
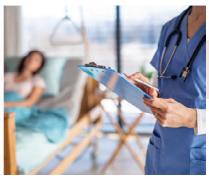

CASE REPORT

Received: 28 September 2025
Last revision: 05 September 2026
Accepted: 08 September 2026
Published: 18 September 2026

Secondary Postpartum Haemorrhage due to Retained Intrauterine Blood Clot with Impaired Uterine Outflow Following Caesarean Section: A Case Report

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ABSTRACT

Introduction: Postpartum hemorrhage (PPH) is the main cause of maternal mortality, ranging from 18%–50% of deaths worldwide. Secondary postpartum hemorrhage (SPPH) is defined as bleeding 24 hours to 12 weeks after delivery. Clinically worrisome uterine hemorrhage develops within 1 to 2 weeks in perhaps 1 percent of women. Such bleeding most often is the result of abnormal involution of the placental site. In some cases, uterine involution is hindered because of infection, retained placental tissue, or other causes. Such subinvolution is accompanied by varied intervals of prolonged lochia as well as irregular or excessive uterine bleeding. Another cause of subinvolution is incompletely remodeled uteroplacental arteries. These noninvoluted vessels are filled with thrombosis and lack an endothelial lining. Perivascular trophoblasts are also identified in the vessel walls, suggesting an aberrant interaction between uterine cells and trophoblasts. Secondary postpartum hemorrhage is most commonly associated with endometritis and retained products of conception, particularly retained placental tissue. However, this case highlights an uncommon mechanism of secondary postpartum hemorrhage associated with a retained intrauterine blood clot, which causes incomplete spiral artery remodeling. **Case:** A 36-year-old woman with approximately 2,000 mL of bleeding 9 days after elective cesarean delivery. Ultrasonography examination showed a blood clot in the uterus measuring 10.77 x 12.85 cm; a 700 mL blood clot was removed from the uterus by suction curettage. Postoperative ultrasound evaluation found the uterus in good condition without blood clots. **Discussion:** In this case, the retained blood clot is attributed to obstructed uterine outflow resulting from the lack of prior dilatation of the internal os, rather than to retained placental tissue. Consequently, this case offers an opportunity to discuss retained intrauterine blood clots as a rare and potentially underrecognized mechanism of secondary postpartum hemorrhage following cesarean delivery. **Conclusion:** In this case, secondary postpartum hemorrhage was caused by uterine subinvolution resulting from the retention of blood clots within the uterus, which prevented complete remodeling of the uteroplacental arteries.

Keywords: Case report, endometritis, late postpartum hemorrhage, uterine subinvolution.

https://doi.org/10.55175/cdk.v53i09.2157
e-ISSN: 2503-2720

INTRODUCTION

Postpartum hemorrhage (PPH) is the leading cause of maternal mortality, accounting for 18%–50% of deaths worldwide.[1] The incidence of PPH is approximately 5%–20% of all deliveries, with a large percentage occurring in developing countries.[2] Postpartum hemorrhage (PPH) is characterized by blood loss exceeding 500 mL from the genital tract after childbirth. It is classified into two types: early (or primary) PPH, if it happens within the first 24 hours after delivery, and late (or secondary) PPH, if it occurs between 24 hours and 12 weeks postpartum.[3] Early postpartum hemorrhage can be caused by uterine atony, birth-related injuries, retained placental tissue, or blood clotting disorders. Late PPH occurs in just 1% of all deliveries, and its causes include retained products of conception, endometritis, uterine artery pseudoaneurysm, and subinvolution of the placental site.[3,4]

Although late PPH affects fewer women than early PPH, delays in diagnosis and treatment can lead to serious complications. Late postpartum hemorrhage can be life-threatening because it usually happens one to two weeks after childbirth, often when patients are already at home. This can lead to a lack of awareness of the seriousness of the bleeding. A significant clinical cause of PPH is vessel subinvolution at the placenta site.[2,5]

Uterine subinvolution is a situation where the uterus fails to return to its pre-pregnancy size and condition promptly. This delayed involution can result in the accumulation of blood within the uterine cavity, leading to the formation of blood clots.[6] Uterine subinvolution may be caused by incompletely remodeled spiral arteries, retained placental fragments, or infections such as endometritis.[3,6] Endometritis is an inflammation limited to the endometrium, the uterus's inner lining. It is the leading cause of infection following childbirth.[7,8]

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Late postpartum hemorrhage related to uterine subinvolution poses risks to maternal health, including the potential for severe blood loss. Understanding the mechanisms and management of this condition is crucial for effective postpartum care and ensuring positive maternal outcomes. We report a case of late postpartum uterine subinvolution caused by a blood clot in the uterus, due to incompletely remodeled spiral arteries and endometritis after elective cesarean delivery.

Following placental delivery, physiological hemostasis at the placental implantation site depends on effective myometrial contraction and progressive involution of the uteroplacental vasculature, including remodeling and occlusion of the spiral arteries. Incomplete remodeling or inadequate involution of the spiral arteries may result in persistent vascular patency and increased susceptibility to bleeding from the placental bed. When this bleeding occurs in the setting of impaired uterine outflow, particularly from the lack of prior dilation of the internal os, postpartum blood may accumulate within the uterine cavity rather than being adequately expelled.2 Persistent intrauterine blood accumulation may subsequently undergo coagulation and form retained blood clots, resulting in hematometra or lochiometra. The retained blood and clot may further interfere with uterine involution and effective myometrial contraction, potentially perpetuating bleeding from the placental implantation site. Intermittent or sudden evacuation of the accumulated blood and clot may then present clinically as secondary postpartum hemorrhage. Thus, in this proposed mechanism, incomplete vascular remodeling may contribute to persistent bleeding. At the same time, impaired uterine outflow facilitates blood retention and clot formation, together contributing to secondary postpartum hemorrhage (SPPH).

CASE

A 36-year-old woman, parity 2, post-cesarean delivery 9 days ago, with a history of cesarean section 6 years ago. The woman was referred to the emergency department with vaginal bleeding 9 days after an elective cesarean delivery. The bleeding was approximately 2,000 mL. On initial examination, the patient's vital signs were: blood pressure 115/74 mmHg, heart rate 117 beats per minute, respiratory rate 20 breaths per minute, body temperature 38.6°C, and oxygen saturation 99%. Physical examination showed that the height of the uterine fundus is at the umbilicus level, higher than normal for this stage, indicating uterine subinvolution. Bloody vaginal discharge was also noted; no signs of infection or complications at the surgical site. The patient appeared clinically anemic. Laboratory examination showed: hemoglobin (Hb) 7.4 g/dL (12–16 g/dL), hematocrit 22.2% (36%–46%), leukocytes 13.9 x 10^3/uL (4.3–10.3 x 10^3/uL), platelet count 153 x 10^3/uL (150–450 x 10^3/uL), prothrombin time 11.5 seconds, and activated partial thromboplastin time 26.7 seconds. The laboratory results showed anemia and leukocytosis.

The first pregnancy ended in miscarriage, requiring a curettage procedure. A year later, the second pregnancy was delivered via cesarean section due to placenta previa totalis. Six years afterward, the third pregnancy was also delivered by cesarean section because of a history of a previous cesarean. The patient had no history of any previous illnesses such as hypertension, diabetes, and anemia. Ultrasonography examination revealed possible organized blood clots inside the uterus measuring 10.77 x 12.85 cm **(Figure 1)**. The diagnosis was uterine subinvolution with endometritis.

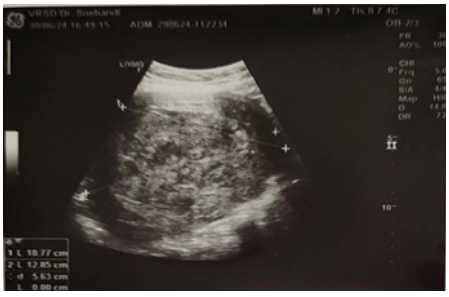

*Photo documentation by Fikhy Rizky.
Figure 1. Ultrasonography shows blood clots in the uterus (arrow).

The patient received IV ceftriaxone 1 gram twice daily and IV drip metronidazole 500 mg three times daily. Uterotonic therapy included oxytocin drip at 20 IU for up to 12 hours and methylergometrine 0.2 mg IV three times daily. Additional medications included tranexamic acid 500 mg IV three times daily and paracetamol 1 gram IV drip three times daily. To manage anemia, a blood transfusion of 2 units (approximately 210 mL per unit) was administered.

Uterine suction curettage was performed with general anesthesia, and 700 mL of blood clots was removed. Post-operative ultrasonography found the uterus was in good condition without clots in the uterus. After the suction curettage, antibiotics and uterotonic agents treatment was continued. A vaginal tampon was kept in place for six hours after suction curettage. The patient was discharged after three days of hospitalization in stable condition.

DISCUSSION

Postpartum hemorrhage is an obstetric significant issue, being a major contributor to maternal morbidity and mortality.[9] However, late PPH received less attention because a majority of patients are treated medically on an outpatient basis, without hospitalization.[4]

During pregnancy and the postpartum period, significant changes occur in uterine vasculature, particularly in the spiral arteries at the placental attachment site. Between 6 and 10 weeks of gestation, extravillous trophoblasts (EVTs) originating from the placenta invade the maternal spiral arteries within the decidua, forming endovascular "plugs" that destroy the muscular and elastic components of the arterial walls while depositing hyaline fibrinoid material.[2,10] These EVTs also replace the endothelial lining and contribute to extravascular remodeling by secreting angiogenic factors, resulting in vascular colonization extending into the inner third of the myometrium. This physiological transformation creates large-caliber, low-resistance vessels that ensure adequate perfusion to the placenta and fetus.[11]

After delivery, uteroplacental arterial involution reverses these changes to prevent excessive bleeding. Maternal endothelial cells gradually replace the EVTs at the placental site, leading to fibrointimal thickening and vascular occlusion. Concurrently, the myometrium contracts, mechanically compressing vessels and promoting thrombosis. These involutional changes, along with necrosis and shedding of the decidua and superficial endometrium,


contribute to uterine remodeling and the reduction of postpartum bleeding.[2]

In normal postpartum involution, clot formation and fibrotic closure of the uteroplacental vessels restore the uterus to its pre-pregnancy state. Failure of this process can result in subinvolution of the placental site, where vessels remain abnormally patent. This condition can cause persistent or delayed postpartum hemorrhage, commonly during the second week after childbirth.[4] Immediately after delivery, or approximately an hour later, the myometrium remains tightly contracted and retracted. This pressure directly compresses large uterine vessels and promotes thrombosis to prevent bleeding. Typically, uterotonic agents enhance this process.[12]

Physiologically, the uterus weighs approximately 1000 grams immediately postpartum and is usually at the level of the umbilicus. By the end of the first week, it should have reduced in size to about 12 weeks of gestation and be palpable at the pubic symphysisv.[13] In this case, on postpartum day 9, the uterus remained at the level of the umbilicus, an abnormal finding indicating uterine subinvolution.

Another anatomical factor relevant to this case is cervical dilation. Normally, during labor, the cervix gradually softens and dilates in response to uterine contractions.[14] In this case, an elective cesarean section was performed before labor process, consequently, there was no cervical dilation or opening of the internal os. Her previous delivery had also been a cesarean section due to placenta previa. In such cases, the absence of cervical opening may impede the drainage of lochia and blood, increasing the risk of clot retention.

Cervical dilatation facilitates lochia and blood drainage, reducing the risk of intrauterine infection and late postpartum bleeding. It can be performed intraoperatively through the uterine incision using a finger, sponge forceps, or similar instruments to gently open the cervical canal and allow lochia to drain freely through the vagina.[14] Mechanical dilatation of the cervix was not performed because it is not routinel practiced at our institution during elective cesarean sections. Current evidence regarding its benefits in preventing postpartum hemorrhage remains inconclusive,[14] and some studies.[14,15] Suggest that routine cervical dilatation may increase the risk of infection or cervical trauma without providing significant advantages.

Vessel subinvolution can also be enhanced by the presence of acute inflammation, which is intensified by the production of proinflammatory factors and localized tissue coagulation disorders.[2] This case had normal prothrombin time (PT) and activated partial thromboplastin time (aPTT) values, indicating no coagulopathy.

Patients undergoing cesarean section are at greater risk for postpartum infections and complications, including metritis and wound infections, with infection-related mortality rates nearly 25 times higher than after vaginal delivery.[16–18] The risk of endometritis and surgical site infections increases to approximately 16%–17% following cesarean section.[19] This case presented with a temperature of 38.6°C, tachycardia (117 bpm), and mild leukocytosis (13.9 × 10^3/µL), suggesting a possible uterine infection. Fever is a key clinical marker for postpartum metritis, with higher temperatures (> 39°C) potentially indicating bacteremia or endotoxemia.[20] Uterine tenderness and abdominal pain are common findings, and leukocytosis may range from 15,000 to 30,000 cells/µL, though elevated white blood cell counts can also be seen following cesarean delivery.[21]

Although fever and leukocytosis were observed, the absence of foul-smelling lochia and uterine tenderness made postpartum endometritis less clinically likely. In this context, the fever and leukocytosis may have reflected a postoperative inflammatory response associated with tissue injury following cesarean delivery rather than a primary infectious process. Nevertheless, without microbiological and laboratory investigations, endometritis could not be completely excluded. The overall clinical presentation, together with the absence of retained placental tissue and the absence of dilatation of the internal os with a retained intrauterine blood clot, supported uterine outflow obstruction as the more likely mechanism underlying the secondary postpartum hemorrhage in this case.

The accumulation of blood clots and retained debris in the uterus further increases the risk of endometritis. In this case, infection and subinvolution occurred concurrently, leading to late postpartum hemorrhage (PPH).[22] The fluid expelled from the uterus on postpartum day 9 was not physiological lochia. Normally, lochia progresses through three stages: lochia rubra (1–4 days, red and blood-filled), lochia serosa (5–9 days, yellowish or pale brown), and lochia alba (10–14+ days, white or clear). The presence of persistent heavy red discharge on day 9 is abnormal. Prolonged red lochia beyond one week may indicate uterine subinvolution.[23]

The patient was treated with methylergometrine and oxytocin, both stimulate uterine contraction and are first-line agents in postpartum hemorrhage management.[5,7] The combination of these uterotonics is known to produce synergistic effects in enhancing uterine tone and controlling bleeding.[24] The definitive management of this case, based on literature from Cunningham, *et al.*,[3] stated that if large clots are seen with sonography in the uterine cavity, gentle suction curettage is considered.

CONCLUSION

Secondary postpartum hemorrhage is most commonly associated with retained products of conception and endometritis; however, retained intrauterine blood clot secondary to uterine outflow obstruction represents a rare and potentially underrecognized mechanism. In this case, the absence of internal os dilatation impaired evacuation of postpartum blood, resulting in intrauterine blood accumulation and clot formation, with uterine subinvolution potentially contributing to persistent bleeding from the placental implantation site. The absence of foul-smelling lochia and uterine tenderness made endometritis less clinically likely, while the absence of convincing internal vascularity within the intrauterine material favored retained blood clot over vascularized retained products of conception. This case highlights the importance of considering uterine outflow obstruction and retained intrauterine blood clot in the differential diagnosis


of secondary postpartum hemorrhage following cesarean delivery. Careful clinical assessment, ultrasonography with color Doppler, and timely restoration of uterine outflow are essential for accurate diagnosis and appropriate management.

Consent for Publication

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

Funding

This case report received no financial support or funding.

Author Contributions

FRH contributed to the conception and design of the case report, patient management, acquisition and interpretation of clinical data, preparation of the figures and clinical documentation, drafting and critical revision of the manuscript, and approval of the final version of the manuscript. AA contributed to patient management, data collection, literature review, interpretation of clinical findings, and critical revision of the manuscript. SZR contributed to the literature review, critical revision of the manuscript, and supervision of the case report. All authors reviewed and approved the final version of the manuscript.

Conflict of interest

The authors report no conflicts of interest.

Declaration on the Use of Artificial Intelligence (AI)

The authors declare that no artificial intelligence tools were used in the conduct of this study or preparation of this manuscript.

Acknowledgments

No acknowledgements are included in this case report.

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