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Case Study

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ACQUIRED UTERINE ARTERIOVENOUS MALFORMATION: CASE REPORT AND UPDATE ON MEDICAL AND ENDOSCOPIC TREATMENT

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ABSTRACT

Introduction: Uterine arteriovenous malformations (AVM) are extremely rare, is characterized by an abnormal direct connection between an arterial network and a venous network, without the involvement of capillaries . Acquired uterine arteriovenous malformations (UAVMs) are rarely discussed in the context of persistent metrorrhagia following a complicated pregnancy. It is crucial to consider them to avoid potentially dangerous and unnecessary hemostatic curettage for the patient. Here, we describe a case of acquired intrauterine arteriovenous malformations occurring after abortion. **Case presentation:** A 29-year-old woman with no significant medical history experienced severe postpartum metrorrhagia following a recent spontaneous abortion. She required hospitalization and two blood transfusions. Diagnostic imaging (Doppler ultrasound, CT angiography, and arteriography) revealed a fundal arteriovenous malformation, primarily supplied by the left uterine artery. Selective embolization with non-resorbable particles and coils was performed. Postoperative imaging showed significant devascularization of the AVM, and follow-up indicated resolution of metrorrhagia, irregular but normal menstrual cycles, and complete disappearance of the malformation within one month. **Discussion:** Uterine arteriovenous malformations can be congenital or acquired, presenting with symptoms such as bleeding, pain, and fertility issues. Congenital AVMs are associated with genetic syndromes, while acquired AVMs often result from trauma or medical procedures. Diagnostic tools, including ultrasound and Doppler, provide detailed views of these vascular anomalies, aiding in accurate diagnosis and management. MRI and angiography further assist in evaluating the lesions. Treatment varies based on lesion characteristics, with hysterectomy considered a last resort. Minimally invasive methods, such as embolization, are preferred due to their high success rates and ability to preserve fertility. Medical management of symptomatic uterine AVM is a reasonable approach in a well selected patient who is hemodynamically stable and has reliable follow-up. **Conclusion:** AVMs are a rare but potentially serious cause of life threatening vaginal bleeding. A high index of suspicion is needed so that a prompt diagnosis can be made. Acquired AVMs should be considered when metrorrhagia persists in the postpartum period. Early diagnosis through pelvic ultrasound combined with Doppler allowed for conservative treatment by embolization in our patient.

KEYWORDS: Uterine arteriovenous malformations, ultrasound with color Doppler, arterial embolization, medical treatment, fertility.

INTRODUCTION

An arteriovenous malformation (AVM) is characterized by an abnormal direct connection between an arterial network and a venous network, without the involvement of capillaries.[1] And may be either congenital or acquired. Acquired uterine arteriovenous malformations (UAVMs) are rarely discussed in the context of persistent metrorrhagia following a complicated pregnancy (miscarriage, trophoblastic diseases).[1] It is crucial to consider them to avoid potentially dangerous and unnecessary hemostatic curettage for the patient.

The actual incidence of acquired uterine arteriovenous malformations is difficult to determine. Fewer than 150 cases have been reported in the literature since 1926, with only 73 cases documented before 1997.[2]

In the prospective study by Hiroyuki Yazawa, which included 959 postpartum or post-abortion patients, only one patient developed a UAVM.[3] The study by O'Brien reported an incidence of 4.5% of UAVM in a population of 464 patients.[4] UAVMs account for 1 to 2% of pelvic bleeding cases.[2]

Here, we describe a case of acquired intrauterine arteriovenous malformations occurring after abortion, observed in the gynecology-obstetrics department of the Centre Ibn Sina. Metrorrhagia was the dominant symptom, with the diagnosis suspected by Doppler ultrasound and confirmed by CT scan. The treatment consisted of embolization, resulting in favorable outcomes for the patients.

CASE REPORT

The patient is a 29-year-old woman with no significant medical history, G2P1: G1: vaginal delivery in 2016 and G2: spontaneous abortion at 3 months one month ago. She presented with severe postpartum metrorrhagia that required hospitalization and two blood transfusions totaling 5 units. Obstetric examination indicated an endometrial source of bleeding.

Doppler ultrasound, CT angiography, and arteriography revealed a fundal arteriovenous malformation with a loosely compact nidus, fed mainly by branches from the left uterine artery and secondarily from the right uterine artery. Venous drainage was directed quickly to the left uterine vein and then to the ipsilateral iliac vein.

Selective uterine embolization using non-resorbable particles was performed. Selective catheterization was done with an Echelon 14 microcatheter and Avigo microguide from the left, then right uterine arteries, with several coils placed. Postoperative ultrasound the next day showed significant devascularization of the vascular anomaly. The follow-up showed cessation of metrorrhagia, normal but irregular cycles, and complete disappearance of the AVM at one month.

DISCUSSION

Intrauterine arteriovenous malformations are complex vascular anomalies forming masses composed of arteries and veins of varying calibers, interconnected by multiple fistulas, resulting in turbulent blood flow. These malformations are considered a defect of the high-flow, low-resistance vascular system, where the normal vascular pattern of capillary networks separating arteries and veins is absent.[5]

Arteriovenous malformations (AVMs) are characterized by abnormal direct connections between venous and arterial vessels without capillary intermediaries. Histologically, they present as fibrous nodules with multiple vessels, including veins and arteries, scattered in the myometrial wall, serosa, and endometrium. Vessel sizes and shapes vary, and necrotic membranes are common.[2,6] AVMs can be congenital or acquired.

- Congenital AVMs are often linked to genetic syndromes like Rendu-Osler disease and arise from embryogenetic defects in genes such as ALK1, RASA1, and PTEN.[2,6] They typically involve extensive vascular networks extending beyond the uterus into the pelvis, often remaining asymptomatic and detected through obstetric history.[7,8]
- Acquired AVMs generally involve fewer or unilateral nourishing arteries and lack a distinct nidus. Histological examination reveals small arteriovenous fistulas connecting intramural arterial branches with myometrial venous plexuses.[7,8] Acquired AVMs are often caused by trauma from intrauterine procedures like curettage, cesarean sections, or myomectomies, which damage or weaken vessel walls, leading to abnormal vessel communication during healing. Other causes include endometrial or cervical cancers, chronic infections, trophoblastic disease, or uterine scarring. Fibroids can also be associated with AVMs due to vascular changes around them.[2,6,9,10] The main suspected cause of acquired AVMs is intrauterine trauma, as supported by our limited case series.

Arteriovenous malformations (AVMs) are most commonly observed in women of childbearing age and rarely in those who have never been pregnant.[11] Symptoms include metrorrhagia and vaginal bleeding, which can be gradual or sudden, often following a traumatic uterine procedure. Severe bleeding can lead to anemia or hemorrhagic shock. Complications may include pelvic varicocele, dyspareunia, pelvic pain, recurrent miscarriages, and infertility due to increased vascularization affecting embryo implantation. Urinary symptoms like incontinence, polyuria, and pollakiuria can also occur. Puberty, pregnancy, thrombosis, infections, biopsies, and trauma can exacerbate symptoms.[12] Severe AVMs may cause cardiovascular issues, with 30% of patients needing blood transfusions.[8] AVMs can also be asymptomatic.[2] During examination, a closed cervix indicates acquired AVM bleeding, while an open cervix suggests trophoblastic retention. Frenken[5] notes that the uterus may feel soft, slightly enlarged, with a pulsatile mass and thrill, though clinical examination lacks specificity.

The beta-HCG hormone level can aid in diagnosing acquired AVMs if it is negative in the presence of a myometrial vascular lesion, though it may be slightly positive after uterine curettage post-miscarriage or abortion, complicating differentiation.[13] For imaging, several techniques are used.

Ultrasound and Color Doppler: Pelvic ultrasound with color Doppler is the primary diagnostic tool for AVMs.[14] It reveals dense, irregular intra-myometrial lesions with hypo- or anechogenic islands, and Doppler color shows multidirectional myometrial hypervascularization with turbulent flow.[12]

Pulsed Doppler: This technique shows vessels with low resistance and high pulsatility indices. Normal systolic peak velocity (PSV) ranges from 9-40 cm/s in healthy myometrium, while AVMs have PSV between 25-110 cm/s with an average of 60 cm/s.[13] Classification is based on PSV: mild (<40 cm/s), moderate (40-60 cm/s), and severe (>60-70 cm/s).[13] CT Scan: AVMs appear with similar density to myometrium and are used

primarily when MRI is contraindicated, mainly in emergencies.[2]

MRI: MRI is effective for precise diagnosis and localization, showing serpiginous intra-myometrial masses and diffuse signal voids in T1, with homogeneous hypersignal in T2.[15] It helps distinguish acquired AVMs from malignant arteriovenous shunts.[16] Angiography: The gold standard for diagnosing AVMs, angiography visualizes the malformation, including feeding and draining vessels, using contrast to reveal the AVM structure.[17] Digital subtraction angiography offers reduced radiation exposure and allows 3D reconstructions.[18] Angio-MRI and angio-CT are non-invasive alternatives for diagnosis but angiography remains essential for therapeutic embolization.[13]

The choice of treatment for patients with uterine arteriovenous malformations (AVMs) depends on the age, size, and location of the lesion, hemodynamic stability, the desire to preserve fertility, and the maximal systolic velocity (PSV) of the lesion on ultrasound. If the PSV is less than 0.39 cm/s, the AVM has a good chance of spontaneous regression. However, if the PSV exceeds 0.83 cm/s, medical or surgical treatment should be considered.[19] Spontaneous regression has been reported in rare cases.[20] In the case of mild bleeding that is well-tolerated by the patient and an asymptomatic uterine arteriovenous malformation (AVM), a medical treatment may be proposed, A meta-analysis conducted in 2021 showed the following success rates for each medical treatment.[21] Progestin 82.5% (Medroxyprogesterone acetate intramuscular every 3 months Oral norethisterone 10 mg p.o. twice); Gonadotropin-releasing hormone agonist 89.2% (Leuprolide acetate or goserelin ‡ letrozole 2.5 mg daily orally x 5 days on initiation); Chemotherapeutic 90.9% (Methotrexate intramuscular single dose or weekly); Combined hormonal contraception 42.9% (Various doses of ethinyl estradiol + progestin orally); Uterotonics 100% (Methylergonovine maleate intramuscular or intravenous followed by daily oral dose Methylergonovine maleate 0.2-0.5 mg orally daily); Danazol 66.6% (Danazol 200-400 mg orally daily).

However, patients with a PSV greater than 83 cm/s generally require surgical intervention. Uterine artery embolization by angiography is now the preferred method, as it is minimally invasive and preserves fertility. This procedure uses angiography to visualize the vascular axis through contrast injection. The vessel is then embolized. Embolization of both uterine arteries is necessary to prevent persistent or recurrent bleeding, as the uterine artery has many collaterals that can lead to reperfusion from the contralateral uterine artery.[5,7] Frequently used embolization materials include gelatin particles, polyvinyl alcohol (PVA), coils, adhesives, detachable balloons, and N-butyl-2-cyanoacrylate.[7,22] Gelatin particles cause temporary mechanical vessel obstruction, allowing recanalization in a few weeks, which increases the risk of reperfusion.[23] Conversely, PVA causes permanent occlusion by adhering to vessel walls. Coils, although permanent, can migrate and occlude non-target vessels. N-butyl-2-cyanoacrylate (NBCA) and ethylene-vinyl alcohol copolymer (Onyx) are used for treating uterine and cerebral arteriovenous malformations, with varying success rates.[24]

Literature on the type of material used for acquired AVMs is limited, but selective embolization appears essential to preserve ovarian function and, consequently, fertility. Particles larger than 500 µm are recommended to avoid the theoretical risk of unintended embolization of the uterine-ovarian anastomosis, which could lead to ovarian ischemia.[20] Embolization techniques often combine absorbable particles, like gelatin sponges, and non-absorbable materials for treating uterine arteries. Camacho observed a success rate of 94.4% with gelfoam particles, using microcoils to occlude large vessels while preserving microvascularization and ensuring immediate sealing with absorbable materials.[25]

Embolization can be repeated in case of partial response but this is not considered a failure of the initial embolization. The success rate of embolization is 61% after a single procedure and 93% after a repeat procedure.[26] Post-embolization PSV values seem to predict the need for iterative embolization.[27] Congenital AVMs have a lower success rate due to their extent and potential involvement in other pelvic and extrapelvic areas, often presenting multiple feeding arteries and draining veins, making the procedure more complex.

The advantages of arterial embolization include a higher success rate with a low complication rate, as shown in Ghai et al.'s study: a success rate of 93% with a complication rate of 0.4%[1], and avoidance of surgical risks. Side effects related to the procedure include mild fever, pelvic pain, groin hematomas, uterine ischemia, pelvic infections such as tubo-ovarian abscesses, endometritis, as well as transient or permanent amenorrhea.[7] Other severe but extremely rare complications have also been reported, most often related to embolization of the internal iliac artery, causing skin necrosis, neurological deficits, and rectovaginal fistulas.[1]

Hysterectomy is generally considered as a last resort, with a utilization rate of 7.5%[20], except in cases of severe hemodynamic failure. Minimally invasive techniques include laparoscopic occlusion of the internal iliac arteries and bipolar coagulation of the uterine arteries. Gene therapy is considered for AVMs associated with endothelial dysfunction. Hysteroscopy, effective and complication-free, is performed in 30 minutes on an outpatient basis, with more than half of patients conceiving within 6 months.[28]

Embolization does not affect menstrual cycles or pregnancy.[8] Menstrual cycles are expected to resume

within one to two months after selective embolization of uterine arteriovenous malformations (AVMs).[29] Regarding fertility preservation, several reports mention successful intrauterine pregnancies following embolization of the uterine arteries: Poppe et al. demonstrated normal placental flow on Doppler ultrasound in pregnant patients who had undergone pelvic embolization.[8] Peitsidis et al.[7] conducted a systematic review of case reports on uterine AVM treatments and identified 17 pregnancies (17%) following uterine artery embolization (UAE) for treating uterine AVMs. Maleux et al.[7] estimated that the average time between UAE and the next pregnancy was 15.6 months in their report. However, there is an increased risk of intrauterine growth restriction and prematurity.[19]

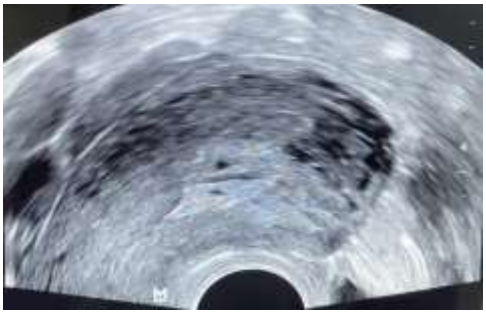

Figure 1: Hypoechoic intramyometrial mass (Image: Souissi Maternity Hospital).

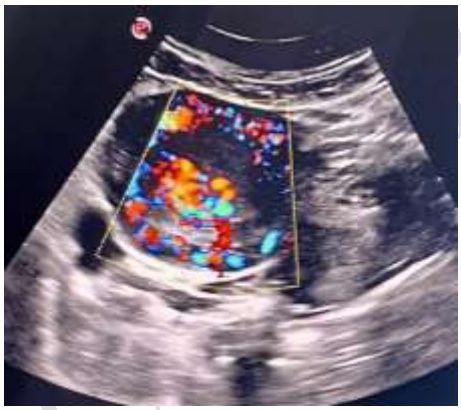

Figure 2: Transvaginal color Doppler ultrasound image showing multiple vascular channels within the myometrium, revealing a mosaic color pattern that may suggest an arteriovenous malformation (Image: Souissi Maternity Hospital).

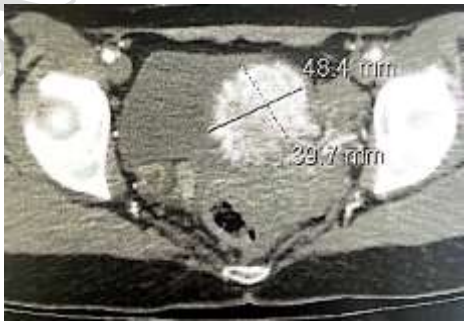

Figure 3: Pelvic CT scan in axial section. (Image: Souissi Maternity Hospital).

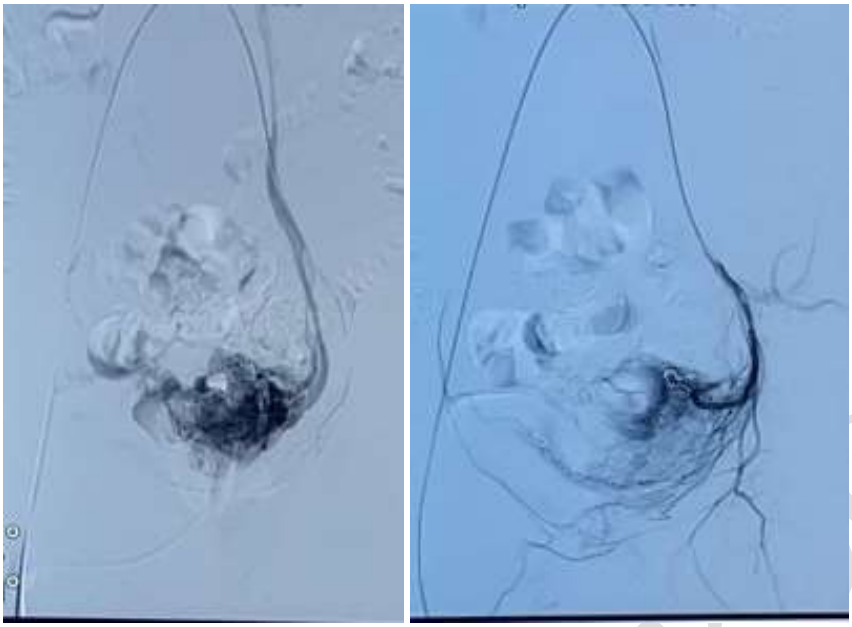

Figure 4: Angiography showing the nidus of the malformation, the feeding artery, and the draining vein of the uterine arteriovenous malformation. (Image: Souissi Maternity Hospital).

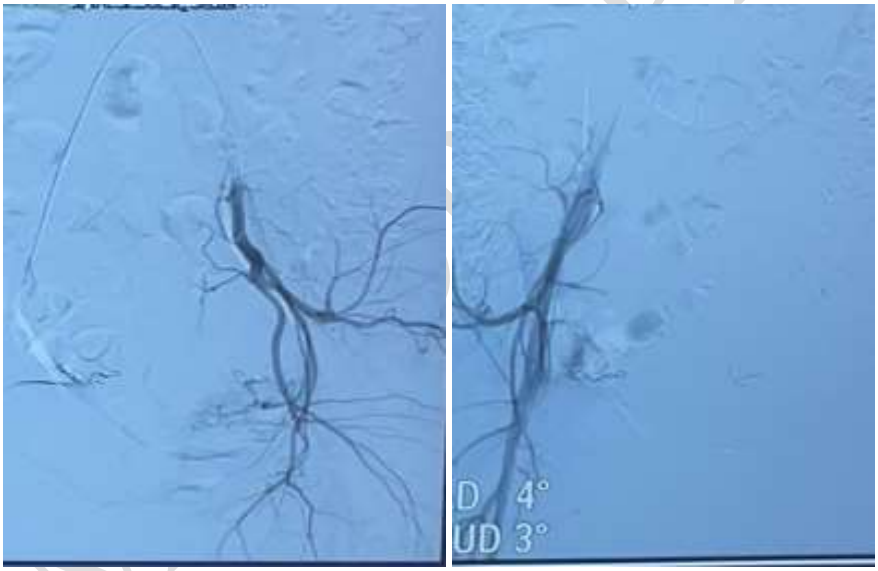

Figure 5: The final control on the arteriography shows the exclusion of the AVM. (Image: Souissi Maternity Hospital).

CONCLUSION

AVMs are a rare but potentially serious cause of life threatening vaginal bleeding. A high index of suspicion is needed so that a prompt diagnosis can be made. Acquired AVMs should be considered when metrorrhagia persists in the postpartum period. Early diagnosis through pelvic ultrasound combined with Doppler allowed for conservative treatment by embolization in our patient.

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