

## **A rare case of posterior uterine rupture with neonatal survival during trial of vaginal birth after cesarean section**

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### **Abstract**

*Posterior wall uterine rupture is a rare complication. Trial of vaginal birth after cesarean section (CS) is a predisposing factor especially when associated with augmentation of labor. Here we report a case of intrapartum uterine rupture during the second stage of labor in a multiparous woman trying vaginal birth after previous CS. Emergency laparotomy was done and the baby was saved. Repair of the site of the rupture in layers with complete hemostasis was achieved.*

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### **Introduction**

Rupture of the uterus is a very rare catastrophic peripartum complication associated with severe morbidity and mortality in the mother and the fetus.<sup>1</sup> The chief risk factor for its occurrence is a previous uterine scar especially

previous cesarean section (CS).<sup>2</sup> It is uncommon for an unscarred uterus is to rupture.<sup>3</sup> Vaginal birth after cesarean (VBAC) is considered a safe mode of delivery in women with one previous lower segment CS (LSCS). When the uterus ruptures in women with a previous cesarean section, the rupture generally involves the site of the previous anteriorly placed scar. Rupture of the posterior wall of in women attempting VBAC has also been described, although it's extremely rare.<sup>4</sup>

Outcomes of uterine rupture (UR) depend on the time between diagnosis of rupture and delivery. They include fetal and maternal complications. Fetal consequences are admission to the neonatal intensive care unit, fetal hypoxia or anoxia, and neonatal death. Maternal consequences are hemorrhage, hypovolemic shock, bladder injury, need for hysterectomy, and maternal death. Morbidity and

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mortality following rupture of the uterus depend on the level of medical care available.<sup>5</sup>

Herein, we report a case of posterior wall UR during a trial of vaginal delivery after CS in a multiparous woman with previous one CS followed by 2 successful VBAC.

### **Case presentation**

A 29-year-old gravida 4 para 3 pregnant at gestational age of 39.4 weeks was admitted at our tertiary university hospital early in labor. The patient had regular antenatal visits in her pregnancy with a primary care provider who had discussed modes of delivery with her. Transverse lower segment CS was done 7 years ago due to late fetal deceleration followed by 2 vaginal deliveries (2 VBAC); first one was 5 years ago followed by the second delivery 3 years later. There was no history of other uterine surgery or procedure.

Abdominal ultrasonography had revealed a single living male fetus, placenta at the fundus of the uterus, amniotic fluid was average and estimated fetal weight was about 3100 grams. Vaginal examination revealed the cervix about 4 cm, 50% effaced, amniotic membrane intact, head station at -1 and left occipito anterior position. Mode of delivery with risks and benefits associated with VBAC was discussed with her and she chose vaginal delivery.

Cardiotocography (CTG) revealed a fetal heart rate baseline about 130 beats/minute with 2 accelerations in 10 minutes, with no efficient uterine contractions. Augmentation of labor was

done by artificial rupture of membrane, but still no efficient uterine contractions and failure of labor to progress for 2 hours was present. Therefore, 5 IU oxytocin in 0.9% normal saline intravenous infusion was initiated at a rate of 20 drops per minute.

After that, the patient had a normal labor progression reaching a fully dilated cervix within 3 hours of oxytocin use. However, 15 minutes after full dilatation of the cervix, the patient developed severe abdominal pain and fetal distress with late decelerations up to 70 beats per minute. Her vital parameters were pulse 120 and blood pressure was 90/60 mmHg. There was no scar tenderness. A decision for emergency LSCS in view of fetal distress in the second stage of labor was taken with a high suspicion of UR.

Urgent abdominal exploration done under general anesthesia revealed marked intra peritoneal blood collection. The scar of the previous LSCS was intact and the baby was outside the uterus. After delivering the baby, the surgeon noted a longitudinal defect about 4 cm in length at the lower segment of the posterior wall of the uterus with active bleeding. Repair was done by absorbable sutures into 3 layers till hemostasis was achieved. Estimated blood loss was about 1800 ml. The patient received 4 units of packed cells and 2 units of fresh frozen plasma intraoperatively. An intraperitoneal drain was placed and abdomen was closed after confirming hemostasis. The neonatal Apgar score at 1 min and 5 min was 1 and 4 respectively and a pediatric resuscitation team handled him in the

pediatric care unit (PCU).

Vaginal exploration was done after the procedure to rule out any unnoticed vaginal tear. The anterior cervical lip appeared normal, but the posterior lip was pulled up due to suturing. The patient was stable in the postoperative period. She was discharged on day 4 with hemoglobin level 10.2 g/dl with a healthy male infant after full recovery of his general condition in the PCU.

### **Discussion**

UR is a rare but disastrous complication mainly affecting women trying vaginal birth after previous CS. The true incidence of UR is unknown but in a retrospective cohort analysis of 20,095 women, the incidence of UR in labor with a previous uterine scar was 5.2 per 1000. The incidence increase in those who received prostaglandin for induction of labor to 24.5 per 1000.<sup>6</sup>

In women with a scarred uterus, most cases of uterine dehiscence and ruptures occur at the site of the previous uterine scar due to fibrosis of the myometrium at the site of the scar. In very rare cases, as in our case, the rupture occurs on the posterior wall of the uterus. The hypothesis is that the posterior wall may be excessively stretched and thinned due to the rigid anterior uterine scar that prevents equal stretching, and may cause atypical UR of healthy tissue.

The site of UR in such conditions isn't expected.<sup>4</sup> This is similar to sacculation in which the posterior uterine wall softens allowing the posterior uterine wall to swell like an aneurysm allowing growth of the fetus into the abdomen

with increased risk of UR.

The risk factors for atypical UR include previous unrecognized uterine perforation during dilatation and curettage or during insertion of an intrauterine device. Risk factors are magnified especially in the setting of excessive uterine distension as the myometrium is focally weakened. However, in our case there was no history of any of the previously mentioned procedures and the patient didn't have any uterine intervention except CS.

Factors which predispose to uterine rupture during VBAC are induction and augmentation of labor, prolonged labor, uterine anomalies, endometritis, multiparity, fetal malpresentation or malposition, and morbid adherence of placenta.<sup>7-9</sup>

In cases of posterior UR in patients of VBAC published between 1997 and 2007.<sup>10-14</sup> It is possible that uterine over distension predisposes weak thinned out musculature to atypical UR. Howe and Olah and Figueroa et al. reported two cases of posterior UR with the use of prostaglandin for induction of labor.<sup>10,12</sup>

There are only five described cases in the literature of posterior UR during labor through "healthy" uterine tissue in women with previous CS. However, in the five instances of rupture through posterior uterine wall in presence of anterior scar, only in two cases was labor induced by prostaglandin, suggesting other factors may play a role. In our view the presence of an inelastic scar comprised of fibrous tissue

on the anterior wall prevents even distribution of forces of contraction. As the uterine muscle undergoes retraction during the active phase of labor, the healthy posterior wall may undergo excessive shortening and thinning compared to the inelastic anterior wall, which could have predisposed to rupture. In our case rupture of the posterior wall occurred during the second stage of labor but our patient received oxytocin to augment labor not prostaglandin.

The use of intrauterine pressure catheters (IUPC) may indicate high intrauterine pressure and diagnose UR earlier. However Beckley et al.<sup>15</sup> found that in spite of use of IUPC, uterine ruptures can occur at low pressure due to increased compliance from the previous cesarean scar. In our patient, the uterine contraction and fetal heart rate were monitored by CTG, there was no excessive uterine contraction and fetal bradycardia was the alarming sign for possible UR, so urgent laparotomy was performed that saved the baby.

Fetal distress and neonatal demise resulting from UR are related to placental abruption and hypovolemia resulting in placental hypoperfusion, which develops rapidly as evident from previous instances of posterior rupture. In our patient, prompt delivery resulted in fetal rescue before fetal compromise could develop. Smith et al.<sup>16</sup> found that the overall risk of perinatal death due to UR was 1 in 2100 and UR was three times more likely to result in death of the infant if the delivery took place in a hospital with <3000 births a year (1 in 1300) compared to 1 in 4700 in hospitals with >3000 births a year.

The management of suspected UR during trial of VBAC should be by early surgical intervention to stop the bleeding site and save the fetus' life. Repair of the rupture site with or without tubal sterilization should be carried out as the most suitable intervention especially in women with repairable tears. In advanced cases, hysterectomy could be the only suitable life-saving line of management. Repair of UR carries a risk of recurrence in subsequent pregnancies between 4% & 19%.<sup>17</sup> UR was reported to account for 4% of maternal mortality in our tertiary hospital in a previous study.<sup>18</sup> This high percentage indicates the magnitude of this problem if missed diagnosis or late intervention was performed.

In conclusion, posterior uterine wall rupture in a patient with previous CS scar is very rare, but must be kept in mind as early intervention is the only way to save the mother and fetus.

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