Massive hemoperitoneum due to rupture of an unscarred uterus diagnosed postpartum: a near-miss maternal death
Tshering Tamang¹, Tshering Wangden²

¹Faculty of Postgraduate Medicine, Khesar Gyalpo University of medical Sciences of Bhutan, Thimphu, Bhutan
²Department of Obstetrics and Gynaecology, Jigme Dorji Wangchuck National Referral Hospital, Thimphu, Bhutan

ABSTRACT
Spontaneous rupture of an unscarred uterus during pregnancy and labor leading to massive hemoperitoneum is a rare occurrence but with significant morbidity to the mother and fetus. Oxytocin use in labour is a known risk factor for rupture of unscarred uterus. A 21-year-old primigravid woman at 39 weeks of gestation with no known risk factors had labor augmented with an oxytocin infusion; she developed tachysystole and delivered vaginally. Two hours postpartum, pallor, abdominal distension, and a dramatic fall in hemoglobin were noted. An abdominal ultrasound revealed a massive amount of free fluid. At emergency laparotomy the hemoperitoneum was confirmed, and rupture of the posterior wall of the uterus was discovered. Repair of the site of rupture, blood transfusion, and intensive care resulted in a satisfactory recovery. When hemoperitoneum develops in the postpartum period, uterine rupture should be strongly suspected, particularly when oxytocin has been utilized during labor. Judicious use of oxytocin, careful monitoring of labour, and high suspicion of uterine rupture when oxytocin is used play vital roles in preventing the disastrous outcome of maternal death.

Keywords: Induction of labour; Oxytocin; Ruptured uterus; Unscarred uterus.

INTRODUCTION
Uterine rupture is a rare but devastating complication of pregnancy, where the uterine wall tears during pregnancy or delivery. It is one of the life-threatening complications encountered in obstetrical practice with significant morbidity and mortality to the mother as well as the fetus. According to a systematic review of maternal morbidity and mortality by the World Health Organization in 2005, the incidence of uterine rupture reported for unselected pregnant women was considerably lower for population-based (5.3 per 10,000) than for facility-based studies (3.1 per 10,000)¹. A prior cesarean scar has been the most important cause for rupture. According to the American College of Obstetricians and Gynecologists, the risk of uterine rupture is 0.5 to 0.7 percent for women with one prior cesarean and 0.9 to 1.8 percent following two or more previous caesarean who undergo a trial of labor². In their twelve-year study, Miller et al. found that the rate of spontaneous rupture of an unscarred uterus during intrapartum period was approximately 1 in 17,000 deliveries³.

CASE REPORT
In July 2016, a 21-year-old woman, primigravida, at 39 weeks of gestation with an uncomplicated antenatal course was admitted to JDWNRH delivery department. The onset of labor and rupture of membranes were reported as six hours prior to admission. Labour was augmented with oxytocin at 5mlU/minute (10 drops per minute) as per department protocol. The infusion was increased incrementally over two hours reaching 25 mlU/minute (50 drops per minute). Tachysystole and variable decelerations (category II) were noted on the fetal cardiotocography (Figure 1). Oxytocin was immediately discontinued, and intrauterine resuscitation provided. Subsequently, the fetal heart tracing returned to category I (Figure 2). Due to reduced frequency of contractions, labor was again augmented with oxytocin (at 5 mlU/minute). Following this resumption of oxytocin, labor progressed rapidly, variable deceleration reappeared (Figure 3), and with a 10-minute second stage, the baby was delivered. Birth weight was 3.7 kg, and Apgar scores were 5 at one minute and 7 at five minutes. Bag and mask ventilation was initially required for the baby with subsequent observation in the neonatal intensive care unit on continuous positive airway pressure ventilation (CPAP).

In the third stage, the placenta was delivered intact with controlled cord traction. The estimated blood loss was approximately 300 ml. Blood pressure and heart rate remained in the normal range. Two hours later the patient developed severe pallor and a complete blood count showed hemoglobin of 7.1g/dl, which was a drop from 13.1g/dl prior to delivery. Upon examination the abdomen was distended, the uterus was contracted, and there was no vaginal bleeding noted. Ultrasound revealed normal contours to the uterus and a large amount of free fluid. Hemoperitoneum with uterine rupture were suspected.

Emergency laparotomy was done under general anesthesia. A massive hemoperitoneum of approximately 2,000 ml of blood and clot was noted, and active bleeding was discovered at the site of rupture on the posterior uterine wall.
Figure 1. Category – II Fetal heart pattern during oxytocin: variable deceleration

Figure 2. Category - I Fetal heart rate pattern after discontinuing oxytocin

Figure 3. Category- III Fetal heart deceleration during oxytocin before 2nd stage
The myometrial tear measured 5 cm horizontally (Figure 4). The tear was sutured in a continuous locking fashion with absorbable suture (1-0 vicryl), which controlled the bleeding. The adnexa and broad ligaments were normal bilaterally. There was no recognizable uterine anomaly. The abdomen and pelvis were irrigated with warm normal saline prior to closure. Three units of packed red blood cells were transfused intraoperatively.

Postoperatively the patient was closely monitored in the intensive care unit. Four units of fresh frozen plasma and an additional unit of packed red blood cells were transfused. She remained stable without further intra-abdominal bleeding. Repeated complete blood count on postoperative day one and day two were 10.1 gm/dl and 10.9 gm/dl, respectively. Parenteral antibiotics (cefaizolin and metronidazole) were continued for three days. She was given discharge on day four with satisfactory post-operative period. The baby recovered without incident and was discharged with the patient.

**DISCUSSION**

A ruptured uterus is a serious, life-threatening complication for both mother and baby. Spontaneous rupture of an unscarred uterus is a rare event. Risk factors include congenital uterine anomalies, grand multiparity, fetal macrosomia, fetal malposition, labor induction, obstructed labor, uterine instrumentation, instrumental delivery, advanced maternal age, external version, uterine trauma, and abnormal placentation⁴.

In a population-based study in the Netherlands, induction of labour with prostaglandins had caused the rupture in 12 percent of scarred uterus and 40 percent of unscarred uterus⁵. The incidence of spontaneous rupture in an unscarred uterus from the same study was 0.7 per 10,000 deliveries. In an Indian study, oxytocin induction was the major cause for rupture, causing ten cases of rupture occurred from fifteen patients with unscarred uterus⁶. In a case control study, Weimar et al concluded that 44% of uterine rupture could be explained by induction of labor in women with one previous caesarean section undergoing a vaginal birth after caesarean (VBAC)⁷.

Reported cases of hemoperitoneum and uterine rupture have been published in the literature. The other causes of hemoperitoneum in pregnancy and labour described in the literature are rupture of ectopic pregnancies, rupture occurring from pregnancies in congenital uterine anomalies, abnormal placentation such as in placenta percreta, placental abruption with rupture, choriocarcinoma eroding the uterine wall, and erosions of uterine vessels due to endometriosis.

Uterine rupture may have an atypical presentation, with presenting clinical features usually including abdominal pain, vaginal bleeding, maternal hypovolemic shock, and hemorrhage. Fetal heart rate patterns associated with uterine rupture are fetal tachycardia, decelerations in first stage of labour with significant variable deceleration, and terminal bradycardia⁸. Urgent surgical intervention is necessary to salvage the fetus and reduce major fetal morbidities including hypoxia, acidosis, and mortality.

Parity, general condition of the patient, and extent of the uterine rupture determine the choice of surgery. Total or subtotal hysterectomy can be done if fertility conservation is not desired. However, a small rupture can be repaired and the uterus preserved.

In this case, spontaneous rupture of the uterus could have occurred during the intrapartum period due to tachysystole from oxytocin augmentation. The site and the extent of rupture on posterior wall remain unexplained. Here, the indication for oxytocin had been the reduced frequency of contractions. During
the initial variable deceleration (category II), emergency caesarean section was deferred due to effective intrauterine resuscitation. Although the presentation may be atypical, the obstetrician should have a high index of clinical suspicion for uterine rupture when abdominal distension occurs in the post-partum period. Definitely, a cause for the considerable fall in hemoglobin needs to be sought and managed appropriately. This case illustrates the importance of observing the accepted and standard indication for oxytocin infusion in its initiation and incremental dosing. The detection of hemoperitoneum and subsequent management averted a near-miss maternal death.

ACKNOWLEDGEMENTS

Dr. Phurb Dorji and Dr. Debbie Klein for their guidance and expert inputs.

REFERENCES


