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Case Report

An unsuspected case of uterine arteriovenous malformation with nidus aneurysm following vaginal delivery: Diagnostic challenges and management☆,☆☆

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ABSTRACT

Uterine arteriovenous malformations (AVMs) are rare but potentially life-threatening cause of postpartum hemorrhage (PPH). Accurate differentiation from other PPH causes, such as retained products of conception (RPOC) and gestational trophoblastic disease (GTD), is imperative, as inadvertent improper management such as uterine curettage may cause catastrophic bleeding with high mortality rates. We present the case of 35-year-old woman who presented with excessive vaginal bleeding 2 months postnormal vaginal delivery. Initial ultrasound findings raised suspicion of uterine AVM with a differential diagnosis of type 3 RPOC. Further imaging with computed tomography angiography (CTA) and digital subtraction angiography (DSA) confirmed an underlying uterine AVM with a nidus aneurysm. The patient subsequently was treated with successful embolization and was discharged in stable condition.

Introduction

Uterine artery arteriovenous malformations (AVM) is rare but potentially life-threatening condition. AVMs can be defined as a vascular structural anomaly involving abnormal communication between arteries and veins that bypass the capillary system [1]. Recognition of the condition as the cause of hemorrhage is important, as it can be treated safely and effectively with transcatheter arterial embolization but may be worsened by uterine curettage, precipitating massive uterine bleeding [2]. This case report presents a unique instance of a uterine AVM with a nidus aneurysm in a young woman following vaginal delivery. The case highlights the importance of distinguishing uterine AVM from retained products of conception (RPOC) and gestational trophoblastic disease (GTD),

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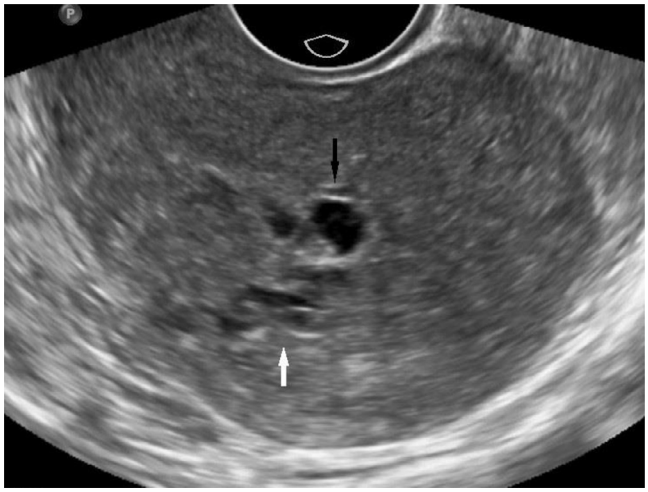

Fig. 1 – Transvaginal Gray scale USG image showing uterus in a retroverted position. Anechoic cystic areas are visible in the upper endometrial cavity (indicated by solid black arrow), with tortuous, serpiginous structures noted in the anterior myometrium (indicated by solid white arrow).

which present with similar symptoms and overlapping imaging findings, but require distinct treatment strategies.

Case Presentation

A 35-year-old woman, gravida 1, para 1, arrived at the emergency department with heavy vaginal bleeding and lower abdominal pain 2 months after giving birth. She had a normal vaginal delivery and a history of 1 previous abortion, which was treated medically. There was no history of dilation and curettage (D&C), cesarean section, or any other uterine surgeries.

Patient was further referred for diagnostic imaging.

Imaging Findings

Transabdominal and transvaginal ultrasound examination revealed cystic areas in the upper endometrial cavity (Fig. 1) showing turbulent swirling intraluminal flow on color doppler study, with few tortuous feeding vessels in anterior myometrium (Fig. 2A and B). Doppler measurements indicated high peak systolic velocity (PSV) of 46.5cm/s and low resistance (resistance index, RI: 0.71) (Fig. 2C and D). The findings raised a differential diagnosis of uterine AVM versus type 3 RPOC.

The patient's negative beta-human chorionic gonadotropin (beta-hCG) result ruled out gestational trophoblastic disease and type 3 RPOC.

To further clarify the diagnosis and plan the management, computed tomography angiography (CTA) was performed, which demonstrated an arterially enhancing lesion within the endometrial cavity with an enhancement pattern following the blood pool dynamics. (Fig. 3A–E). Tortuous enhancing feeder vessels were seen in the anterior myometrium along with early filling of the draining vein, confirming the preliminary diagnosis of uterine AVM with a nidus aneurysm. (Fig. 4A–C).

Digital subtraction angiography (DSA) was performed under local anesthesia in the Interventional Radiology suite. Right common femoral artery puncture was performed and the right uterine artery was identified angiographically which was selectively catheterized using a microcatheter. Selective angiography of the right uterine artery demonstrated tortuous myometrial vessels feeding an aneurysmal nidus in the endometrial cavity, with early venous drainage. (Fig. 5A and B). Embolization of the right uterine artery was achieved using 500–700 μ polyvinyl alcohol (PVA) particles and gelatin sponge, effectively obliterating the aneurysmal nidus. Post-procedure angiography confirmed successful occlusion of the AVM with no remaining vascular blush. (Fig. 6A and B). The left uterine artery was also evaluated and showed no evidence of feeder vessels to the AVM.

Patient had regular follow ups in the clinic with no sign of recurrence.

Discussion

Uterine arteriovenous malformation (AVM) is a rare condition characterized by direct communication between uterine arteries and veins, bypassing the capillary system [1]. The true incidence is not yet known. AVMs may be congenital or acquired [3,4]. The congenital form is relatively uncommon and occurs due to a defect in the development of blood vessels during embryonic growth or an early interruption in the formation of the capillary network, resulting in multiple abnormal connections between arteries and veins [5,6]. These congenital AVMs often penetrate the surrounding tissue and can cause an elaborate collateral vascular network. Furthermore, these congenital lesions can grow as pregnancy progresses [3,7]. However, most of the acquired AVMs occur after uterine tissue damage. Spontaneous miscarriage, dilation and curettage (D&C), Cesarean section, carcinoma of the cervix or endometrium, uterine infection, trophoblastic disease, myomata, endometriosis or exposure to diethylstilbestrol are among the reported causes of AVM [8].

Historically, the diagnosis of AVM was made following laparotomy. Subsequently angiography became the 'gold standard' technique [8]. On angiography true AVMs are recognized by early venous contrast filling of vascular plexus in the endometrium or myometrium. These appear as heterogeneous lesion with arterial phase enhancement following the blood pool. Arterial feeders may be visible, typically arising from 1 or both uterine arteries, with disruption of the endo-myometrial junction, serpentine vessels within the myometrium, and increased vascularity in the parametrial region [4,8]. Computed tomography angiography (CTA) helps in better determining the extent of the lesion in the pelvis and subsequent vascular supply.

More recently, transvaginal ultrasonography with color doppler imaging has been proposed [4,8]. While ultrasonography (USG) is the preferred initial diagnostic tool, it has its limi-

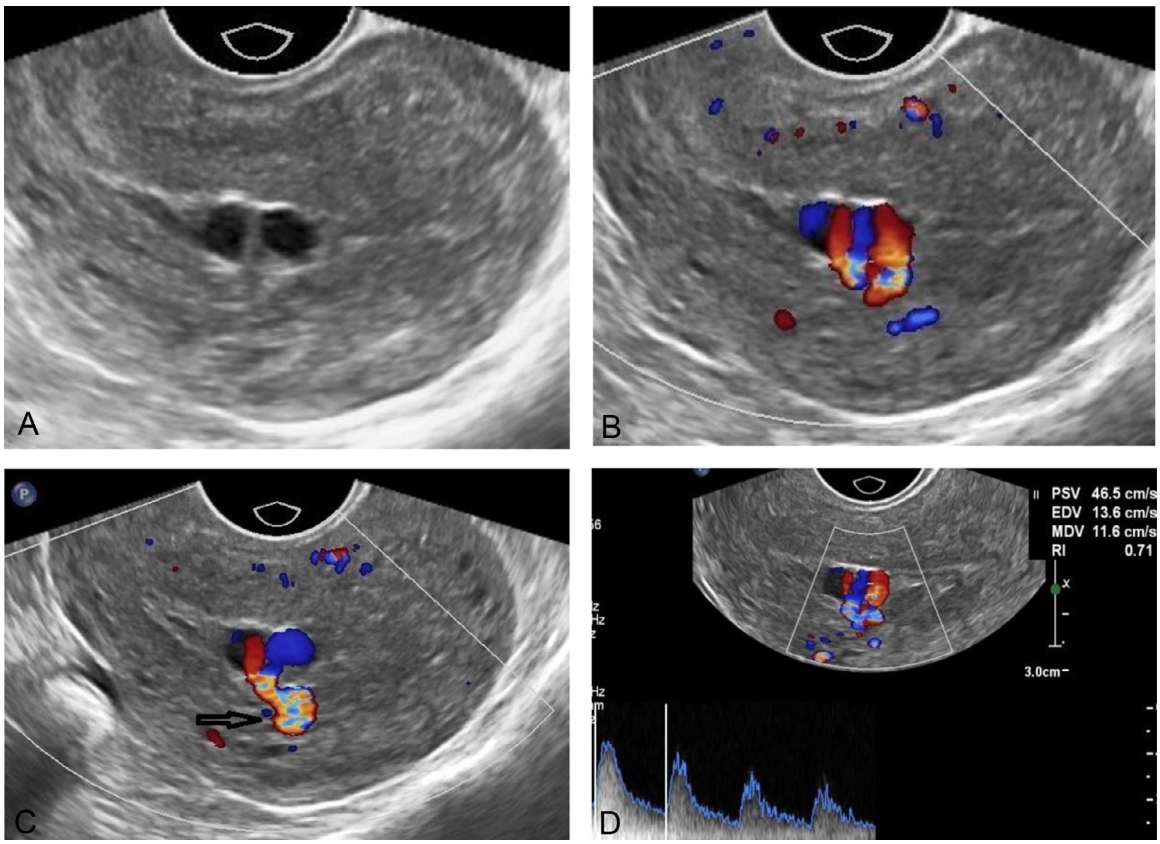

Fig. 2 – (A and B) Gray scale and color doppler ultrasound image of the uterus showing anechoic cystic spaces in the upper endometrial cavity, exhibiting internal swirling color flow suggestive of turbulent blood flow. (C) Color doppler imaging highlights a tortuous feeder vessel in the anterior myometrium (indicated by hollow black arrow) a characteristic feature of AVM. (D) Pulse wave Doppler shows high peak systolic velocity (PSV) with low resistance flow, reinforcing the diagnosis of AVM.

tations. Angiography is now typically reserved for cases where surgical intervention or therapeutic embolization is necessary [8].

The ultrasonographic characteristics are nonspecific and include the presence of hypoechoic tubular structures within the myometrium [9,10]. Color Doppler ultrasound offers a more detailed image, displaying a color mosaic with thickened vessels and reversed blood flow. Pulsed Doppler reveals low-resistance vessels with high pulsatility indices and elevated peak systolic velocity (PSV). In normal myometrium, vessels have a peak systolic velocity ranging from 9 to 40cm/s and a resistance index between 0.6 and 0.8. In cases of uterine arteriovenous malformation, both systolic and diastolic velocities are 4 to 6 times higher [11,12].

Depending on the PSV of the lesions, they can be classified as follows [13,14]:

Mild: PSV <40cm/s, where expectant management is recommended.

Moderate: PSV of 40–60cm/s, where medical treatment is recommended.

Severe: PSV >60–70cm/s, where arterial embolization or surgical treatment is recommended.

Computed tomography scan with contrast, nuclear magnetic resonance imaging, hysterosalpingography and hysteroscopy have also been described for diagnosing AVM.

The differential diagnosis of AVM includes several conditions including RPOC and gestational trophoblastic disease.

Gestational trophoblastic disease and RPOC can have similar presentations with menorrhagia [15]. On imaging, both conditions appear as heterogeneous areas, with the abnormality typically located in the endometrial cavity, while AVMs are centered in the myometrium [16]. GTD may exhibit the loss of the junctional zone with invasion into the surrounding myometrium or parametrium. Beta hCG levels are useful in distinguishing between pregnancy-related and nonpregnancy-related postpartum hemorrhage (PPH). GTD typically presents with elevated beta hCG levels, whereas retained products of conception (RPOC) show a slower decline in beta hCG levels [10]. If beta hCG levels are negative, the possibility of the uterine AVM should be considered [15].

Color doppler ultrasound demonstrates a hyper vascular mass having flow from the endometrium extending into myometrium in RPOC. In AVM, blood flow is mainly concentrated in the myometrium [16], characterized by intense flow and color aliasing, with high peak systolic velocity (PSV) and low resistance index (RI) ranging from 0.2 to 0.5. However, differentiation between the 2 can sometimes be challenging. In such cases, vascular grading of RPOC assists in properly triaging patients to determine the most appropriate management approach [17,18].

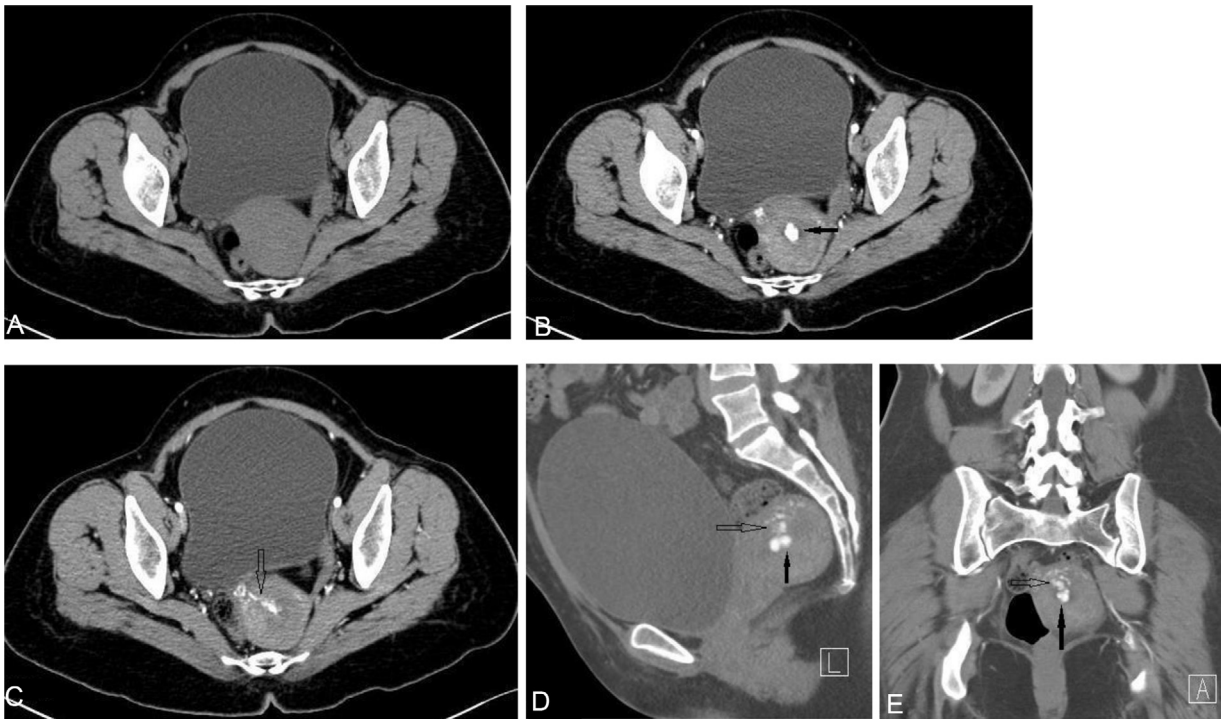

Fig. 3 – CT angiography images in multiple planes demonstrating vascular characteristics of uterine AVM with nidus aneurysm: (A) Noncontrast axial CT image showing no hyperdensity or calcification in the uterus or endometrial cavity. (B) Axial arterial phase image illustrating a bulbous, arterially enhancing lesion within the endometrial cavity that follows the blood pool (indicated by solid black arrow). (C) Tortuous feeding vessels seen in the anterior myometrium (indicated by hollow black arrow). (D and E) Sagittal and coronal reformatted images showing multiple tortuous vessels in the anterior myometrium (indicated by hollow black arrow).and a prominent arterially enhancing lesion in the endometrial cavity, indicating nidus aneurysm (indicated by solid black arrow).

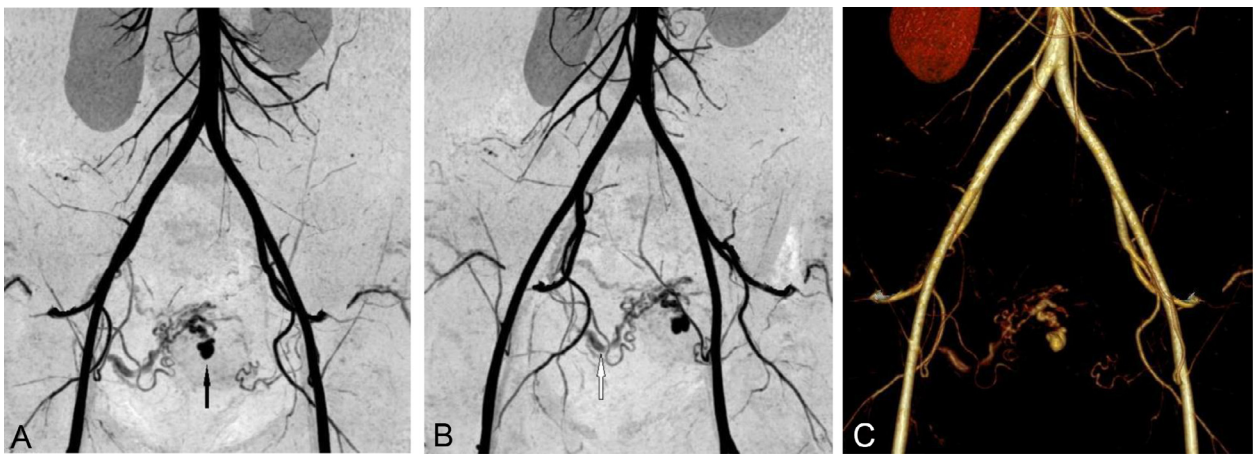

Fig. 4 – Coronal Maximum Intensity Projection (MIP) images demonstrating aneurysmal characteristics in uterine AVM. (A and B) MIP images show an enhancing aneurysmal lesion within the endometrial cavity (indicated by solid black arrow) with associated feeder vessels and an early draining vein (indicated by solid white arrow), consistent with high-flow AVM. (C) 3D reformatting further delineates the aneurysmal focus and adjacent tortuous vessels, aiding in surgical planning.

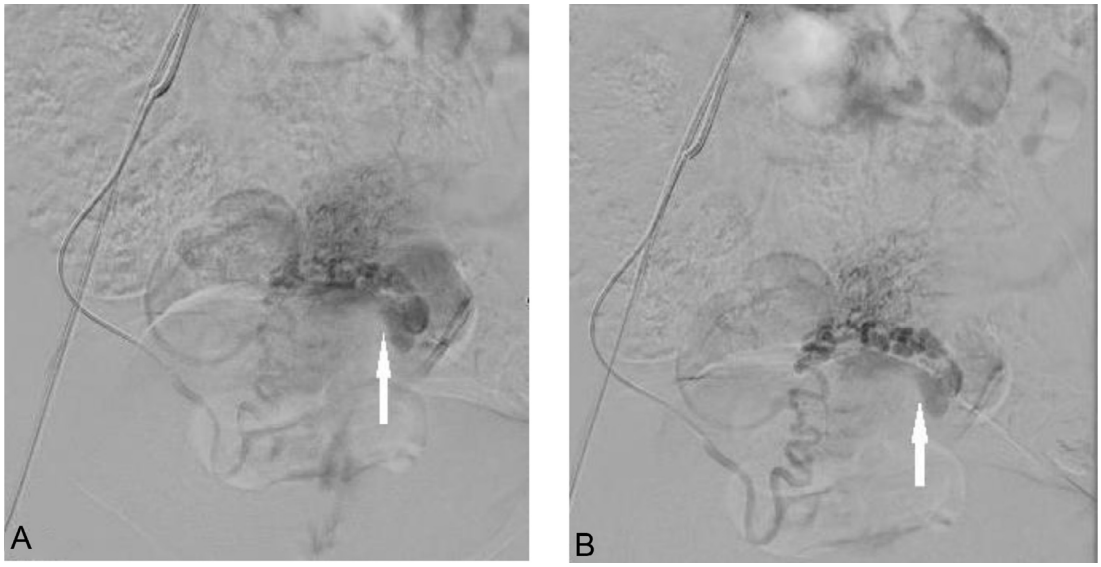

Fig. 5 – (A and B) Digital subtraction angiography (DSA) images with selective catheterization of the right uterine artery showing classic features of AVM with nidus aneurysm: Tortuous myometrial vessels feeding into a bulbous, enhancing aneurysmal structure within the endometrial cavity, with an early-draining vein (indicated by solid white arrow) confirming the presence of a high-flow arteriovenous shunt.

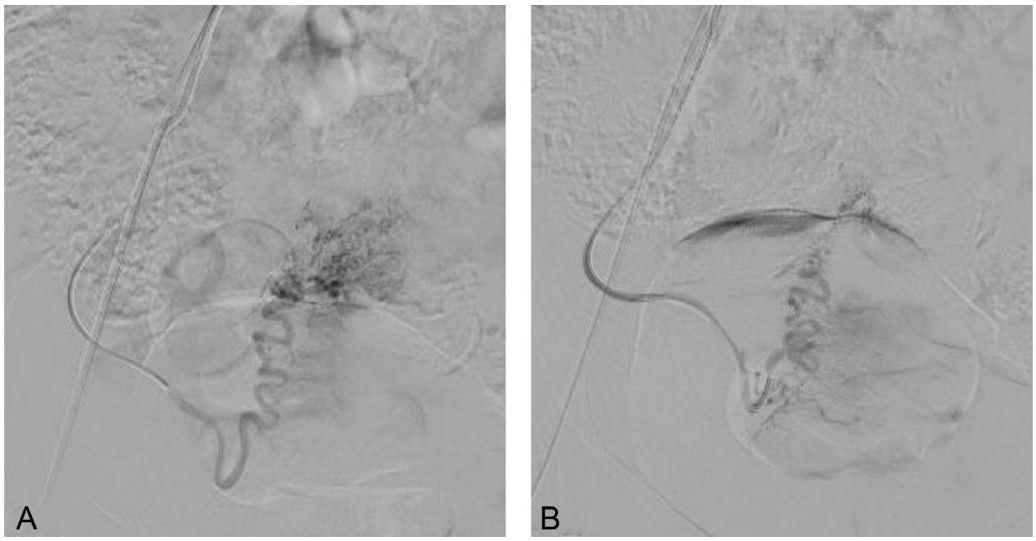

Fig. 6 – (A and B) Postembolization check angiogram showing the occluded nidus aneurysm with no contrast filling, indicating successful treatment.

On color and duplex Doppler ultrasound, pseudoaneurysms appear as cystic structures filled with blood, displaying swirling arterial flow. In contrast, arteriovenous malformations (AVMs) show a dense network of vessels with low-resistance and high-velocity arterial flow. When both conditions occur together, the ultrasound will reveal characteristics of both: a vascular tangle with intense flow indicative of an AVM, alongside the swirling blood flow seen in a pseudoaneurysm [2].

In our case the abnormality was centered in the endometrial cavity, but the patient had normal beta HCG levels. More so, there were associated tortuous myometrial feeder vessels with large vascular anechoic areas in the endometrial cavity showing swirling internal flow, thus making the possibility of RPOC and GTD less likely. Additionally, there was aneurysmal dilatation of the nidus with active contrast extravasation.

Until recently, the only treatment options for AVM were hysterectomy or (bilateral) ligation of the internal iliac arteries [3,4,8]. However, in recent years, there has been a growing trend toward treating these cases with transcatheter embolization of the uterine arteries [8,13]. This approach allows the menstrual cycle to remain unaffected, and it preserves the possibility of pregnancy and normal vaginal delivery [8].

Thus, appropriate history, imaging findings along with negative serum beta hCG helps in confirming the diagnosis.

Conclusion

This case highlights the importance of considering uterine AVM as a differential diagnosis in patients with intractable postpartum bleeding and differentiating it from other causes of postpartum hemorrhage. In the case of dilated vascular areas within the lesion, possibility of the nidus aneurysm should be considered. The use of combined imaging techniques, including Doppler ultrasound, CT angiography, and DSA, allowed for accurate diagnosis and minimally invasive treatment. Early identification and treatment of uterine AVM are essential to prevent severe hemorrhage and reduce the risk of future complications. Transcatheter embolization proved to be an effective treatment in this case, preserving fertility and normal uterine function.

Patient consent

Informed consent was obtained from the patient for her data and images to be used in this study and published in this journal. The patients understood that their anonymity would be preserved, and no identifiable information would be disclosed.

Author Contribution

All authors contributed to the planning, literature review, and creation of this case report. Each author has reviewed and approved the final version of the manuscript.

Ethical Guidelines

Not applicable.

Ethics Approval and Consent to Participate

Ethics committee approval was not necessary for this type of case report at our facility.

Availability of Data and Materials

Not applicable.

REFERENCES

[1] Lowe LH, Marchant TC, Rivard DC, Scherbel AJ. Vascular malformations: classification and terminology the radiologist needs to know. InSeminars in roentgenology 2012;47(2):106–17. doi:10.1053/j.ro.2011.11.002.

[2] Kwon Jung Hyeok, Kim Gi Sung. Obstetric Iatrogenic Arterial Injuries of the Uterus: Diagnosis with US and Treatment with Transcatheter Arterial Embolization. RadioGraphics 2002;22(1):35–46 org (Crossref). doi:10.1148/radiographics.22.1.g02ja0735.

[3] Beller U, Rosen RJ, Beckman EM, Markoff G, Berenstein A. Congenital arteriovenous malformation of the female pelvis: a gynecologic perspective. American journal of obstetrics and gynecology 1988;159(5):1153–60. doi:10.1016/0002-9378(88)90435-8.

[4] Ghosh TK. Arteriovenous malformation of the uterus and pelvis. Obstetrics & Gynecology 1986;68(3):40S -3S. PMID: 3737074.

[5] Kasznica John, Nisar Nauman. Congenital Vascular Malformation of the Uterus in a Stillborn: A Case Report. Human Pathology 1995;26(2):240–1 *DOI.org (Crossref)*. doi:10.1016/0046-8177(95)90043-8.

[6] Jain KA, Jeffrey RB Jr, Sommer FG. Gynecologic vascular abnormalities: diagnosis with Doppler US. Radiology 1991;178(2):545–9. doi:10.1148/radiology.178.2.1987622.

[7] Geary M, McParland P. Multiple and Massive Arteriovenous Malformations in Pregnancy. European Journal of Obstetrics, Gynecology, and Reproductive Biology 1996;64(1):147–50 PubMed. doi:10.1016/0301-2115(95)02271-6.

[8] Timmerman D, Wauters J, Van Calenbergh S, Van Schoubroeck D, Maleux G, Van Den Bosch T, et al. Color Doppler imaging is a valuable tool for the diagnosis and management of uterine vascular malformations. Ultrasound in obstetrics & gynecology 2003;21(6):570–7 PubMed. doi:10.1002/uog.159.

[9] Clavero Bertomeu L, Castro Portillo L, Fernández-Conde de Paz C. Uterine Arteriovenous Malformation: Diagnostic and Therapeutic Challenges. Diagnostics 2024;14(11):1084 DOI.org (Crossref). doi:10.3390/diagnostics14111084.

[10] Yoon DJ, Jones M, Al Taani J, Buhimschi C, Dowell JD. A systematic review of acquired uterine arteriovenous malformations: pathophysiology, diagnosis, and transcatheter treatment. American Journal of Perinatology Reports 2016;6(01):e6–14 DOI.org (Crossref). doi:10.1055/s-0035-1563721.

[11] Moynihan HV, Richardson J, Loveridge K. Fertility-preserving management of uterine arteriovenous malformation in a 16-year-old female. Cureus 2021;13(9). doi:10.7759/cureus.18162.

[12] Timmerman D, Van den Bosch T, Peeraer K, Debrouwere E, Van Schoubroeck D, Stockx L, et al. Vascular malformations in the uterus: ultrasonographic diagnosis and conservative management. European Journal of Obstetrics & Gynecology and Reproductive Biology 2000;92(1):171–8 DOI.org (Crossref). doi:10.1016/S0301-2115(00)00443-7.

[13] Hoang VT, Van HA, Trinh CT, Pham NT, Huynh C, Ha TN, et al. Uterine arteriovenous malformation: a pictorial review of diagnosis and management. Journal of Endovascular Therapy 2021;28(5):659–75 DOI.org (Crossref). doi:10.1177/15266028211025022.

[14] Giurazza F, Corvino F, Silvestre M, Cavaglià E, Amodio F, Cangiano G, et al. Uterine arteriovenous malformations. InSeminars in Ultrasound, CT and MRI. WB Saunders 2021;42(1):37–45. doi:10.1053/j.sult.2020.08.002.

[15] Cura M, Martinez N, Cura A, Dalsaso TJ, Elmerhi F.

Arteriovenous malformations of the uterus. Acta Radiologica 2009;50(7):823–9. doi:10.1080/02841850903008792.
[16] Sellmyer MA, Desser TS, Maturen KE, Jeffrey RB Jr, Kamaya A. Physiologic, histologic, and imaging features of retained products of conception. Radiographics 2013;33(3):781–96 DOI.org (Crossref). doi:10.1148/rg.333125177.
[17] Nakasholo T, Khan N, Dunn Z, Snyman L, Ismail SM. Uterine arteriovenous malformations, clinical and radiological considerations: A report of two cases. Radiology case reports 2021;16(7):1924–9. doi:10.1016/j.radcr.2021.02.018.
[18] Sridhar D, Vogelzang RL. Diagnosis and treatment of uterine and pelvic arteriovenous malformations. Endovasc Today 2018;17(1):73.