

Spontaneous Haemoperitoneum in the Third Trimester: A Case Report

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Abstract

Spontaneous haemoperitoneum in pregnancy (SHiP) is a rare but potentially catastrophic condition for both mother and the fetus, particularly in late gestation when clinical features overlap with more common obstetric and non-obstetric presentations. We present this rare case of a term pregnant patient who presented with gastrointestinal symptoms and later developed progressive anaemia without overt bleeding. Her presenting symptoms initially delayed suspicion of intra-abdominal haemorrhage. Emergency caesarean section converted to exploratory Laparotomy confirmed intra-abdominal bleeding. Intraoperative findings excluded Obstetric and common non-obstetric causes for the bleeding, directing to spontaneous rupture of Omental vessels as the plausible explanation in this case with unusual clinical course. Careful monitoring of the clinical condition, timely interventions and multidisciplinary input managed to save both mother and the baby.

Keywords

Spontaneous Hemoperitoneum, Third Trimester, Omental Vessel Rupture, Intra-Abdominal Haemorrhage

1. Introduction

Spontaneous haemoperitoneum in pregnancy (SHiP)—defined as non-traumatic intraperitoneal bleeding during pregnancy or up to 42 days postpartum, has an estimated incidence on the order of 4 - 5 per 100,000 births, and yet carries substantial perinatal risk. Reported aetiologies can be categorised into Obstetric related and non-Obstetric causes. Non-Obstetric causes include rupture of utero-ovarian vessels, endometriosis-associated bleeding, visceral artery aneurysms and

spontaneous rupture of Mesenteric or Omental vessels, with the splenic artery being the most frequently implicated aneurysm in pregnancy [1]-[6]. The third trimester is a high-risk period owing to increased pelvic venous congestion, hormonally mediated vascular changes, and increased intra-abdominal pressure.

Clinically, SHiP presents with a spectrum ranging from vague abdominal pain to hypovolaemic shock; fetal status may be initially reassuring, which can obscure the diagnosis. The rarity and non-specificity of features often delay recognition, and the diagnosis is frequently made intraoperatively at caesarean or laparotomy [1]-[5]. We present a case of spontaneous haemoperitoneum in the third trimester encountered during labour ward on-call, possibly due to rupture of the Omental blood vessels. We contextualize it within contemporary literature to highlight diagnostic, operative, and systems-based learning points.

Spontaneous rupture of omental vessels is an exceedingly rare cause of intra-abdominal haemorrhage, with only isolated cases described in the literature. The omentum, richly vascularised through branches of the gastroepiploic and mesenteric vessels, is susceptible to rupture in the presence of venous congestion, fragile vascular walls, or sudden increases in intra-abdominal pressure. Reported risk factors include obesity, trauma, coagulopathy, and anticoagulant use, although rupture can occur without identifiable predisposition. The clinical course is often acute, with intraperitoneal bleeding necessitating urgent surgical exploration; however, self-limiting ruptures with spontaneous haemostasis have occasionally been described, complicating intraoperative identification of the bleeding source. In pregnancy, this diagnosis is particularly challenging due to symptom overlap with more common obstetric emergencies. Our case appears to represent a rare example of spontaneous rupture of omental vessels with spontaneous resolution, discovered only through indirect operative findings.

2. Case Presentation

2.1. Patient Information and Presentation

A 29-year-old gravida 3 para 2 at 37 + 1 weeks presented with 24-hour history of colicky abdominal pain, vomiting, diarrhoea, and malaise. She had morbid obesity with the BMI of 53 and a history of prior caesarean section. There was no history of trauma or vaginal bleeding.

2.2. Clinical Findings

On assessment, her observations were stable. The abdomen was soft with mild generalised tenderness; the uterus was non-tender, and a reactive fetal cardiotocography (CTG) was recorded. Speculum examination showed a closed cervix with no bleeding. Laboratory tests revealed haemoglobin (Hb) 105 g/L, mildly elevated inflammatory markers and an unremarkable coagulation profile. A bedside ultrasound confirmed the fetal heartbeat, fetal movements, normal umbilical artery Dopplers and a fundal placenta. No retroplacental blood clots were seen.

She was initially treated for gastroenteritis with intravenous fluid and antiemet-

ics. However, she continued to feel unwell with nausea, vomiting, diarrhoea and ongoing abdominal pain more towards the upper abdomen.

Over next 24 hours, she experienced persistent pain and malaise with a progressive Hb decline to 72 g/L despite no external bleeding. Her repeat coagulation profile remained unchanged, and platelet counts were stable above $200 \times 10^9/L$. Given her VTE risk profile (obesity, prior caesarean, reduced mobility) and recent respiratory symptoms, pulmonary embolism (PE) was considered. A Computed Tomography Pulmonary Angiogram (CTPA) undertaken a week earlier had been negative. She had been on therapeutic Enoxaparin for 2 days, which was discontinued when PE was excluded prior to this admission. Worsening symptoms, uterine irritability, and unexplained anaemia prompted decision-making for expedited delivery for suspected concealed abruption.

2.3. Operative Course

An emergency lower-segment caesarean section was undertaken under regional anaesthesia. On entering the peritoneal cavity, nearly 1 L of dark, altered blood and clot was encountered. The uterus was intact with no dehiscence; amniotic fluid was clear; the placenta appeared normal, with no abnormal adherence or bleeding from the placental site.

Given the significant haemoperitoneum without an obvious obstetric source, the incision was extended to a midline laparotomy, and the procedure was converted to general anaesthesia. General surgeons examined the upper abdomen, small and large bowel, mesentery, and omentum. No active bleeding, visceral injury, or vascular lesion was identified. The distribution of clots within the upper abdomen raised suspicion of a self-resolved spontaneous rupture of Omental vessel. Haemostasis was satisfactory. Three intra-abdominal drains had placed; one anchored under the liver, second under the spleen and third in the Pouch of Douglas for surveillance. Estimated blood loss was 2.5 L. The patient received 4 units of packed red blood cells and 4 units of fresh frozen plasma intraoperatively. The neonate was delivered in good condition.

2.4. Postoperative Course and Follow Up

The patient was managed in a high-dependency setting for 48 hours with serial observations, Hb monitoring, and thromboembolism prophylaxis. Postoperative Hb was 98 g/L at 6 hours and 105 g/L on Day 1. Drain output was minimal and non-progressive. A postoperative CT abdomen and pelvis, performed to exclude visceral aneurysm or solid organ pathology, was unremarkable. The patient recovered uneventfully and was discharged on postoperative day 5 with outpatient follow-up. Both mother and baby were clinically well at the 6 weeks postnatal check.

3. Discussion

3.1. Diagnostic Challenges in Late-Pregnancy Haemoperitoneum

This case illustrates several well-described barriers to timely diagnosis. First, the

presenting symptoms—abdominal pain, gastrointestinal upset, malaise—are common in pregnancy and often benign. Second, no evidence of classical obstetric emergency symptoms such as vaginal bleeding, uterine tenderness, or non-reassuring antenatal CTG. Third, haemodynamic compensation in healthy parturients can mask significant intraperitoneal blood loss until late. The literature consistently notes that SHiP is frequently diagnosed at laparotomy or caesarean section because non-specific features and overlapping differentials obscure early recognition [1] [3] [6] [7].

In our patient, early laboratory values were not dramatic; however, the **trend**—a progressive Hb fall without external blood loss—was a crucial clue to concealed haemorrhage. Emphasising trajectory over single values is supported by obstetric haemorrhage guidance and by case series in which serial Hb decline was a herald sign of intra-abdominal bleeding despite temporarily reassuring maternal–fetal observations [7] [8].

3.2. Differential Diagnosis: Obstetric Causes

3.2.1. Uterine Rupture

Rupture is a key consideration in women with a uterine scar who present with abdominal pain, bleeding, loss of station, and fetal compromise. However, rupture can be atypical; posterior or fundal ruptures may present predominantly as haemoperitoneum [8]. Intraoperative inspection excluded rupture in this case.

3.2.2. Placental Abruption

Concealed abruption can mimic intra-abdominal bleeding with abdominal pain and anaemia yet typically features uterine tenderness/tonic contraction and non-reassuring FHR. In this case, clear liquor, a normal placenta, and reassuring CTG argued against abruption.

3.2.3. Spontaneous Rupture of Utero-Ovarian Vessels

Utero-ovarian venous rupture is a classic cause of SHiP. Pathophysiologic mechanisms include venous engorgement, increased intra-abdominal pressure, and hormonally mediated weakening of venous walls. Historical and modern reports describe third-trimester cases with sudden pain, intraperitoneal bleeding, and no uterine pathology; many require urgent laparotomy and ligation of bleeding venous channels [1] [3] [7]–[9]. Despite careful exploration, no such source was identified here, but a self-limiting rupture cannot be excluded.

3.2.4. Invasive Placentation (Percreta)

Placental invasion through the myometrium can cause uterine or extra-uterine bleeding; absent imaging and operative signs made this unlikely in our patient.

3.3. Differential Diagnosis: Non-Obstetric Causes

3.3.1. Visceral Artery Aneurysm (VAA) Rupture

Splenic artery aneurysm (SAA) is the most frequently reported VAA in pregnancy and carries high maternal and fetal mortality when rupture occurs. Systematic re-

views emphasise the often-occult nature of SAA before rupture, variable aneurysm size (many ≤ 2 cm), and the need for prompt vascular control when suspected [2] [5] [10]-[12]. In our case, postoperative CT excluded SAA and other VAA.

3.3.2. Spontaneous Mesenteric or Omental Vessel Rupture

Rare case reports implicate fragile mesenteric venules or omental vessels as bleeding sources, potentially precipitated by abrupt changes in intra-abdominal pressure or venous congestion. This was the most possible suspected cause in the indexed case. Given the risk factors such as morbid obesity, increased risk for thromboembolic events, recent history of treatment with Enoxaparin for suspected pulmonary embolism, favour the likelihood of spontaneous rupture of mesenteric and Omental vessels. These bleeds can tamponade spontaneously, yielding negative exploration—mirroring our findings [7] [11].

3.3.3. Endometriosis-Related SHiP

Contemporary data highlight a strong association between endometriosis and SHiP, particularly in the second half of pregnancy. Mechanisms include rupture of decidualised endometriotic implants or friable utero-ovarian/parametrial varices within dense adhesions. Reviews and cohort data report that endometriosis is present in a substantial proportion of SHiP cases, and recurrence has been documented [3] [5] [6] [13]. Our patient had no known endometriosis, and no implants were seen, making this less likely but not excluded.

3.3.4. Solid Organ or Hepatic Causes

Spontaneous hepatic rupture (e.g., HELLP-related) and splenic pathology can cause haemoperitoneum; clinical context, labs, and CT imaging mitigate these in our case.

3.3.5. Coagulopathy

Diffuse oozing with deranged coagulation parameters would raise suspicion. Here, coagulation studies were not markedly abnormal, and the intra-abdominal blood was predominantly dark and clotted, arguing against a systemic coagulopathy.

3.4. Impact of Morbid Obesity on Surgical and Anaesthetic Complexity

The patient's morbid obesity (BMI 53) significantly increased the technical complexity of both the caesarean section and the subsequent exploratory laparotomy. Excess adipose tissue contributed to reduced surgical field visibility, impaired access to deeper structures, and difficulty in achieving adequate exposure. Prolonged time was required for abdominal entry, haemostasis, and closure, with increased risk of wound complications and infection. Intraoperatively, thick abdominal wall layers and limited retraction space also complicated identification of the bleeding source. Anaesthetic management was further challenged by airway considera-

tions, reduced cardiopulmonary reserve, and altered pharmacokinetics. These factors collectively underscore how morbid obesity not only heightens surgical risk but also complicates intraoperative decision-making in the context of an unexpected haemoperitoneum.

4. Conclusion

Spontaneous haemoperitoneum in pregnancy remains a diagnostic and therapeutic challenge owing to its rarity, atypical presentation, and significant maternal-fetal risk. This case highlights the need for a high index of suspicion for intra-abdominal bleeding in pregnant patients with non-specific gastrointestinal symptoms and unexplained anaemia, even in the absence of overt bleeding. Prompt recognition, timely surgical intervention, and coordinated multidisciplinary care are essential for optimising outcomes. Importantly, this case underscores a key learning point for obstetric triage teams: progressive anaemia without overt bleeding should prompt early surgical review. Reporting such cases contributes to greater awareness among clinicians and may facilitate earlier diagnosis in future presentations.

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Conflicts of Interest

The authors declare no conflict of interest.

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