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**Term Delivery Complicated by Uterine Rupture with No Prior History of Cesarean Section or Uterine Curettage Following Oxytocin Use and Arrest in Second Stage of Labor
A Case Report**

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Term Delivery Complicated by Uterine Rupture with No Prior History of Cesarean Section or Uterine Curettage Following Oxytocin Use and Arrest in Second Stage of Labor: A Case Report

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Study Design A

Data Collection B

Statistical Analysis C

Data Interpretation D

Manuscript Preparation E

Literature Search F

Funds Collection G

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Patient: Female, 34-year-old
Final Diagnosis: Uterine rupture
Symptoms: Contractions • spontaneous rupture of membranes
Clinical Procedure: —
Specialty: Obstetrics and Gynecology

Objective: Rare disease

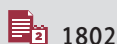
Background: Uterine rupture during delivery in an unscarred uterus may be associated with oxytocin dose during second stage arrest and with underlying maternal factors. This report is of a 34-year-old woman, gravida 5, para 3, with no previous history of cesarean section (CS), who had a uterine rupture at term delivery following the use of oxytocin for second-stage arrest.

Case Report: A 34-year-old Afghani woman, gravida 5, para 3 was admitted at term for delivery. The current pregnancy had been normal and the estimated birth weight was approximately 4000 g. There was no history of steroid treatment or any underlying connective tissue disease, and no history of dilation and curettage. Oxytocin was given as per protocol, starting at 20 ml/h of a dilution of 10 IU/1 L sodium chloride (NaCl). Subsequent labor progress was complicated by arrest of descent in the second stage of labor, necessitating cesarean section delivery. After opening the abdominal wall, a uterine rupture with several large blood clots was discovered, freely floating in the peritoneal space, about 500 ml in volume. The rupture stretched from the left side of the uterine body and down into the thin lower segment. The tissue in this area had diffuse hematoma.

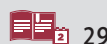
Conclusions: Although uterine rupture mostly occurs in women with previous CS, this report has shown that uterine rupture can occur in pregnancy complicated by arrest in the second stage of labor.

Keywords: Cicatrix • Rupture, Spontaneous • Uterine Rupture • Uterus

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Background

Uterus rupture is an obstetric emergency with possible serious complications associated with significant maternal and perinatal morbidity and mortality [1]. A uterine rupture is the total separation of the uterus' 3 layers. The majority of uterine ruptures take place during a trial of labor following a surgical birth while the uterus is gravid [2]. One uterine rupture is thought to occur per 5000 to 7000 births [3,4]. Around the world, uterine rupture is becoming more common in both scarred and unscarred uteri [5]. The risk of uterine rupture with an unscarred uterus has been reported to be roughly 1 rupture per 10 000 to 25 000 births [6,7].

The etiologies of uterine rupture have changed over time, from obstructed labor and multiparity towards rupture of scar tissue after CS [8]. Although it has been reported in non-pregnant women, uterine ruptures typically happen in pregnant women when the uterus is exposed to trauma, infection, or cancer [9]. However, it can be challenging to diagnose individuals with an unscarred uterus since uterine rupture occurs suddenly in these cases [10]. Unscarred uterine rupture is rare, although it is becoming more common [11].

The majority of uterine ruptures involving unmarked uteri can be attributed to one of the following causes: (1) trauma, (2) a genetic condition linked to uterine wall weakening, (3) a prolonged induction or augmentation of labor, or (4) overstretching of the uterine wall [1]. Dilation and curettage is the greatest risk factor of uterine rupture without previous of CS, especially among women with age of gestation less than 28 weeks [12].

Long-term uterine exposure to oxytocin and other uterotonic drugs increases uterine wall stress and can cause rupture, especially when labor is obstructed [3,13]. Compared to women without a rupture, women who experience a uterine rupture are more likely to have received oxytocin for the induction or augmentation of labor [6].

This report is of a 34-year-old woman, gravida 5, para 3, with no previous history of CS, who had a uterine rupture at term delivery following the use of oxytocin for second-stage arrest.

Case Report

A 34-year-old Afghani woman, gravida 5, para 3 was admitted at term for delivery. She had a history of 1 spontaneous abortion at approximately gestational week 12 and 3 uncomplicated vaginal births, with no history of dilation and curettage. The birth weight was around 3000 g in all 3. Her body mass index prior to the latest pregnancy was 32.9. The current pregnancy had been normal and the estimated birth

weight was approximately 4000 g, with no history of steroid treatment or any underlying connective tissue disease. GDM screening was negative. No cervical ripening was used. To ensure the correct medical history and patient information, an interpreter was used.

The patient's spontaneous rupture of membranes at gestational age 40+3 (0620), and she was admitted with regular contractions. After admittance, the contractions slowed to 2-3 per 10 minutes, and she was 3-4 cm dilated. Eight hours after the water broke (1420), oxytocin was given as per protocol, starting at 20 ml/h of a dilution of 10 IU/1 L NaCl. After 1 hour (1520), the oxytocin infusion had gradually been increased to 60 ml/h. The cardiotocography (CTG) was described as pathological due to complicated variable decelerations, but normalized after change of position and 1 L intravenous NaCl was given. The oxytocin infusion was continued at 80 ml/h with 5 contractions per 10 minutes. The CTG showed early uniform decelerations, and for internal registration a fetal scalp electrode was attached. Three hours and 20 minutes after oxytocin was started (1740) a bradycardic episode occurred and the oxytocin infusion was therefore stopped (1745). After position change, the CTG again normalized. There was no change in the Bishop score vaginally. Another bradycardic episode occurred and again she was placed on the side. Scalp lactate was measured at 1.5 mmol/L.

An epidural was given for pain management, and oxytocin was started again at 20 ml/h after a break of 2 hours and 25 minutes (2010). One hour and fifteen minutes later (2125), the infusion had gradually been increased to 100 ml/h. The CTG was described with uniform early decelerations and was classified as normal. The oxytocin infusion was reduced again to 80 ml/h due to hyperstimulation with 5-6 contractions/10 min. Then, the patient developed fever and was given another liter of intravenous NaCl.

Two hours after the oxytocin infusion was restarted (2210), the progress was satisfactory, she was fully dilated and awaiting the head to descend more deeply into the birth canal. She was given paracetamol and antibiotics (ampicillin), but the fever persisted. The CTG showed gradually increasing tachycardia with decreasing variability. Two hours later (0010), the progress had again halted and there was no change vaginally. The patient had second-stage arrest. At that time, the CTG was classified as suspicious due to tachycardia and it was difficult to determine the nature of the decelerations. The obstetrician on call had been informed continuously and at that point it was decided to perform a CS grade III (the baby must be out within 1 hour after the decision is reported) in spinal anesthesia on the indication of dystocia, fever, and threatening asphyxia. The CS was performed 19 hours and 25 minutes (0145) after the water broke and 11 hours and 25 minutes after the first oxytocin was administered.

Through a horizontal incision of the abdominal wall, we found a uterine rupture with several large blood clots, measuring about 500 ml in volume. The rupture stretched from the left side of the uterine body and down into the thin lower segment (deep cervical laceration). The tissue in this area was more or less disseminated of the hematoma. The uterus was opened in the lower segment and the uterus was digitally widened. As anticipated, the amniotic fluid was blood-stained. The baby was redeemed alive from inside the uterus, but the head was not installed deep in the pelvis. The Apgar score was 10/1, 10/5 and 10/10. Birth weight was 4180 g. The placenta was removed in a normal manner, with no signs of pathology. The bladder and internal genitals were without any pathology. There was a tear in the right side of the uterus, which was closed using Vicryl 2/0. Due to uterine atony, standardized dosages of concentrated Syntocinon (oxytocin) infusion, Tranexamic acid, Methergine (methylergometrine) and Carboprost were administered. In total, the patient was estimated to have lost 1500 ml of blood.

Discussion

This report shows that uterine rupture can occur during second-stage arrest and that it is important to monitor the dose of oxytocin and uterine contractions, even in women without previous CS.

An extensive PubMed search revealed many cases of uterine rupture of an unscarred uterus, most presented in retrospective protocol studies worldwide. The etiology behind unscarred uterus can be traced to several conditions, such as:

- 1) Trauma: Dilation and curettage is one of the major risk factors of uterine rupture without history of CS, especially among those with a gestation age less than 28 weeks [12].
- 2) Uterine wall weakness is associated with genetic disorder: The uterine wall is compromised in disorders such as Ehlers-Danlos and Loey-Dietz, which raises the chance for rupture [14,15].
- 3) A prolonged induction or augmentation of labor, like the case we present. Long-term uterine exposure to oxytocin and other uterotonic drugs increases uterine wall stress and can result in rupture, particularly when labor is obstructed [3,13]. Women with a rupture are more likely received oxytocin to induce or augment labor [6].
- 4) Overstretching of the uterine wall [1]. Conditions such as GDM with macrosomia, polyhydramnios, multiple pregnancy, and uterine anomaly can extend the smooth muscle tissue of the uterus beyond its optimal range [13,16], which is associated with a higher risk of uterine rupture.
- 5) High parity or older maternal age; however, definitions of high parity vary in the literature, with the lowest being parity >3 [17]. However, a retrospective study by Figueiró-Filho

et al did not find any associations between uterine rupture and maternal age or gestational age [18].

- 6) Antepartum fetal death and previous first-trimester miscarriages [6].

In Denmark, there have been 20 identified cases with complete uterine rupture in an unscarred uterus between 1997 and 2008, which were presented in a cohort study published in 2015 [19]. However, the study included women with previous history of laparoscopic surgery, curettage, and bicornate uterus. The study findings suggest that obstructed labor, especially in multiparas with the need for epidural analgesia and augmentation by oxytocin, is significantly related to uterine rupture in women with an unscarred uterus [19].

The knowledge about risk factors is not very accurate due to the rarity of the diagnosis [19,20]. The literature fails to differentiate between complete and partial uterine rupture [20]. The variety of obstetric care around the world and the difference in the use of oxytocin for augmentation (eg, dosage, timing) is not described in the existing literature, and in many of the ruptures presented in the literature, the medical history of the individual women is not described, thus limiting generalizability and comparability.

Historically there has been a rise in uterine ruptures simultaneously with the rise of CS [20]. One could argue that timely diagnosis of uterus rupture is difficult in patients with unscarred uteruses, because clinicians mainly expect ruptures in scarred tissue. Severe maternal and neonatal morbidity and mortality are more often observed in women that do not have a history of uterine scarring [5,11,21,22], including history of curettage [6].

Due to the unexpectedness of the diagnosis, a delay in treatment has been described [22-25]. A study comparing diagnosis intrapartum and delayed postpartum diagnosis identified independent risk factors for delayed diagnosis of uterus rupture to be an unscarred uterus, epidural analgesia during labor, and grand multiparity [26].

Uterine rupture refers to a separation of the uterine layers and of the overlying visceral peritoneum and is often associated with pain, uterine bleeding, fetal distress, and even protrusion or expulsion of the fetus and/or placenta into the abdominal cavity [27]. In developing countries, there is a higher incidence of uterus ruptures and an increased risk of negative maternal and fetal outcomes. This highlights the importance of the availability of qualified, timely antenatal and delivery care services [5,28,29]. However, rupture of the unscarred uterus also occurs in high-income countries; therefore, caution should be exerted when these risk factors are present.

Conclusions

This report has shown that uterine rupture can occur during labor complicated by second-stage arrest and has highlighted the importance of monitoring the dose of oxytocin and uterine contractions. Although uterine rupture is most common

in women with previous CS, an obstetric complication must be considered in all women, regardless of identified risk factors, if correct management and optimal outcome are to be achieved. Awareness of this differential diagnosis is important especially if labor is obstructed in women with higher parity and oxytocin is used for augmentation.

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