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Secondary Refractory Postpartum Hemorrhage From Deep Infiltrating Endometriosis Of The Posterior Uterine Wall And Bowel: A Case Report

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Abstract
Background: Deep infiltrating endometriosis (DIE) involving the posterior uterine wall and bowel serosa is an uncommon but under-recognized cause of severe, difficult-to-localize hemorrhage during cesarean section. Vascular fragility and dense adhesions in these areas can precipitate coagulopathic bleeding that is refractory to conventional hemostatic techniques.
Case Presentation: A 24-year-old primigravida with a history of significant dysmenorrhea underwent induction of labor at 40+4 weeks for post-datism, followed by emergency lower-segment cesarean section (LSCS) for a non-reassuring non-stress test (NST), with a healthy neonate delivered. Intraoperatively, the patient was found to have ovarian endometriosis with rectosigmoid colon involvement, and severe endometriosis over the posterior uterine surface, from which profuse, diffuse, non-localizing bleeding began at the time of uterine closure. Hemostatic sutures were placed and a drain left in situ. The patient developed a rapid hemoglobin drop (11 to 8.2 g/dL), thrombocytopenia (2.5 to 1.5 lakh/μL), and oliguria, prompting transfer to the medical intensive care unit (MICU). Imaging confirmed a 1-liter hemoperitoneum, necessitating re-exploration with massive transfusion (packed cell volume [PCV], fresh frozen plasma [FFP], random donor platelets [RDP], and cryoprecipitate). At re-exploration the uterine bed was found to be dry, with the ongoing source identified as oozing from bowel-surface endometriotic implants; hemostasis was achieved with topical hemostatic agent and temporary abdominal closure was performed. A second re-exploration on day 3, after platelet transfusion, confirmed complete hemostasis and definitive closure was completed. GnRH agonist therapy was administered for endometriosis, and the patient was managed on broad-spectrum antibiotics (vancomycin and piperacillin-tazobactam) in the MICU. She recovered well and was discharged on day 11.
Conclusion: Endometriosis involving the posterior uterine wall and bowel serosa should be recognized as a potential cause of refractory, non-localizing intraoperative and postpartum hemorrhage. A staged, damage-control approach including temporary abdominal closure, correction of coagulopathy, and planned re-exploration permitted definitive hemostasis and good maternal outcome without further surgical intervention.
Keywords: *Deep infiltrating endometriosis; postpartum hemorrhage; cesarean section; damage control surgery; hemoperitoneum; coagulopathy; temporary abdominal closure*

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I. Introduction

Postpartum hemorrhage (PPH) remains a leading cause of maternal morbidity and mortality worldwide. While uterine atony, retained products of conception, and genital tract trauma account for the majority of cases, less common structural causes such as endometriosis involving the uterine serosa and adjacent bowel are rarely reported as a cause of intraoperative or postpartum hemorrhage [1,5]. Deep infiltrating endometriosis (DIE) is characterized by fibrotic, vascular, and adherent tissue planes that can bleed diffusely when disturbed, and such bleeding may be difficult to localize and control with standard techniques such as compression sutures or uterine artery ligation [2-4]. We report a case of severe, refractory, non-localizing hemorrhage arising from endometriotic involvement of the posterior uterine wall and bowel serosa following an emergency cesarean section, managed successfully with a staged damage-control surgical approach.

II. Case Presentation

Antenatal and Intraoperative Course

A 24-year-old primigravida with a history of significant dysmenorrhea was admitted at 40+4 weeks of gestation for induction of labor in view of post-datism. Induction was performed with dinoprostone gel per unit protocol. In view of a subsequently non-reassuring NST, the patient was taken up for emergency lower-segment cesarean section. Incision-to-delivery time was 7 minutes, and a live female infant weighing 3.1 kg was delivered with Apgar scores of 10 and 10 at 1 and 5 minutes, respectively. Intraoperatively, the patient was noted to have

ovarian endometriosis with rectosigmoid colon involvement, in addition to severe endometriosis over the posterior uterine surface. After uterine closure, profuse bleeding was identified at the time of inspection from the posterior uterine surface; the bleeding point could not be definitively localized. Multiple hemostatic sutures were placed, with persistent oozing despite these measures. Estimated blood loss at this stage of the surgery was 1 litre. A pelvic drain was placed and the abdomen closed.

Immediate Postoperative Course

Pre-operative hemoglobin was 11 g/dL, falling to 8.2 g/dL immediate postoperatively. Platelet count dropped from 2.5 lakh/µL to 1.5 lakh/µL. The patient remained asymptomatic, vitally stable but developed decreased urine output, prompting transfer to the MICU. Emergency ultrasonography of the abdomen and pelvis revealed hemoperitoneum with an estimated volume of approximately 1 litre. She was transfused 2 units PCV and 4 units FFP while re-exploratory laparotomy was arranged.

First Re-exploration

At re-exploration, the posterior uterine wall was found to be dry with no active bleeding. However, oozing was noted from endometriotic implants over the bowel serosa.

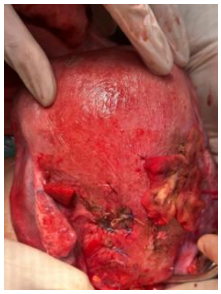


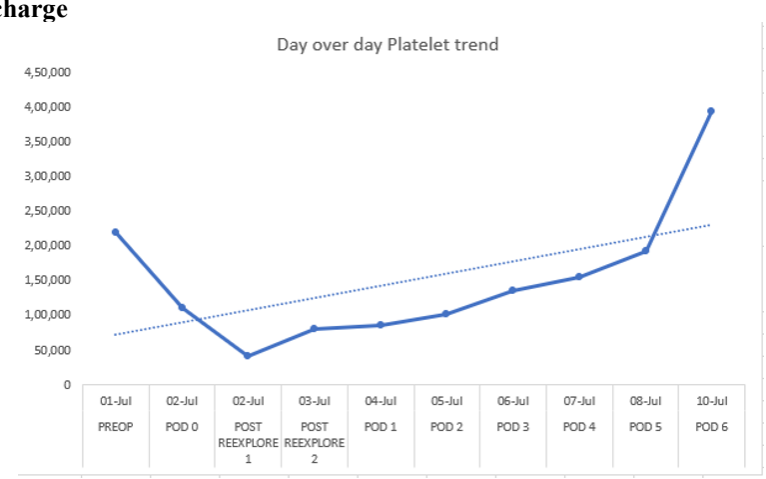
Figure 1. Intraoperative photograph at first re-exploration showing the posterior uterine wall with hemostatic sutures in situ; the surface was dry at this stage, with the ongoing bleeding source subsequently localized to bowel-serosal endometriotic implants

The General Surgery team was consulted intraoperatively; following discussion, hemostasis was achieved using a topical absorbable hemostatic agent placed over the bowel and posterior uterine wall and a temporary abdominal closure was performed in view of ongoing coagulopathy and the need for further correction before definitive closure. The patient was transfused a further 4 units PCV, 6 units FFP, 6 units RDP, and 6 units cryoprecipitate, and was managed in the MICU. Injection GnRH agonist was administered for the underlying endometriosis.

Second Re-exploration (Day 3)

Following correction of coagulopathy with RDP transfusion, the patient underwent a second planned re-exploration on day 3. No active oozing or bleeding was identified at this stage, and definitive abdominal closure was performed. She was maintained on higher-spectrum antibiotic cover (injection vancomycin and piperacillin-tazobactam) and monitored closely in the MICU.

Recovery and Discharge



Platelet counts trended upward over the following days. The patient was shifted from MICU to the ward on day 6 and made an uneventful recovery thereafter, and was discharged in stable condition.

Clinical Timeline:

DAY	EVENT
Day 0	Induction of labor (dinoprostone) for post-datism at 40+4 weeks
Day 0	Emergency LSCS for non-reassuring NST; ovarian/rectosigmoid/posterior uterine endometriosis noted; profuse non-localizing bleeding at closure; EBL 1L; hemostatic sutures + drain placed
Day 0 (post-op)	Hb 11→8.2 g/dL, platelets 2.5→1.5 lakh/µL, oliguria; MICU transfer; USG shows ~1L hemoperitoneum
Day 0-1	First re-exploration: posterior wall dry, bowel-surface endometriotic oozing found; topical hemostatic agent; temporary abdominal closure; massive transfusion; GnRH agonist given
Day 2	Second re-exploration: no active bleeding; definitive closure; vancomycin + piperacillin-tazobactam continued
Day 6	Platelets improved; shifted MICU → ward
Day 11	Discharged in stable condition

III. Discussion

Most published reports describe spontaneous hemoperitoneum during pregnancy secondary to rupture of decidualized endometriotic implants or utero-ovarian vessels. Persistent postpartum hemorrhage originating from diffuse bowel serosal endometriotic implants following cesarean delivery has rarely been reported [5-7], and its intraoperative recognition can be challenging. Unlike atonic PPH, bleeding from endometriotic implants is diffuse, often non-localizing, and may not respond to compression sutures or standard hemostatic techniques, as the source lies in friable, vascularized fibrotic tissue rather than a discrete uterine vessel. In this patient, a preceding history of significant dysmenorrhea was retrospectively consistent with underlying endometriosis, which was found intraoperatively to involve not only the posterior uterine surface but also the ovaries and rectosigmoid colon [2-4], a pattern in keeping with multifocal deep infiltrating disease rather than an isolated uterine finding.

In this case, the initial impression of uterine source bleeding at the time of primary closure prompted hemostatic sutures and drain placement; however, ongoing occult hemorrhage, evidenced by falling hemoglobin, thrombocytopenia, and oliguria, indicated an unresolved bleeding source, subsequently confirmed on imaging as significant hemoperitoneum. At re-exploration, the true source was identified as bowel-surface endometriotic oozing rather than the uterine wall itself, underscoring the importance of systematic intra-abdominal inspection beyond the uterus when postoperative bleeding is unexplained.

The subsequent coagulopathy reflected in the falling platelet count likely represented a combination of dilutional and consumptive effects from ongoing blood loss and transfusion, and was managed with a damage-control approach: temporary abdominal closure, correction of coagulopathy with component transfusion, and planned re-look laparotomy once physiological parameters had normalized. This staged strategy, well established in trauma and general surgery, is less commonly described in obstetric hemorrhage [8-10] but proved effective here, avoiding repeated high-risk surgery in a coagulopathic patient while ensuring source control was eventually confirmed.

Adjunctive GnRH agonist therapy was used to address the underlying endometriosis, and broad-spectrum antibiotic cover was maintained given the prolonged intra-abdominal exposure and temporary closure, mitigating infective risk during a complex postoperative course.

This case highlights that endometriosis, even when recognized intraoperatively, can be an unpredictable and diffusely bleeding surgical field, and that a low threshold for imaging, early senior involvement, and willingness to adopt a staged damage-control approach are key to a favorable outcome.

IV. Conclusion

Deep infiltrating endometriosis of the posterior uterine wall and bowel serosa is an under-recognized but important cause of severe, non-localizing hemorrhage during and after cesarean section. Early recognition of ongoing occult bleeding, prompt imaging, correction of coagulopathy, and a staged damage-control surgical approach including temporary abdominal closure and planned re-exploration can achieve definitive hemostasis and good maternal outcome even in complex cases.

Declarations

Consent for Publication: Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Conflict of Interest: The authors declare no conflict of interest.

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Author Contributions:

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