Ruptured Uterine Artery Pseudoaneurysm: An Overlooked Cause of Late Postpartum Haemorrhage*

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Abstract: We report an exceptional case of secondary postpartum hemorrhage (PPH) twenty days after cesarean delivery, resulting from a ruptured uterine artery pseudoaneurysm (UAP). The diagnosis was initially confounded by a septic shock necessitating inotropic support in the intensive care unit. Intense vaginal bleeding occurred eleven days after uterine curettage. Doppler ultrasound showed an anechoic focus in the inferior part of the uterus with turbulent flow. Bilateral internal iliac artery angiograms revealed a left uterine artery pseudoaneurysm that was successfully embolized. UAP is a rare cause of unexplained PPH that requires a high index of suspicion for diagnosis. This first report in the anesthesia literature serves to focus our awareness on its possible occurrence, and gives track to its management. Pitfalls in UAP diagnostic are highlighted (delayed presentation, possibility of spontaneous hemostasis, and lack of typical findings on colour Doppler sonography in hypotensive patients). These pitfalls may further be confounded by a concomitant sepsis. Angiography and selective uterine artery embolization is the treatment of choice.

Key words: Uterine artery; pseudoaneurysm; postpartum hemorrhage; sepsis; embolization.

We describe a patient with late postpartum hemorrhage (PPH) in which the diagnosis was complicated by the presence of a septic shock. This late PPH was due to the rupture of an uterine artery pseudoaneurysm (UAP). UAP is a rare and potentially life threatening entity if not diagnosed and treated in a timely manner.

Case report

A 30 year old Gravida 2 Para 2 woman presented with giddiness, lower abdominal pain and persistent vaginal bleeding for twenty days after a term cesarean delivery for preeclampsia in another hospital. Apart from an uneventful cesarean section 4 years ago, she denied other medical problems.

She was fluid resuscitated, and 200 ml of blood clots were evacuated on speculum examination. Vaginal pads were inserted for tamponade. She received 1 g of IV ceftriaxone and 500 mg of metronidazole 500 mg. An ultrasound scan revealed uterine cavity distension with possible blood clots or retained products of conception. She underwent an emergency evacuation of the uterus under general anesthesia and control of the airway by a laryngeal mask. This curettage evacuated retained bits of placenta and membranes. An oxytocin infusion was commenced.

In the post-anesthesia recovery room, she bled vaginally again, prompting a return to the operating theatre, where a second uterine evacuation was performed under general anesthesia with endotracheal intubation. At that time, IV Carbetocin 100 mcg, and carboprost 250 mcg were administered. Intraoperative arterial blood sampling revealed a pH of 7.23, a PaO2 of 264 mmHg, a PaCO2 of 42.1 mmHg, a base excess of -10 mmol/L, and a hematocrit of 21%. She received red blood cell and fresh frozen plasma transfusion, after which her vital signs stabilized. Thereafter, she was transferred to the Intensive Care Unit (ICU). Over the next 14 hours, she became febrile, hypotensive, and oliguric, despite receiving another 2.5 L of crystalloids, 1000 mL of colloid, and 685 mL of red blood cell concentrate. Invasive blood pressure and central venous pressure (CVP) monitoring was instituted, and noradrenaline commenced. Her leucocytosis increased from 9.4 to 21.9 on 8 October 2010.


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16.1 \times 10^9/L, platelets dropped from 394 to 107 \times 10^9/L. She also developed coagulopathy. Antibiotics were changed to IV imipenem, vancomycin, and clindamycin, as well as oral doxycycline. More blood clots were removed from the vagina on speculum examination, but no active bleeding was found. The Hb decreased from 8.6 to 5.4 g/dL, prompting further red blood cell and FFP transfusion, and an additional IM carboprost 250 mcg. Twenty-one hours after surgery, the patient improved and noradrenaline was discontinued. Oxytocin was continued at 15 IU/h. Over the next 72 hours, the patient remained hemodynamically stable, but continued to ooze blood vaginally, with a persistent low grade temperature. Blood, urine, and sputum cultures were negative, and Clindamycin and Imipenem were changed to IV piperacillin/tazobactam. She was transferred to the ward on the 4th postoperative day. Her only residual symptoms were continuous vaginal ooze that was attributed to residual sepsis and coagulopathy.

On the 11th postoperative day, she had a sudden 200 mL vaginal bleed. An urgent Doppler ultrasound of the pelvis showed an anechoic focus in the inferior aspect of the uterus, with central arterial flow. Bilateral internal iliac angiograms were performed, revealing a 5 mm focal aneurysm of the left uterine artery (Fig. 1); it was embolized with gel foam. Successful obliteration of the pseudoaneurysm was confirmed on the follow up Doppler two days later. The patient vaginal bleeding stopped completely, her Hb improved to 12.2 g/dL, and she was discharged home two days post-embolization. Twenty days after the embolization, a new Doppler ultrasound scan showed normal uterus and ovaries, and no aneurysmal dilation of the left uterine artery.

**Discussion**

While common causes of secondary postpartum hemorrhage (PHH), such as retained products of conception, subinvolution of the placental bed, and endometritis are well-known and easily diagnosed, uterine artery pseudoaneurysm (UAP) is a rare cause of PHH that requires a high index of suspicion for diagnosis. To our best knowledge, it has never been reported in the anesthesia literature.

A pseudoaneurysm is a blood-filled cavity, with turbulent flow, that communicates with the arterial lumen. It results from an inadequate sealing of a lacerated artery. This breach can occur as a consequence of surgery, penetrating trauma, neoplasm or infection. UAPs have been observed after abortion, repeated curettage, cesarean section, or even uncomplicated vaginal delivery (1). Tissue damage from electrosurgery used during hysterectomy, and laparoscopic myomectomy have also been implicated. Lack of knowledge of UAP may lead to wrong diagnosis, and confusion with endometritis, retained products of conception or hypermenorrhea. The delay in accurate diagnosis can result in a rupture of the UAP and profuse hemorrhage. In the present case, the difficulty in proper UAP diagnosis was also attributable to a delayed presentation in the form of a PHH, in the context of a septic shock. In addition, abnormal uterine bleeding secondary to UAP can cease spontaneously. Indeed, UAP rupture has already been reported to occur 3 months after a cesarean section (2), and spontaneous hemostasis has been reported for up to 4 weeks before a new bleeding onset, masking the true diagnosis (3).

The primary diagnostic tool for UAPs is transvaginal ultrasonography, typically revealing uterine anechoic foci on B-mode imaging (4). A color Doppler ultrasound spinning blood flow pattern confirms the diagnosis. This mosaic flow pattern is due to the multi-directional swirling of arterial flow at varying velocities (5). Contrast medium-enhanced CT or MRI identifies the perfusion sac. Angiography excludes other vascular anomalies (arterio-venous malformations and fistulas) as a cause of PHH, and provides definitive diagnosis of

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UAP, enabling concurrent therapeutic endovascular embolization (1). Indeed, selective uterine artery embolization is the preferred treatment modality. This treatment preserves fertility (6), as opposed to surgical options (arterial ligation and hysterec­my). A novel alternative therapeutic approach has also been described, where ultrasound-guided percutaneous or transvaginal direct thrombin injection into the UAP achieves hemostasis (7), but no other studies have substantiated its efficacy and safety. In other settings, surgery and life-saving hysterec­my may have to be resorted to, in cases of acute massive bleeding and no time or expertise for arterial embolization.

When our patient presented with vaginal bleeding, she was also febrile, tachycardic, hypotensive and coagulopathic. Streptococcal toxic shock syndrome from infected retained products of conception was the presumptive diagnosis, but bacterial cultures were negative. There was also no suggestion of an aneurysm on the first ultrasound. However, the vaginal ooze and fever persisted after the correction of coagulopathy. On the 11th postoperative day, our patient developed a 200 mL vaginal bleed, which prompted a urgent Doppler ultrasound, and showed the anechoic focus with turbulent flow typical of an aneurysm. Transcatheter selective arterial embolization of the UAP was undertaken with no recrudescence of symptoms in our case.

This highlights the pitfalls in UAP diagnosis, particularly in a shocked patient. Another report concurs with ours, where the UAP was not evident on the color Doppler ultrasound performed at patient first presentation for a hemorrhagic shock. A re-scan 10 h later, when the patient was more hemodynamically stable, revealed the lesion (8). A search for underlying bleeding disorders, like von Willebrand disease (9) is also prudent. Splenic artery aneurysm is the commonest (60%) of all visceral artery aneurysms, and its rupture needs to be ruled out in any peripartum women presenting with severe upper abdominal/chest pain, profound hemodynamic instability, and cardiovascular collapse (10). Although rare, splenic artery aneurysm rupture is associated with a 25% mortality, increasing to 75% among pregnant women. In that case, fetal mortality is estimated to be of 95% (11).

Despite a recent proliferation of reports in radiology and gynaecology journals, UAP has eluded the domain of obstetric anaesthesiology. It is timely that it be highlighted now, as anesthesiologists worldwide become increasingly involved as periope­rative physicians, in tandem with the obstetricians, to ensure patient safety. This case also serves to heighten awareness of UAP diagnosis pitfalls. Not only do delays in diagnosis lead to life-threat­ening consequences of the hemorrhagic shock, patient morbidity is also increased by greater blood transfusion requirements, lengthened hospitaliza­tion, and fertility loss if hysterec­my ensues.

In conclusion, UAP rupture is a rare cause of unexplained PPH, which requires a high index of clinical suspicion for diagnosis. Transvaginal color Doppler sonography is highly sensitive and specific for pseudoaneurysm detection, and arterial embolization is the treatment of choice with good outcome (12).

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References