Unscarred uterine rupture - case report and literature review

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Summary

Background: Spontaneous uterine rupture is a life threatening event, and the diagnosis is difficult in an unscarred uterus. Many factors can help prevent the catastrophic consequences. *Case:* A 38-year-old multipara in labor was admitted at 39.5 weeks of gestation. Ultrasound suggested a macrosomic fetus but the cervix was well dilated. Labor was immediately monitored. Two hours later, the fetus developed progressive heart rate decelerations. While evaluating the unexplained anomaly, epigastric pain and vaginal bleeding prompted emergency cesarean delivery. The uterine tear was repaired with good evolution but the infant died a few days later. *Conclusion:* The association of multiparity, uterine distension and active labor could be considered as risk factors of uterine rupture in cases of unexplained anomalies in an unscarred uterus, making a catastrophic event preventable.

Key words: Risk factors; Uterine rupture; Unscarred uterus.

Introduction

Spontaneous uterine rupture in pregnant women at term can be a catastrophic event with life-threatening maternal and fetal consequences. The incidence is reported to be one in 16,000 in an unscarred uterus [1]. It is reponsible for 5% of maternal deaths with a peak incidence in the 26-35-year age range [2]. In developing countries, fetal mortality is between 36% and 79% [3].

Uterine rupture is generally more related to a scarred uterus since the frequency of uterine operations (cesarean, myomectomy, hysteroscopy, etc.) is increasing. In a non-scarred uterus the diagnosis is difficult to make, especially when epidural anesthesia is used [4].

Many etiological factors have been described by different authors: grand multiparity, cephalopelvic disproportion, fetal malpresentation, oxytocin stimulation of labor, and anomalies [3, 5-7]. The majority of reported cases of uterine ruptures have occurred during labor or late pregnancy but spontaneous rupture of an unscarred uterus in early pregnancy has been described [8, 9]. Many cases have been reported in primigravida women with no apparent cause and other cases have occurred in a non-laboring uterus with no risk factors [10-12].

The objective of reporting this case of spontaneous uterine rupture is to put into consideration factors which can transform this catastrophic event into a preventable one [3, 13].

Case report

A 38-year-old African woman, gravida 3, para 4, was admitted in July 2003 at 39.5 weeks of gestation for spontaneous active labor. Previously, she had had one normal vaginal delivery for 1,900-g and 2,100-g twins in 1989 and one single full-term normal vaginal delivery in 1998 of a 3,500-g infant. The estimated date of confinement was based on a sonogram at 14 weeks' gestation. The pregnancy had been uncomplicated.

Revised manuscript accepted for publication February 22, 2007

The patient was a healthy woman with no medical or surgical history. She denied the use of any drug, trauma or surgery. She was first consulted at 12 weeks of gestation. Routine maternal serum α-fetoprotein and β-HCG levels were normal at 16 weeks' gestation. The second-trimester ultrasound demonstrated no evidence of any fetal anomalies. A sonogram in the third trimester was suggestive of a macrosomic fetus but a 1-hour glucose screen was normal.

Upon presentation, the patient was already in labor. Her initial vital signs included a blood pressure of 100/60 mm Hg and a normal pulse. She weighed 69 kg. Vaginal examination revealed cervical effacement with 6 cm dilation. Continuous fetal monitoring showed no anomaly and uterine contractions were already strong and regular.

Her initial laboratory studies revealed a hematocrit of 33.4%, platelet count 301,000/mm^3, a normal coagulation profile and negative urine screen.

However, because of a uterine fundal height of 38 cm and vertex presentation at the –2 station, an ultrasound was performed and suggested a macrosomic fetus and fundal placenta.

Since the patient was multipara and already in active labor with a well dilated cervix, an epidural catheter was placed and intravenous oxytocin using a standard protocol started. The cervix was dilated 7 cm and forewater rupture produced normal liquid.

Moderate and regular contractions were achieved (Figure 1). Two hours later, progressive synchronous fetal heart rate decelerations appeared and the oxytocin infusion was immediately stopped (Figure 2). The patient's cervix was dilated 10 cm and the vertex presentation was still at the –2 station. Her vital signs remained stable. While evaluating the unexplained heart rate anomaly, epigastric pain appeared accompanied by vaginal bleeding and fetal bradycardia (Figure 3). The patient was rushed to the operating room for emergency cesarean section. Surgical anesthesia was achieved through the epidural catheter. Immediately after opening the abdomen, a hemoperitoneum was apparent and the baby was extruded into the abdominal cavity through a total vertical anterior uterine scar. The female infant weighed 3,900-g with Apgar scores of 2 and 2 at 1 and 5 min, and cord pH was 6.66. The placenta was then delivered without any difficulty. Further inspection showed uterine rupture extending to the left uterine vessels. The uterine vessel injury was controlled and the uterine tear was repaired in two

Fig. 1

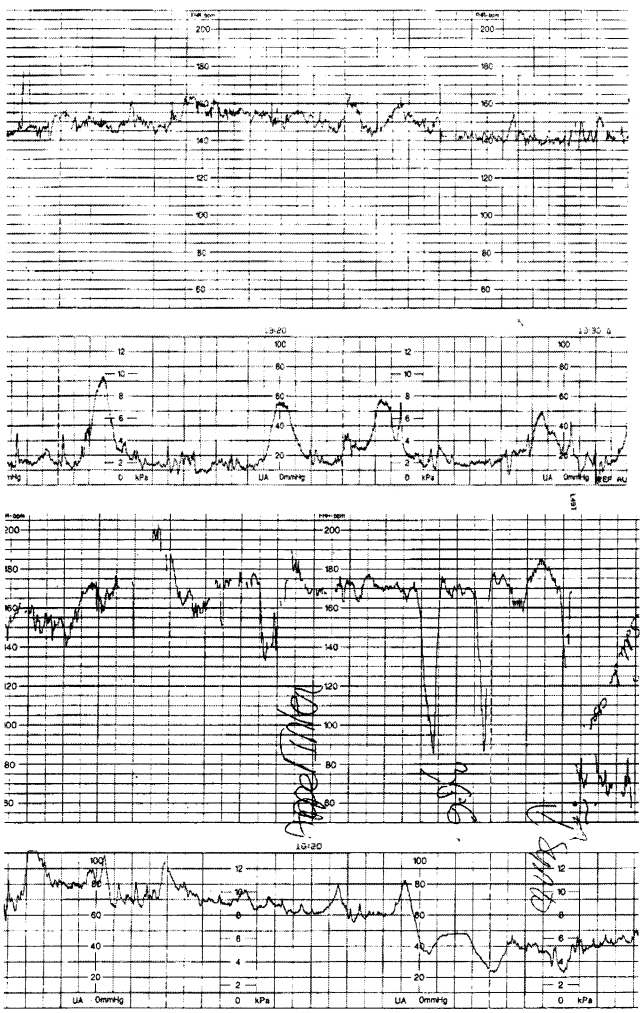

Fig. 2

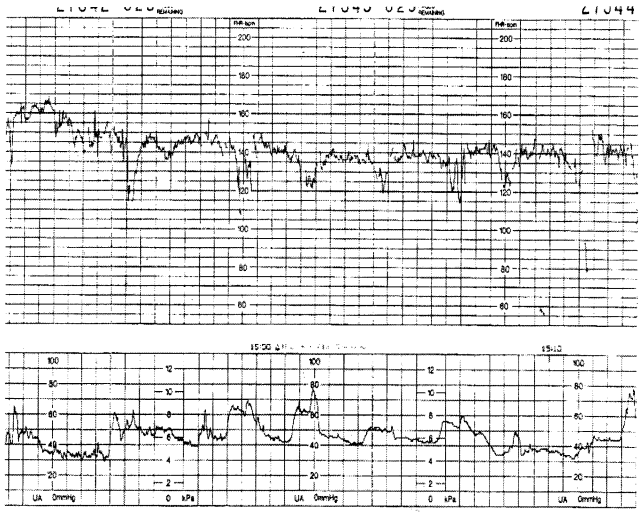

Fig. 3

Figure 1. — Monitoring during labor.

Figure 2. — First unexplained fetal heart rate anomalies.

Figure 3. — Fetal heart rate associated with vaginal bleeding.

layers with Vicryl sutures. Preoperative blood loss was moderate and a blood transfusion was avoided. The patient's postoperative period was uneventful and her hematocrit was 25.8%. She was discharged home on the sixth day with Tardyferon® and Parlodel®, and had an Implanon® contraception.

The infant had been immediately reanimated and intubated. Unfortunately, the evolution was bad. Fetal cerebral death was confirmed by electroencephalogram on postoperative day 1. The baby died on postoperative day 16.

At five week's postnatal follow-up, the patient was well. The pelvic examination was normal and hematocrit was 37.3%.

Discussion

Uterine rupture (UR) usually occurs in a scarred uterus following for example cesarean section, myomectomy or uterine perforation [14-16].

In an unscarred uterus, UR is a rare but catastrophic event. It is a real emergency threatening maternal and fetal life. The reported incidence of this event is 0.03-0.08% [17]. In an attempt to make UR preventable, many authors have described risk factors without confirming the cause-effect role of these factors on uterine rupture.

Exceptional cases of UR in an unscarred uterus have been described with undiagnosed uterine perforation, placenta percreta, adenomyosis, seatbelt accidents, fundal pressure and in vitro fertilization [18-22].

More cases of uterine rupture have been reported in primigravida women [4, 10]. Other authors have described this rare incident in early pregnancy with a high risk of misdiagnosis and higher maternal and fetal morbidity and mortality [8, 9]. Nonetheless increasing parity has been associated with an increased incidence of uterine rupture in many cases [9, 23]. In our case, the patient was effectively gravida 3 with a possible myometrial weakness from repeated pregnancies and uterine overdistension because of suspected macrosomy.

Another very frequent risk factor is labor. In fact, most cases of uterine rupture occur during labor [4, 5, 10, 24]. Even though prolonged uterine stimulation with oxytocin is not seen in modern obstetric practice, caution should be exercised during labor with oxytocin infusion in high parity women. Our patient was indeed in labor with oxytocin infusion for two hours.

Unscarred uterine rupture was also seen after induction of labor with misoprostol [17, 25, 26]. Caution

should also be exercised when using misoprostol, particularly in multiparous women and even in cases of intrauterine fetal death.

In a non-laboring uterus with no apparent previous factors, the situation is more catastrophic [11, 12, 27, 28]. A good outcome in these cases is related to early presentation and prompt clinical assessment and surgical treatment. Abdominal pain and vaginal bleeding are generally the revealing clinical symptoms and fetal heart rate deceleration confirms fetal repercussion.

In our case, such as in other similar cases, continuous cardiotocographic monitoring in labor permitted diagnosis of fetal distress and a rapid surgical delivery of the infant to be undertaken [29, 30]. Emergent laparotomy was performed in all cases and the extent of the surgical procedure depended on the nature of the uterine tear. In patients with an unscarred uterus, as in our case, the tears were mostly longitudinal and complete. Most authors have recommended hysterectomy (77%) rather than repair of the uterus. However, in young women who wish to have more children, there is a predefinite reason to preserve the uterus. Our patient effectively had uterine repair with an uneventful recovery.

Spontaneous rupture of an unscarred uterus, although rare, should be included in the differential diagnosis of any pregnant woman with unexplained abdominal pain, vaginal bleeding or heart rate anomalies. In addition to maternal physical examination, an immediate ultrasound and countinuous fetal heart monitoring during labor are very helpful in evaluating fetal well being.

In our case, the patient was already in active labor and even though macrosomy was suspected the possibility of spontaneous delivery was accepted because of the history of multiple normal deliveries in her past. Although there was no uterine scar, macrosomy and multiparity are alarming enough risk factors when heart rate anomalies appear even if the patient's cervix was completely dilated. Later on, these anomalies were followed by vaginal bleeding and emergent cesarean section with good patient recovery but infant death a few days later.

Spontaneous unscarred uterine rupture is a rare but serious and catastrophic event. A detailed history identifying risk factors such as the association of multiparity and uterine overdistension to prolonged labor may be able to make it preventable. Immediate resuscitation and early surgical intervention – conservative or not – will result in the best outcome for mother and fetus.

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