CASE REPORT

Rupture of the posterior cul-de-sac during trial of labour after caesarean section

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Accepted 21 November 2017

SUMMARY

Rupture of the vaginal wall in unobstructed labour is a rare entity in the developed world. This case report describes rupture of the posterior cul-de-sac in a healthy 34-year-old multiparous woman attempting trial of labour after caesarean section. The woman presented to the labour ward at term with spontaneous onset of contractions. In the second stage of labour, the woman experienced sudden severe abdominal pain, different in character from the contraction pain. Therefore, the baby was delivered by ventouse extraction. As the woman continued to experience severe immobilising abdominal pain during the hospital stay, a CT scan was performed which revealed a haematoma and free fluid at the right side of the uterus. A laparotomy was performed 3 days postdelivery, during which a rupture of the posterior cul-de-sac was found and closed with a continuous suture. The woman was discharged 3 days after laparotomy in good clinical condition.

BACKGROUND

In this case report, we describe the clinical challenge of identifying trauma to the posterior cul-de-sac after delivery. Lack of knowledge of this rare event resulted in a 3-day diagnostic delay and could have led to serious complications including peritonitis, septic shock, intestinal adhesions and possible evisceration of the intestines or even maternal death.

The aim of this article is to create more awareness of the symptoms and risks of posterior vaginal wall or cul-de-sac rupture. Furthermore, we provide insight in the process of diagnosing ruptures of the posterior cul-de-sac, so that possible complications can be prevented.

CASE PRESENTATION

A healthy 34-year-old gravida 3, para 1 woman presented to the labour ward at 40 weeks and 4 days of gestation with spontaneous onset of contractions. The obstetric history included a spontaneous abortion at 7 weeks, complete without intervention. Subsequent pregnancy was a twin pregnancy in 2011 requiring emergency caesarean section for failure to progress in labour at 8 cm dilatation. Two healthy sons were born, and no uterine abnormalities were found during the caesarean. There was no history of other gynaecological surgery.

The woman had regular antenatal visits during the current pregnancy without any abnormal findings. Abdominal ultrasound at 36 weeks of gestation

revealed a single living male fetus in cephalic presentation and normal fetal growth along the 26th percentile. After counselling, the couple opted for trial of labour after caesarean section (TOLAC).

On presentation, the woman had painful contractions every 3–4 min, with membranes intact, 5 cm dilatation and reassuring cardiotocography (CTG). The woman requested pain relief, and after informed consent, remifentanyl was initiated. Concomitantly, amniotomy was performed to enable internal fetal monitoring with a scalp electrode which revealed slightly meconium-stained liquor. After initiation of remifentanyl, oxytocin infusion was started as contractions appeared to decrease in strength and frequency (two contractions per 10 min). Oxytocin 1 cc (5 IU) was added to 49 cc of normal saline. The infusion was started at 0.1 IU per hour and increased according to protocol to a maximum of 0.2 IU per hour until the contractions were deemed satisfactory. Two hours later, the woman progressed to full dilatation with a strong urge to push.

During the second stage of labour, the woman complained of sudden abdominal pain in the fundal region. The pain was not associated with contractions and differed in character from the contraction pain. CTG showed five contractions per 10 min with evident relaxation between contractions. At this stage, uterine rupture or placental abruption was considered. The CTG, however, was reassuring, and on abdominal ultrasound, no signs of intra-abdominal fluid or intrauterine bleeding were seen. Oxytocin infusion was discontinued which did not have any effect on the severity of the abdominal pain. As vaginal examination revealed that the fetal vertex had descended below the spines, the decision was made to terminate labour by ventouse extraction. Following local infiltration of the perineum with lidocaine, the suction cup was placed on the fetal scalp and checked for entrapment of vaginal or cervical tissue. After two tractions and a mediolateral episiotomy, a healthy son was born, birth weight 3740 g, Apgar 9/10, arterial umbilical cord pH 7.19 and base excess –8.9 mmol. The placenta and membranes were delivered by controlled cord traction, and inspection did not reveal any pathology. The episiotomy was sutured under local anaesthesia.

An hour after delivery, the woman complained of dizziness and abdominal pain. Vaginal bleeding increased with a sudden fall in blood pressure to

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To cite: Graafmans LDC, Dijksterhuis MGK, Vos LD, et al. *BMJ Case Rep* Published Online First: [please include Day Month Year]. doi:10.1136/bcr-2017-221149

Figure 1 Abdominal CT image with intravenous contrast. (A) Axial CT image: evidence of an organised haematoma (indicated by an arrow) on the dorsal side of the uterus, surrounded by a minimal amount of free fluid. (B) Coronal CT image: no signs of active bleeding. The myometrium on the right lateral side of the uterus does not light up during the arterial or venous phase (indicated by an arrowhead). The collection of fluid on the dorsolateral side of the uterus has a close relationship with the myometrium possibly indicating a uterine rupture (indicated by an asterisk).

90/50 mm Hg. Abdominal ultrasound was repeated, and there was no suspicion of retained products of conception. However, a trace of free fluid in the abdomen was noticed. Inspection of the vagina wall uncovered a high left vaginal wall laceration. The defect was sutured after which the bleeding ceased. Total blood loss was estimated to be 900 mL.

During the hospital stay, the abdominal pain increased over the course of 3 days and was severe enough to immobilise the woman. During this period, the woman was haemodynamically stable. There were no signs of abdominal guarding, and vaginal bleeding was normal for the postpartum period.

INVESTIGATIONS

Multiple abdominal and vaginal ultrasounds during the hospital stay did not show any abnormal findings. After 3 days, the blood test revealed a sudden drop of 4.83 g/dL in haemoglobin level (14.34 to 9.51 g/dL). Abdominal ultrasound was repeated and

did not show signs of retained product of conception, but did reveal some free fluid at the left side of the uterus. As this was not enough to explain the drop in haemoglobin level, a CT scan was performed. CT showed a haematoma and free fluid at the right side of the uterus, possibly indicating a posterior uterine rupture (figure 1).

DIFFERENTIAL DIAGNOSIS

The woman in our case experienced severe, acute abdominal pain starting in the second stage of labour and increasing in intensity after delivery. Sudden abdominal pain in the last stage of labour could indicate uterine rupture or placental abruption. However, vital signs remained normal, and both contractions and intermittent relaxations of the uterus were present. There was no increased vaginal bleeding, the fetal head was still palpable below the spines and reassuring CTG was present. Based on these clinical findings, uterine rupture or placental abruption seemed unlikely. Ultrasound revealed no free fluid or subplacental haematoma. However, in 50% of the cases, ultrasound is not able to detect a placental abruption or exclude a uterine dehiscence or rupture.¹

During the 3 days after delivery, the woman complained of constant and severe abdominal pain. At this time, the differential diagnosis included uterine rupture and rupture of the cervix or vaginal wall. However, vaginal bleeding remained normal for the postpartum period, the abdomen was not rigid and vital signs remained stable. Repeat ultrasound did show a trace of free fluid, yet not enough to indicate a rupture.

TREATMENT

Based on the CT images, a laparotomy via low transverse incision was performed, revealing old blood in the abdomen. After aspiration of 200 mL old blood, exploration revealed no signs of uterine rupture. On further inspection, a 3 cm horizontal defect was found in the posterior cul-de-sac. The defect was located just below the level of the cervix and could not be closed abdominally due to the large postpartum uterus. Therefore, the rupture was closed using a vaginal approach with a continuous suture. After ensuring haemostasis, the abdomen was closed. Postoperative intravenous antibiotics were given for 48 hours and continued orally for 2 weeks.

OUTCOME AND FOLLOW-UP

Postoperative period was uncomplicated, and the woman could be discharged with minimal abdominal pain 3 days after laparotomy.

Two weeks after delivery, the woman was fully recovered and did not experience any complaints.

DISCUSSION

We report this unusual case of spontaneous rupture of the posterior cul-de-sac in a 34-year-old woman. Vaginal rupture is a rare occurrence in the Western world. A MEDLINE search revealed six publications about vaginal rupture during labour.

Cetinkaya *et al*² attributed a vaginal posterior fornix rupture in a nulliparous woman with intrauterine fetal death at 32 weeks of gestation to the use of misoprostol for induction of labour. Bertaud and Ratto³ described spontaneous rupture of the posterior cul-de-sac during labour. This was probably caused by previous pelvic infection that altered the tensile properties of the birthing canal. Young *et al*⁴ reported a nulliparous woman with known vaginal atresia suffering from a large laceration in the posterior vaginal fornix during spontaneous premature labour at

28 weeks of gestation. It was speculated that the atretic vagina could not withstand the mechanic forces associated with labour. Shantha and Depares⁵ reported a fornix posterior rupture during TOLAC. In this case, labour progressed well, and oxytocin was not used. Eventually, no cause was found for the rupture.

Two other case reports described rupture of the fornix anterior during labour. In one case, a woman undergoing TOLAC experienced rupture of the fornix anterior after amniotomy at 8 cm dilatation. After amniotomy, a fetal bradycardia occurred, and the woman was transported to the operating room where the rupture was detected.⁶ The cause of the rupture was unknown. In the other case, a woman was induced with oxytocin during TOLAC at 39 weeks of gestation. After 4 hours of pushing and CTG with severe variable decelerations, the woman was taken to the operating room, where rupture of the anterior fornix and posterior bladder wall was detected.⁷

These six cases described different causes of vaginal wall rupture, including use of oxytocin (with prolonged second stage of labour), use of misoprostol, previous pelvic infection, vaginal atresia and previous caesarean section. CTG abnormalities were present in almost all cases in the reviewed literature. The current case shared some similarities with the cases described, such as previous caesarean section and use of oxytocin. However, fetal heart rate pattern remained normal, and other risk factors such as grand multiparity, placenta percreta, obstructed labour, endometriosis, vacuum curettage or uterine anomalies were absent.⁸

In retrospect, the use of oxytocin in a woman with a scarred uterus may have contributed to the posterior cul-de-sac rupture. It is common practice in our hospital to start oxytocin as soon as possible when contractions appear to decrease as remifentanyl becomes less effective over time and would result in ineffective pain management during the last phase of labour.⁹ However, considering the woman's obstetric history, it might have been safer to wait a little while longer before starting with oxytocin.

It is difficult to understand why the woman experienced a posterior cul-de-sac rupture instead of a rupture of the old uterine scar. The caesarean section in 2011 was uncomplicated, with no signs of an abnormally thin lower uterine segment or posterior cul-de-sac problems. The growth of the fetus in this pregnancy followed the 26th percentile. Fundal pressure was not performed during labour. Furthermore, the ventouse extraction was started according to protocol with a head below the spines and was uncomplicated.

We theorise that the lower uterine segment was not properly formed during labour. Fibrosis of the old caesarean scar could have caused a stronger mechanical force on the posterior cul-de-sac which might explain the rupture.¹⁰ It could possibly explain why half of the cases with posterior fornix rupture in the literature were in women with a previous caesarean section. Another question that could be raised is whether the rupture of the posterior cul-de-sac could have been seen during a more thorough vaginal inspection after delivery? Although this is possible, the suspicion for a second rupture was low since vaginal bleeding ceased after vaginal wall repair.

In conclusion, this case report discusses the clinical challenges associated with posterior cul-de-sac rupture in unobstructed labour. Symptoms may be limited to sudden onset of abdominal pain that is different in character from the contraction pain. Absence of additional symptoms can lead to delayed diagnosis and put the women at risk for complications, such as peritonitis, thrombosis and intestinal evisceration. Persisting abdominal pain postdelivery should raise suspicion for concealed rupture of the vaginal or uterine wall, especially in the absence of other plausible diagnoses.

Learning points

- ▶ Oxytocin should be used with caution in women with a scarred uterus.
- ▶ Rupture of the posterior cul-de-sac is a rare complication and may be a clinical challenge to obstetricians.
- ▶ Persistent abdominal pain should raise suspicion of concealed ruptures when another explanation is not apparent.
- ▶ A CT scan must be considered in an early phase of acute abdominal pain postdelivery.

Contributors LDCG is responsible for drafting and revision of the article. MGKD and JvB were responsible for treatment of the patient. LDV contributed to imaging and investigations. MGKD and JvB revised the manuscript critically for important intellectual content. LDCG, MGKD, LDV and JvB approved the final version of the manuscript to be published.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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