Clinical Reports

Uterine rupture associated with the use of vaginal prostaglandin E₂ suppositories

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The authors present an obstetrical case of silent uterine rupture due to prostaglandin E₂ vaginal suppositories. Although this complication has been reported in the obstetrical literature, lack of attention to it in the anaesthesia literature, causes us to call this problem to the attention of anaesthetists.

Key words

DRUG: prostaglandin E₂; COMPLICATION: uterine rupture; SURGERY: hysterectomy

Within the past few years, there has been an increasing use of prostaglandin E₂ (PGE₂) vaginal suppositories for the termination of pregnancy in the mid-trimester. Along with its benefits, potential serious side effects are possible from this intervention: one in particular is the risk of uterine rupture.¹⁻⁹ Anaesthetists must be aware of this potential catastrophe.

A case of spontaneous rupture of the uterus following induction of labour for mid-trimester termination of pregnancy by intravaginal PGE₂ suppositories is reported:

Case report

A 32-year-old gravida III, para I, ab I, presented at

26 weeks by dates with chief complaints of periorbital oedema, headaches and blurred vision for about one week. The history of the present illness dated back to approximately two weeks before admission, when the patient complained of shortness of breath and decreased foetal movements. She was seen in the obstetrical clinic and diagnosed as being pre-eclamptic with a blood pressure of 150/105 torr. However, she refused admission at that time.

On admission, at approximately 28 weeks, she presented with uncontrolled hypertension. Past medical history was unremarkable. Physical examination revealed a slightly obese white female in no acute distress, slow in mentation, but oriented to person, place and time. Initial vital signs were blood pressure 190/120 torr, pulse 100/min, respiratory rate 20/min, and a temperature of 37.3 °C (98.8 °F). Periorbital oedema was noted. Chest auscultation revealed scattered rhonchi, without wheezing. Abdominal examination demonstrated a 24-week size nontender fundus. Extremities revealed 1+ to 2+ pitting oedema and deep tendon reflexes were 2+ to 3+ bilaterally. On pelvic examination the cervix was found to be 1 to 2 cm dilated, 30 per cent effaced and the presenting part was undetermined. B-scan ultrasonography did not reveal foetal movement or foetal cardiac activity. Initial laboratory studies showed normal electrolyte values, a haematocrit of 32.3 per cent, platelet count of 114,000 mm³, fibrinogen of 205 mg per cent and PT/PTT of 9.6 sec and 34.2 sec, respectively.

Attempted induction of labour with oxytocin was

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unsuccessful. The blood pressure continued to be elevated in the range of 227/142 torr. An hydralazine infusion was begun, which subsequently stabilized the systolic blood pressure in the range of 180 torr and diastolic pressure in the range of 100 torr. Urine output decreased to about 40–50 ml per hour. Since vital signs were relatively stable, a 20 mg prostaglandin E₂ suppository was inserted.

The patient did well until a few hours later when her blood pressure was noted to be in the range of 100/60 torr. The hydralazine infusion was discontinued. Following an anaesthesia consultation, a central venous line was placed and CVP was noted to be zero and appropriate fluid resuscitation was initiated. The patient was still asymptomatic, apart from mild intermittent lower abdominal pain. Approximately three hours later, she spontaneously delivered a stillborn female infant. Post-delivery, she was placed on a pitocin drip at 60 mu per minute. The blood pressure continued to be in the range of 100/60 torr. Examination revealed a soft abdomen and minimal vaginal bleeding. Post-delivery laboratory studies showed a haematocrit of 12 per cent, platelet count of 64,000 mm³, fibrinogen of 96 mg per cent, PT/PTT of 11.6 and 48.5 sec respectively. At that time, the patient had a tonic seizure. Bimanual examination revealed a large gap in the wall of the uterus which was felt by the examiner to extend into the lateral vaginal fornix.

The patient was immediately taken to the operating room and a total abdominal hysterectomy and right salpingo-oophorectomy was done under general anaesthesia. She was given six units of packed red cells, eight units of cryoprecipitate, and ten units of platelets. Postoperative laboratory studies showed a haematocrit of 25 per cent, fibrinogen of 145 mg per cent and platelet count of 174,000 mm³. The remaining postoperative course was essentially uneventful. She was discharged on the thirteenth day.

Prostaglandin E₂ (PGE₂) is a powerful oxytocic which reliably initiates labour even in the presence of an "uninducible" cervix. Prostaglandin is prone to increase intrauterine pressure to a level beyond that of normal labour¹⁰ but with a lag in cervical changes. The two most common traumata reported following prostaglandin administration for therapeutic abortion are either cervico-vaginal fistulas or "Dührssen incision"-like lateral tears.^{11–13} In this

case, even within the minimal dosage there was a rupture in the right lateral aspect of the uterus. At laparotomy, there was no indication of any congenital weakness of the uterine wall. Hence, it is reasonable to assume that the mechanism leading to the rupture was intense and prolonged uterine contractions combined with a rigid cervix. Even though PGE₂ suppositories can be extremely useful for the management of intrauterine foetal death, this potential catastrophic event is a rare, but possible, complication.

In our case, none of the classical signs or symptoms of uterine rupture¹⁴ namely vaginal bleeding, peritoneal irritation and unstable vital signs were present until after delivery of the placenta. This absence of clinical symptomatology might be misinterpreted in the face of the sympathectomy associated with regional analgesia and thereby lead to a medical catastrophe. Thus, the additional monitoring features of an external tocodynamometer for monitoring uterine contractions, frequent abdominal palpation, placement of an intrauterine pressure catheter, and continuous monitoring of the patient's vital signs are important in the diagnosis of early uterine rupture in patients in whom prostaglandin PGE₂ has been used.

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Résumé

Les auteurs rapportent un cas de rupture utérine silencieuse attribuable à l'utilisation d'ovules vaginaux à la prostaglandine E₂. Cette complication est plus fréquemment rapportée dans les publications obstétricales que dans la littérature d'anesthésiologie. Aussi, les auteurs croient-ils important d'attirer l'attention des anesthésistes sur ce sujet.