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Case Report

Spontaneous hemoperitoneum in pregnancy: report of two cases

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ABSTRACT

The condition known as spontaneous hemoperitoneum in pregnancy (SHiP) is characterized by an accumulation of fluid in the abdominal cavity and a vague collection of symptoms. Specifically, intrauterine pregnancy increases the chance of death for both the mother and the fetus, making SHiP a potentially lethal illness when it coexists. Here, we discuss two cases of spontaneous hemoperitoneum in pregnancy that resulted from placenta accreta spectrum and endometriosis, and happened in the second and third trimesters, respectively.

Keywords: Spontaneous hemoperitoneum in pregnancy, Endometriosis, Placenta accreta spectrum

INTRODUCTION

Pregnancy-related spontaneous hemoperitoneum, or SHiP, is an uncommon but hazardous disease associated with high rates of morbidity and mortality. The risk of SHiP is elevated by endometriosis, especially in the third trimester of pregnancy. The development of tissue outside the uterine cavity that resembles endometrial tissue is a characteristic of this chronic inflammatory illness.[1] The spontaneous rupture of utero-ovarian arteries linked to endometriosis could be caused by three factors: adhesions may impose tension on these vessels when the uterus grows during pregnancy; decidualization of endometrial foci during pregnancy may result in uteroovarian vessel rupture; and endometriosis-induced chronic inflammation may cause uteroovarian vascular leakage.[2,3] Poor uterine decidua development in the lower uterine segment is the cause of placenta accreta spectrum. The abnormal invasion of placental tissue into the uterine wall with penetration into adjacent organs like the rectum or bladder. The clinical signs of placenta increta, which include bleeding, uterine rupture and inversion, and bladder invasion, are related to the location of placental implantation, the extent of myometrial invasion, and the breadth of improperly adherent placental tissue.[4] The history of uterine surgery, including as caesarean sections (CS), dilatation and curettage, myomectomy, and endometritis, is known to be linked to placenta percreta.[5] Repeated CS6 is the most common risk factor for aberrant placentation and placenta percreta. There could be a risk to the life of both the mother and the fetus.

CASE REPORTS

Case 1

A 30-year-old woman with a known case of stage 4 endometriosis and frozen pelvis, who became pregnant with twins after undergoing her fourth IVF cycle, complained of repeated episodes of vomiting and abdominal pain. She used insulin and had overt diabetes mellitus on OHA. A decrease in hemoglobin (8.6) and aberrant LFT (SGOT-60, SGPT-130, SERUM ALBUMIN-2.5) were found during routine tests. On ultrasound, the fetus's growth and the abdominal organs were normal, and there was mild to moderate ascites. When she was brought in for an emergency ascitic tap, hemorrhagic fluid was discovered. After receiving magnesium sulfate and betnsaol prophylaxis, she was scheduled for an emergency laparotomy and caesarean

section under general anesthesia due to her hemoglobin level falling, hemorrhagic ascitic tap, and enlarged belly.

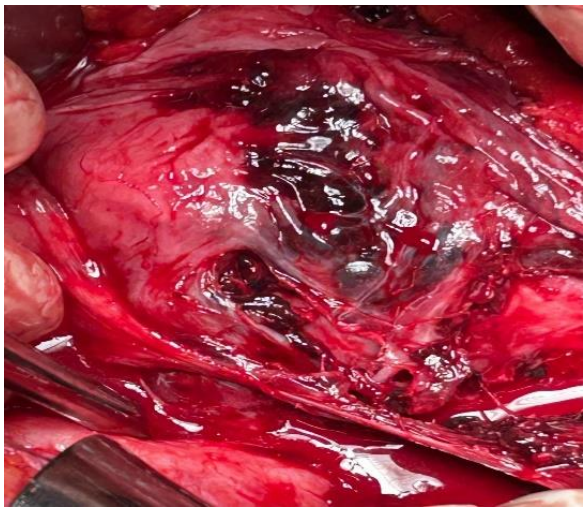

Figure 1: Intra op-visualization of multiple endometriotic deposits over the anterior surface of uterus with bleeding spots.

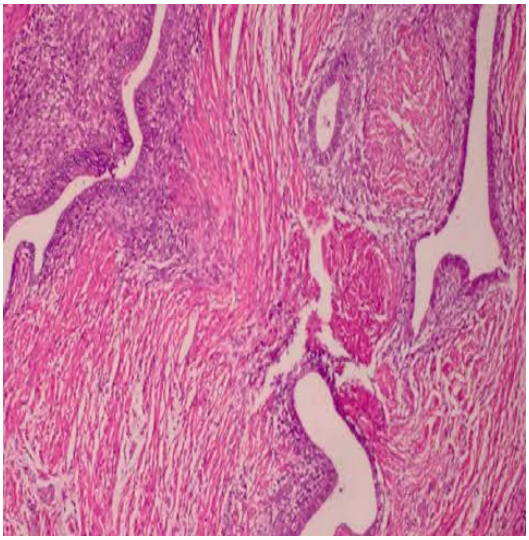

Figure 2: HPE showing decidualized endometrial tissue with continuous infiltration from the peritoneal surface to a depth of more than 5 mm.

It involved the live delivery of two premature female infants weighing 1.12 kg and 1.1 kg via caesarean surgery. During the procedure, there was a large amount of hemoperitoneum (about 900 ml), several leaking endometriotic deposits on the fundus, sigmoid, and ileocecal junction, as well as numerous pelvic organ adhesions. Endometrial deposits adhered to by the right tubes and ovaries inside the tuboovarian mass drip points. Excellent cauterization hemostasis was seen. She did a good job of maintaining her hemodynamic stability. Following a pathological examination of the removed tissue, decidualized endometrial tissue was found, displaying a continuous infiltration from the peritoneal surface down to a depth exceeding 5 mm. This led to the identification of endometriosis with deep infiltration and the preference for endometriosis-related spontaneous hemoperitoneum during pregnancy.

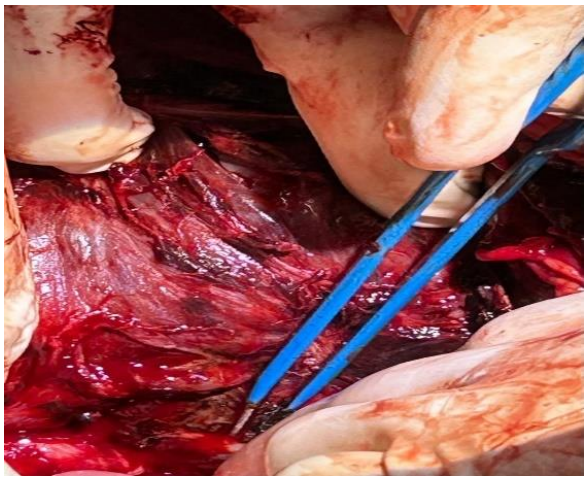

Figure 3: Cauterisation of the endometriotic deposits.

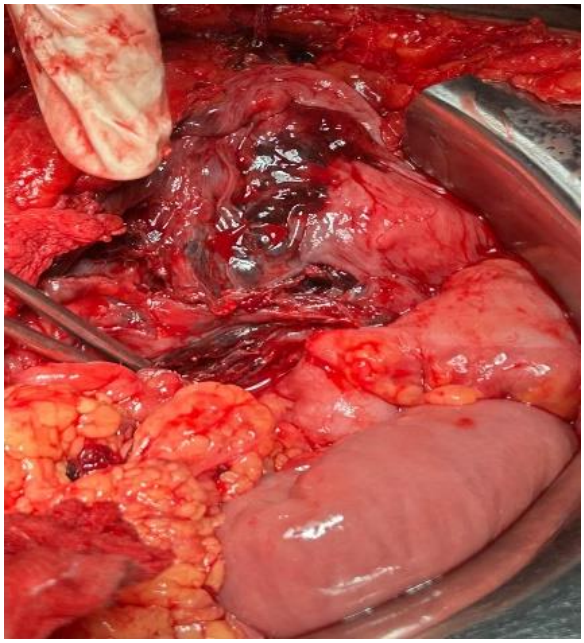

Figure 4: Intraop showing multiple oozing spots, with dense pelvic adhesions.

Case 2

A 26-year-old, G2P1L1, 19 weeks, and prior LSCS (breech presentation) reported to the hospital one day with a scan report indicating a significant volume of hemorrhagic fluid. She reported having lower stomach ache and being exhausted for a single day. She was hypotensive and tachycardic upon arrival at the ER (90/60 mmHg, 110 bpm, 94%); scan report: SLIUG gross oligohydramniosis lasting 18 weeks plus 6 days, accompanied by a large volume of hemorrhagic fluid in the peritoneal cavity. She was scheduled for a last-minute

laparotomy. When intraoperative blood clots of 1500 ml entered the peritoneal cavity, they were evacuated. Lower section was seen extending from anterior surface to left of broad ligament, tightly adhering to bladder with placenta invasion. There are bleeding points. There was a dead fetus that emerged from the abdominal cavity. Following B/L internal iliac artery ligation, subtotal hysterectomy was performed. 3.5 ml of blood were lost in total. Bladder integrity examined abdomen sealed in layers and drained intraperitoneally was supplied once total hemostatis was achieved. Six pints of PRBC, five FFP, two platelets, and two albumins were given. She was extubated and moved to a face mask with stable vitals on the second postoperative day. On day 4, the drain was removed with little serous fluid. The foleys catheter was left in place for 20 days. On day nine, her condition improved and she was released. Suggested HPE report was of placenta percreta.

DISCUSSION

A unique challenge is endometriosis-related SHiP, which is often associated with decidualized ectopic endometrial involution during pregnancy. Progesterone resistance and endometriosis interact intricately to cause decidualization to revers, proinflammatory cytokine release, matrix metalloprotease release, and apoptotic factors to release. These events set off a cascade of events that culminate in bleeding. Unknown, venous, or arterial blood loss are the three categories. The spontaneous rupture of utero-ovarian vessels associated with endometriosis may be caused by three factors: adhesions putting strain on these vessels when the uterus enlarges during pregnancy; decisionalization of endometrial foci during pregnancy leading to utero-ovarian vessel perforation; and chronic inflammation caused by endometriosis causing uteroovarian vessel leakage.[7] This case highlights the need for further investigation into the relationship between spontaneous hemoperitoneum during endometriosis, pregnancy, and progesterone resistance.[8] The abnormal growth of trophoblastic tissue into the uterine wall is referred to as placenta accreta spectrum (PAS). A placenta that adheres inappropriately, commonly referred to as a creta or placenta adherenta, is classed as a grade 1 placenta accreta. There are significant regions where the placental tissue is directly connected to the surface myometrium in this case since there is no decidua between the myometrium and the placental villi. When placenta improperly invades the myometrium without apparent placental tissue penetrating the uterine serosa, it is referred to as placenta increta, or grade 2. The grade 3 sign, placenta percreta, is further classified into three subgroups based on the way the placenta invades the bladder (grade 3b), the other pelvic organs (grade 3c) or the uterine serosa (grade 3a).[8] Many explanations have been proposed to explain the genesis of PAS. The most prevalent one has to do with an alteration in the endometrium myometrium interface that stops the normal development of trophoblastic tissue near a uterine scar. A modest lesion of the decidua and superficial muscle fibers to a substantial and deep myometrial defect can be caused by a uterine scar.[9-11] The decidua plays a critical role in trophoblastic invasion regulation. In the absence of successful re-epithelialization, trophoblastic tissue proliferates invasively in the scar region.

CONCLUSION

This example highlights the complexity of spontaneous hemoperitoneum during pregnancy, particularly in cases of advanced endometriosis. The encouraging results provide important information about this rare but dangerous illness and emphasize the necessity of prompt and careful management. In addition to highlighting the necessity of a multidisciplinary approach, the study deepens our knowledge of the challenges posed by spontaneous hemoperitoneum during pregnancy. The potentially fatal signs of this illness first emerge in the early stages of pregnancy. In situations since they are the main candidates for PAS, patients who have already undergone a caesarean section should be given particular consideration. It is imperative that healthcare providers recognize this sickness and do not minimize it. Problems with PAS should be detected early. Since SHiP presents a significant risk to both the mother's and the child' lives, prompt diagnosis and care are crucial. Knowledge regarding SHiP and associated risk factors-such as pelvic endometriosis, may improve outcomes for the mother and the fetus by facilitating diagnosis and accelerating the therapeutic process.

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