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CASE REPORT

Secondary postpartum hemorrhage of an adenomyotic patient after a history of atony managed with B-Lynch sutures: A case report

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ABSTRACT

Secondary postpartum bleeding is defined as abnormal uterine bleeding occurring between the 24th hour and the 12th week postpartum, affecting approximately 1–2% of postpartum women. In the c-section of a 19-year-old pregnant woman (gravida 2, para 1, c-section 1) at term with polyhydramnios, uterine atony was managed with medical treatment and hemostatic B-Lynch sutures. After being discharged on the third day with no abnormal findings, she presented with acute, abundant vaginal bleeding on postpartum day 15. The uterus was semi-contracted, with subinvolution. Ultrasound revealed a 21 mm heterogeneous area in the endometrium and adenomyosis. The bleeding was managed with uterotonics and intrauterine balloon tamponade following revision curettage, and was attributed to late atony. Due to the timing of secondary postpartum hemorrhage and its rarity, there is a high probability of misdiagnosis. To prevent serious morbidity and mortality, the etiology should be evaluated promptly, and appropriate intervention should be undertaken.

Keywords: Adenomyosis, curettage, placenta, postpartum hemorrhage, uterus.

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INTRODUCTION

Postpartum hemorrhage is significant uterine bleeding that occurs after delivery. Primary postpartum hemorrhage is defined as abnormal bleeding within the first 24 hours of the postnatal period, while long-term bleeding occurring between 24 hours and 12 weeks is diagnosed as secondary postpartum hemorrhage.[1,2]

Approximately 1–2% of postnatal women are affected by secondary postpartum hemorrhage.[1,2] Primarily, the uterus fails to contract adequately to prevent hemorrhaging. The most common causes are retained placental products, intrauterine infection, and subinvolution of the placental site. Nonetheless, the main cause is often not established.[1–4]

In this paper, a secondary postpartum hemorrhage case of a woman who underwent a c-section complicated by atony, which was resolved with B-Lynch compression sutures 15 days ago, is presented.

CASE REPORT

A 19-year-old pregnant woman (gravida 2, para 1, c-section 1) with polyhydramnios presented to the ER (emergency room) with persistent uterine contractions, leading to a c-section. During the c-section, the placenta was easily separated from the uterus by controlled cord traction. Subsequently, the uterus became atonic, and medical treatment was administered along with uterine massage. The uterus was unresponsive to the medications; therefore, hemostatic B-Lynch sutures were performed, improving the uterine tone. Her post-op hematocrit levels decreased by 4.6% (hemoglobin of 9.1g/dL decreased to 7.6 g/dL). The patient recovered immediately with a contracted uterus and was discharged on the 54th hour post-labor without any need for blood replacement.

On postpartum day 15, she presented to the ER with acute, abundant vaginal bleeding. The ultrasound showed an adenomyotic uterus with subinvolution and an endometrial line of 21 mm, presumed to be coagulum. The patient had no fundal tenderness, and the uterus was semi-contracted. Her blood pressure was 75/50, heart rate was 85 bpm, and she had no fever. Intravenous tranexamic acid (1g in 100 cc of saline), intramuscular methylergonovine (0.2 mg/mL), and intravenous bolus saline infusion were administered. Medical attempts failed to control the bleeding, so a revision curettage was performed. After the suctioning of massive amounts of blood and the administration of tranexamic acid and uterotonics, bleeding was not sufficiently controlled. A Bakri balloon was then placed into the uterus, and vaginal packing was done for hemostasis. Subsequently, the patient was taken to the intensive care unit for close observation.

On the first round of blood tests, a decrease in the hematocrit level by 6% was observed (hemoglobin of 9.7 g/dL decreased to 7.7 g/dL). Two units of erythrocyte suspension and four units of fresh frozen plasma were administered. Additionally, 20 units of IV oxytocin, 400 mcg of rectal misoprostol, intravenous ceftriaxone, and metronidazole were administered. After 24 hours, the vaginal pack and the Bakri balloon were removed uneventfully. On the 30th hour, the patient was transferred to the maternity ward.

On the fourth day, a 3D ultrasound showed the uterus in involution with an irregular endo-myometrial junction, adenomyosis, and a 16 mm coagulum in the endometrial line. A hyperechoic area of 6x15 mm was observed at the fundus. A hysteroscopy was performed, and the area appeared as a clot. A biopsy was obtained from the spot, while the rest of the cavity appeared normal, with no retained placental tissue.

The patient was discharged with third-generation cephalosporin on the fifth postoperative day, the 19th day postpartum, with Hb: 7.8 g/dL, Hct: 24.8%, WBC: 7000/mm^3, and CRP: 6 mg/L. She did not develop a fever during her hospital stay.

The pathology result revealed bleeding necrotic decidua with endo-myometrial tissue fragments. She did not attend her outpatient follow-up appointments.

DISCUSSION

Postpartum hemorrhage is an obstetric emergency that requires prompt and efficient management. The appropriate treatment depends on the situation and may involve medical treatment, surgical procedures, or a combination of both. In cases where medical treatment fails, additional surgical procedures, such as compression sutures, are recommended.

The B-Lynch suture is an example of a compression suture that is straightforward to perform and effective in controlling acute hemorrhage. However, like any surgical procedure, it has potential complications, including uterine ischemic necrosis, synechia formation, and infection. Although the incidence of these complications increases when compression sutures are combined with vessel ligation, they may also result solely from the extreme degree of compression caused by the sutures.[5]

Following placental delivery, the uterus begins to contract to its nonpregnant size, a process known as uterine involution. Through the contraction of myometrial muscle fibers, the intramyometrial vessels constrict, reducing blood flow, especially at the placental site. With these contractions, large vessels at the placental site start to thrombose, preventing further blood loss. If the contractions are inadequate, uterine atony may ocur.[6]

The junctional zone of the uterus plays a significant role in uterine contractility; when disrupted, abnormal uterine contractility can lead to failure to progress in labor, uterine hyperstimulation, atony, and placental retention, as seen in women with adenomyosis. Adenomyosis results in structural changes in the myometrial junctional zone, leading to altered decidualization and early placentation, which may cause placental retention. Placental retention can impair myometrial contractile force, contributing to postpartum hemorrhage. Another aspect of postpartum hemorrhage possibly linked to adenomyosis is its association with changes in angiogenesis, which create fragile, vulnerable neovascularizations. In an adenomyotic uterus, hypertrophied muscle cells with poor contractile ability may prevent the placental attachment site from fully contracting after delivery.[7,8] The ADENO study revealed that when confounders are controlled, there is a slightly increased risk of postpartum hemorrhage in patients with adenomyosis.[9]

Another study by Giorgi M, et al.[10] found that among patients undergoing postpartum hysterectomy due to postpartum hemorrhage, the histologically confirmed prevalence of adenomyosis was 39.4%.

Retained placental products also disrupt myometrial contractility, leading to postpartum hemorrhage. Although the diagnosis is primarily histological, ultrasonography can suggest its presence. The most sensitive finding (80%) is a thickened endometrium with a cut-off value of 10mm, but its specificity is low (30%). A solid, echogenic intracavitary mass lesion has moderate positive predictive value (PPV) (80%). Blood clots may mimic retained placental products on grayscale imaging, but with Doppler, the intrinsic vascularity can help differentiate the two entities. While the absence of vascularity is compatible with blood clots, the presence of any vascularity in the endometrium on Doppler ultrasonography has a 96% PPV for retained products of conception.[11] A meta-analysis by Sundararajan, et al.[12] concludes that the best predictor of retained placental products is an echogenic/hyperechoic mass rather than a thickened endometrium, as well as findings from color Doppler studies.

Another factor to investigate in secondary postpartum hemorrhages is intrauterine infections. Endometritis is almost always a clinical diagnosis, characterized by fever, uterine tenderness without other recognized causes, and purulent discharge from the uterus. These intrauterine infections may result from retained products of conception. Ultrasound findings are often nonspecific, such as a thickened heterogeneous endometrium or even normal postpartum findings.[13,14]

Subinvolution of the placental implantation site should be considered in cases of secondary or recurrent postpartum hemorrhage when intrauterine infection and retained placental products are absent. This condition is primarily caused by the abnormal persistence of dilated placental site vessels that fail to thrombose and obliterate after placental delivery. Ultrasonography may reveal increased vascularity at the previous placental implantation site and increased low-resistance blood flow.[15]

CONCLUSION

In the presented case, secondary postpartum bleeding was attributed to late atony and was managed with uterotonics and intrauterine balloon tamponade following revision curettage.

Secondary postpartum hemorrhage is often an overlooked entity. Due to the timing of its occurrence and its rarity, there is a high probability of misdiagnosis. The underlying mechanism is usually impaired uterine contractility. Various factors may contribute to the development of impaired contractility, as in this case, where late atony was due to adenomyosis and a history of compression suture use. To prevent serious morbidity and mortality, the etiology should be evaluated promptly, and appropriate intervention should be undertaken.

Statement

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