

## Spontaneous Hemoperitoneum in the Immediate Post-Partum Caused by Endometriosis and Labour

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### Introduction

Spontaneous hemoperitoneum (SH) during pregnancy, although uncommon, may arise from a wide variety of causes. It has been reported to occur during pregnancy, labour, and the early postpartum period. Though advances in anaesthetic, resuscitative, and operative techniques have played a role in lowering the maternal mortality rate from 49% in 1950 to 3.6% in 1987, the perinatal mortality rate in women with SH remains high at 36% [1,2,3]. Prompt diagnosis and intervention are critical for improving outcomes in these patients. We hereby present a case of SH in the early post-partum period, caused by the tearing of an adhesion. This adhesion being due either to endometriosis, to the aftereffects of a previous surgical procedure or both.

### Case Report

A 37-year-old woman, gravida 1, para 1, was admitted for spontaneous labour after 37 weeks of gestation. She was known for suffering from severe endometriosis. Five years before the patient was presented to us, she underwent two laparoscopic procedures: one was an ovarian cystectomy for endometrioma, the other was the excision of an utero-sacral endometriosis implant (AFS III score). A second look laparoscopy revealed residual endometriosis type 1. Then, three years before she entered our service, an IRM imaging was performed because of the reappearance of clinical symptoms; it revealed other digestive, ovarian and parametric lesions.

Because of her primary infertility, she had obtained a pregnancy by IVF treatment. Her antenatal course had been uneventful. The labour was spontaneous, with peridural analgesia. She delivered a girl at 6pm, weighing 3670g, forceps were used because of an abnormal cardiofoetal rhythm, but there were no other immediate concerns. During perineal tear and episiotomy refection, a hypotension occurred (blood pressure 65/45 mmHg). It was well corrected by crystalloid. The other patient's vital signs were stable (pulse 87beats/minute, temperature 37.2°C). She did not mention any pain and she had no vaginal bleeding. Haemoglobin was evaluated at 9.5g/dL with the Hemocue® system (Haemoglobin last month at 11g/dL).

During the following hours, she called for abdominal and shoulder pains: the uterus was tonic, hemodynamics were stable and paracetamol analgesics soothed the patient immediately. Early in the morning, at 07am, the pain got stronger, the volume of uterus was abnormal and the patient suffered from hypotension without vaginal bleeding. Initial imaging with ultrasound showed a large amount of fluid and blood inside the abdominal cavity extending to the upper abdomen. The pelvic organs appeared normal considering her postpartum status. No obvious source of bleeding was identified. Hemoglobin was measured at 8g/dL. She was immediately transferred to the operating room. Under general anesthesia, the uterine revision was negative and did not reveal any uterine rupture. The examination of vagina and cervix did not show any active bleeding.

A laparoscopic exploration revealed an enlarged postpartum uterus and 2L of blood and fresh blood clots dispersed throughout the abdomen and pelvis. Because the uterus was too voluminous, and because of uncontrolled bleeding, we converted to laparotomy.

After the evacuation of the blood, an initial inspection of the uterus revealed it to be intact. The liver, the spleen and the whole upper part of the abdominal cavity were safe. Then, we observed active bleedings. One in the posterior uterus wall and in the mesosigmoid, whether it was secondary to endometriosis or whether it was an aftereffect of an adhesion tearing; another one from a part of the right uterine artery subsequent another adhesion ruptures. The bleeding stopped immediately with interrupted sutures. The exploration revealed endometriosis digestive implants.

The blood loss was evaluated at 2.5 L. Three units of packed red cells and PFC were transfused perioperatively.

She was stable the following days and was discharged home 10 days later in a satisfactory condition.

### Discussion

This report relates an uncommon case of SH in the early post-partum period resulting from endometriosis and post-operative aftereffect adhesion tearings. In this case, it might result from labour contractions, from expulsive effort, from progesterone's fall and/or from the traction made by a vaginal operative delivery using spatulas.

SH during pregnancy is a rare but dramatic complication. It is defined as an unprovoked intraperitoneal bleeding that may be idiopathic or related to many causes such as uterine rupture, placental abruption, spontaneous ruptures of uterine varicosities, placenta percreta, liver or splenic rupture in preeclampsia, trauma, ectopic pregnancy and other rare causes such as a spontaneous rupture of a splenic artery aneurysm, or a ruptured appendix. The most common cause of SH is spontaneous utero-ovarian vessel rupture in pregnancy [1-12]. SH in pregnancy occurred before labour for 61%, intrapartum for 18% and puerperal for 21%. The majority of cases reported occurred in the third trimester (29% before 33 weeks of gestation, 39% between 33 and 37 and 32% at term for Ginsburg).

In other cases, active bleeding is described from endometriotic lesions with or without tears [4,13,14]. Inoue et al suggested that endometriosis may be involved in the rupture of these vessels [5]. They thought that chronic inflammation due to endometriosis may render utero-ovarian vessels more friable, or the resultant adhesion may give further tension to these vessels when the uterus is enlarged during pregnancy. Endometriosis seems to be a major risk factor of SH in pregnancy [2,5,12,14]. Brosens et al. [2] in their series noted that 52% of all patients suffering from SH during pregnancy reported

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in the last 20 years suffered from known or unknown endometriosis. Reviewing the literature, we can differentiate various major etiologies of spontaneous hemorrhage in endometriosis during pregnancy, which are explained by physiopathology: utero-ovarian vessel ruptures and bleeding endometriosis implants [14]. An alternative explanation supposed by Brosens et al. [2] may be the involution of decidualizing ectopic endometrium during pregnancy secondary to the fall of progesterone during the post-partum period [12,15,16]. Passos et al, as Cole et al. [6,7] explained that the presence of surgical scars related to previous laparoscopic surgeries may have further weakened vessel walls or provided points of fixation that could have been torn by the normal contractile process of the uterus. In the three cases, described by Zhang et al. [17] patients had had previous surgical endometriosis care.

In our case, SH is secondary to endometriosis-like or previous surgery adhesions tears. We supposed that the endometriotic and surgical history of the patient, the fall of progesterone, the strength of the expulsive efforts and the spatula traction may have torn the tight adhesion between the uterus and sigmoid due to endometriosis. Even if not described we can suggest that a uterine massage, as fast involution uterine after birth can tear these same adhesions.

For some authors, IVF treatment in similar cases may be an additional risk factor [2,17]. However, all Brosens's patients suffered from endometriosis which is a major risk factor of SH as we have said before. We cannot conclude for the IVF treatment responsibility.

The diagnosis remains difficult and is often made in per-operative because of unspecific symptoms such as sudden abdominal pain without obstetrical etiology found (such as described before) and hours or days later, hypovolemic collapse. The symptoms can be confounded with typical complaints of normal pregnancy as contractions. In our case, the patient was transferred to the operating room 12 hours after the onset of the first symptoms. Some maternal deaths have been described in such pathologies [8].

As we report here, we have to keep in mind that endometriosis is a risk factor for SH in pregnancy.

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