vigorous labor leading to rapid delivery [11]. Hypertensive disorders are significantly associated with precipitous labor possibly due to concurrent placental abruption or fetal rejection process causing early fetal expulsion [2]. As in this case, the patient had a history of chronic hypertension.

Many maternal-fetal complications in precipitous labor result from an imbalance of labor forces, mainly strong uterine contractions against a non-dilated cervix and a high-resistance birth canal [11]. Uterine rupture, defined as a full-thickness tear of the uterine wall, is a major obstetrical complication related to precipitous labor and is associated with significant morbidity and mortality, including PPH requiring large blood transfusions [8,12]. The overwhelming majority of uterine rupture cases occur in multiparous patients with a history of cesarean delivery or prior uterine surgery [8]. The rupture of an unscarred uterus, as seen in this case, is known as a primary uterine rupture. It is an extremely rare event, with a reported prevalence of 0.005–0.007% [4,7].

This is the first case report suggesting that a precipitous delivery can lead to immediate uterine artery laceration. The uterine artery is a branch of the internal iliac artery and supplies the distal uterus and cervix. Delayed uterine artery pseudoaneurysm (UAP) is another vascular complication of precipitous labor that has been previously reported in literature [12]. UAP is an arterial wall injury in only the tunica adventitia, the outermost layer of the three-layered arterial wall, whereas true aneurysms involve all three layers of the arterial wall [13,14]. In the case of precipitous labor, the strong forces, rigorous contractions, and rapid descent of the fetus can cause local trauma to the vasculature and damage to vessel walls, leading to the formation of UAP [13,14]. Turbulent blood flow predisposes the weakened arterial wall to continual enlargement and ultimately rupture [13,14]. It is possible that a similar vascular pathophysiology caused an immediate uterine artery laceration in the reported patient, leading to massive PPH, notably with retroperitoneal bleeding.

PPH is defined by the American College of Obstetricians and Gynecologists as blood loss of 1000 mL or more after delivery, regardless of mode of delivery [3]. The reported patient experienced a 3000 mL of blood loss with hemodynamic instability. She was successfully treated with aggressive resuscitation, hysterectomy, and prompt IR arterial embolization. Uterine rupture, uterine artery laceration, and PPH may result from precipitous delivery and lead to devastating maternal outcomes. Meticulous assessment in obstetrical-related trauma due to precipitous delivery is critical. Aggressive resuscitation with medical and surgical management should be utilized to improve outcomes. It is important to consider both uterine rupture and uterine artery laceration in severe obstetrical hemorrhage.

4. Conclusion

Precipitous labor is associated with severe complications. Complications of precipitous labor can include uterine rupture and vascular trauma such as lacerations of the uterine artery or its branches. These can lead to massive PPH that is difficult to control. Precautionary assessment of vascular damage to the uterine artery subsequent to precipitous labor is critical to decrease maternal morbidity and mortality. In the event of uterine artery laceration, prompt arterial embolization may be indicated.

Contributors

Stacey Chung contributed to patient management, literature review, and drafted the manuscript.

Khadija Alshowaikh contributed to literature review and editing of the manuscript.

Tamar Yacoel contributed to patient management and editing of the manuscript.

Kanchi Chadha contributed to editing of the manuscript.

Antonia P. Francis contributed to patient management and editing of the manuscript.

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Patient consent

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Provenance and peer review

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Conflict of interest statement

The authors declare that they have no conflict of interest regarding the publication of this case report.

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