Section: Vascular Surgery

Case Series

UTERINE ARTERY EMBOLISATION IN UTERINE AV MALFORMATION – A CASE SERIES

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ABSTRACT

Background: Uterine arteriovenous malformations (UAVMs) are rare but potentially life-threatening vascular anomalies in women of reproductive age, often presenting with acute or chronic heavy uterine bleeding. Most cases are acquired, usually following obstetric procedures, uterine trauma, or surgery, and may lead to severe haemorrhage if not promptly recognised. Diagnosis is challenging because the symptoms can mimic other causes of abnormal uterine bleeding. However, ultrasonography with colour Doppler is the preferred initial tool, supported by CT or MR angiography to define the lesion and its vascular supply. Digital subtraction angiography (DSA) remains the gold standard, allowing both definitive diagnosis and treatment of this condition. Uterine artery embolisation (UAE) has emerged as the mainstay of therapy, offering effective haemorrhage control while preserving fertility, in contrast to hysterectomy, which is definitive but non-fertility-sparing. This case series reviewed five cases of UAVMs, along with their clinical presentation, diagnostic evaluation, treatment with UAE, and follow-up outcomes.

INTRODUCTION

Uterine arteriovenous malformations (UAVMs) are rare, life-threatening vascular anomalies in women of reproductive age, characterised by direct arteriovenous shunting without an intervening capillary network.[1] UAVM can be congenital or acquired, while most of the reported cases are acquired.[2] They commonly present with heavy or irregular uterine bleeding and may occur spontaneously or following conditions like uterine trauma, surgery, obstetric events, or trophoblastic disease.[1] Diagnosis of UAVMs relies on a multimodal imaging strategy, such as initial grayscale and colour-spectral Doppler ultrasound (USG), 3D CTA to map vascular anatomy and feeders, MRI for soft-tissue contrast, and digital subtraction angiography (DSA) as the invasive gold standard, helping both diagnosis and embolisation.[3] The uterine artery embolisation (UAE) is a preferred alternative to hysterectomy in symptomatic cases due to its minimally invasive nature and uterine preservation potential.[1]

In resource-limited settings, embolic agents such as polyvinyl alcohol, gelatin sponge, or glue have also produced haemostatic and reproductive success, including resumption of menstruation and conception in post-pregnancy UAVMs.[4,5] Although complications such as post-embolisation syndrome and non-target embolisation exist, studies support UAE as a safe, effective, and fertility-sparing treatment for symptomatic UAVMs.[6] In this case series, we describe five cases of UAVMs, along with their clinical presentation, diagnostic evaluation, treatment with UAE, and follow-up outcomes.

CASE PRESENTATION

Case 1: A 24-year-old woman (P1L1), with a history of lower segment caesarean section (LSCS) and last childbirth (LCB) 10 months prior, presented with sudden heavy menstrual bleeding (HMB) of 15 days' duration. The urine pregnancy test (UPT) was negative, and the serum β-hCG level was 1.27mIU/ml. USG with Doppler revealed a uterine AVM with a feeding vessel in the posterior myometrium. CT angiography revealed an arteriovenous malformation (AVM) in the posterior wall adjacent to the fundus (3.6 × 2.5 cm), supplied by both uterine arteries [Figure 1]. Her haemoglobin

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level was 8.8 g/dL, and she received one PRBC transfusion. Bilateral UAE was performed via right CFA retrograde access under local anaesthesia using a 6Fr 11 cm sheath, 6Fr pigtail catheter, and 5Fr vert catheter over an 0.035 wire. The uterine artery was selectively cannulated using a Progreat microcatheter, and 500–750 μm PVA particles with lipiodol were injected into the right uterine artery until flow reduction was achieved, followed by gel foam injection [Figure 2]. The same procedure was performed on the left uterine artery. The 45-minute procedure resulted in complete occlusion, and the bleeding ceased. At 4 weeks, she developed amenorrhoea, but no complications were observed during the 6th or 12th months of follow-up, and by Day 3 of the 12th month, the FSH level was 10.2 mIU/ml. During the 16th month follow-up, USG showed no residual AVM with normal periods, preserved ovarian function, and complete recovery.

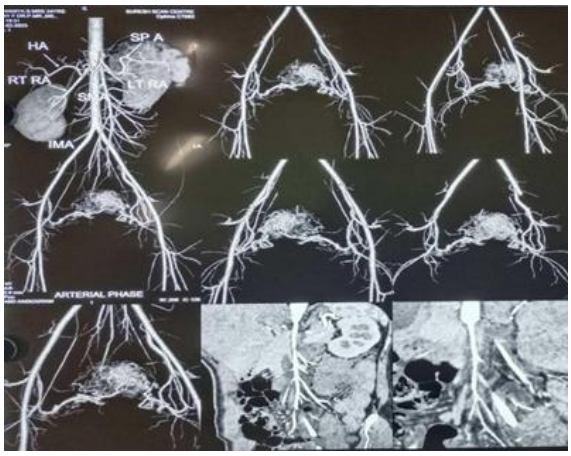

Figure 1: CT angiogram

Figure 2: Fluoroscopic image during UAE demonstrating selective cannulation of the left uterine artery with a microcatheter and PVA particle injection

Case 2: A 33-year-old woman (P2L2A1) with a history of normal vaginal deliveries (NVD) and a recent medical termination of pregnancy (MTP) with a temporary abortion trial (TAT) before 1.5 months, presented with sudden HMB for 15 days. The UPT was negative, serum β-hCG 0.9mIU/ml. Pelvic MRI revealed a heterointense lesion (6.2 × 3.9 cm) with multiple flow voids, and CT angiography confirmed a uterine AVM in the posterior anterolateral myometrium and endometrial cavity, supplied by both uterine arteries [Figure 3]. The haemoglobin level was 7.9 g/dL, and two PRBC transfusions were administered. Bilateral UAE was performed via right CFA retrograde access with a 6Fr 11 cm sheath, 6Fr pigtail catheter, and 5Fr vert catheter over a 0.035 wire. Selective cannulation of the uterine arteries was achieved using a Progreat microcatheter. PVA particles (500–750 μm) mixed with lipiodol were injected into the right uterine artery until reduced flow was observed, followed by gel foam injection [Figure 4]. The same procedure was performed on the left uterine artery, and the procedure lasted for 45 min. Angiography confirmed the occlusion and cessation. On follow-up, she had amenorrhoea at 4 weeks, normal periods at 6 months, and no AVM on 10-month USG with full recovery.

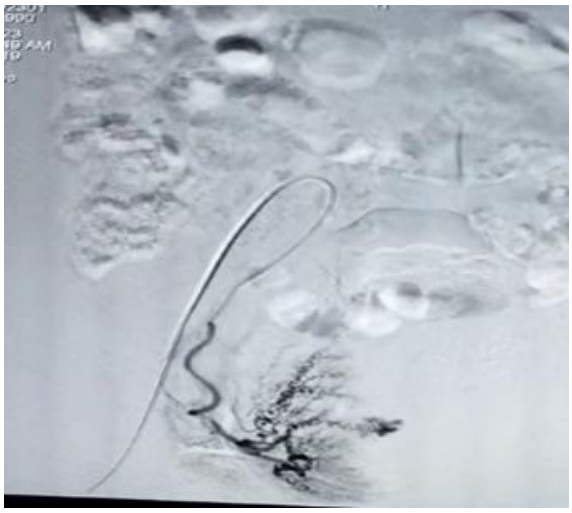

Figure 3: DSA shows right uterine AVM

Figure 4: Image showing a catheter positioned during UAE and PVA particles injection

Case 3: A 28-year-old woman (P1L1A1) who had undergone a first-trimester abortion 2 months earlier presented with HMB lasting 21 days that was unresponsive to tranexamic acid and contraceptive pills. The UPT was negative, serum β-hCG

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0.7mIU/ml. USG with Doppler revealed a uterine AVM with a feeding vessel, and CT angiography demonstrated a posterior myometrial AVM (2.5 × 2 cm) supplied by both uterine arteries [Figure 5]. The patient's haemoglobin level was 7.5 g/dL; therefore, the patient required two PRBC transfusions. Bilateral UAE was performed via the right CFA retrograde access using a 6Fr sheath, pigtail, and vert catheter over an 0.035 wire. The uterine arteries were selectively cannulated using a Progreat microcatheter. PVA particles (500–750 μm) mixed with lipiodol were injected until a reduced flow was noted, followed by gel foam injection [Figure 6]. The same procedure was performed on the left uterine artery. The procedure lasted for 45 min, complete occlusion was achieved, and bleeding ceased. At 4 weeks, she developed amenorrhoea, and at 6 months follow-up, the FSH on the 3rd day was 11.4 mIU/ml; USG showed no AVM with normal menses and complete recovery.

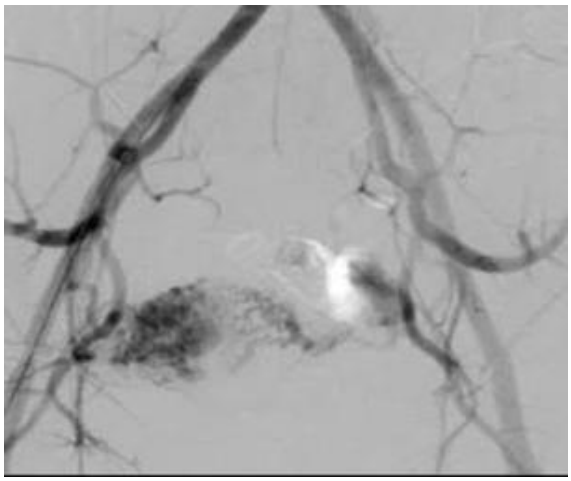

Figure 5: DSA shows Bilateral uterine artry AVM

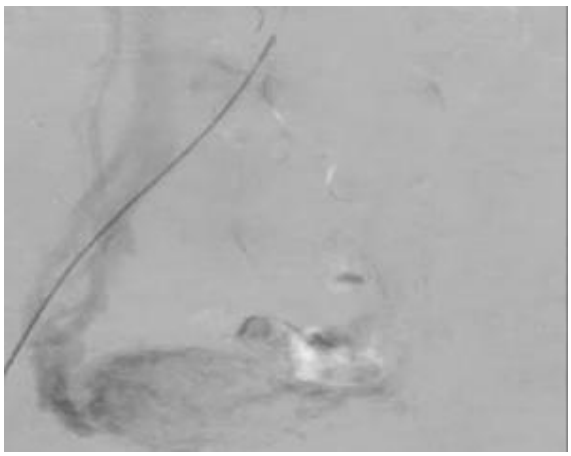

Figure 6: Intra-procedural fluoroscopy illustrating the gel foam and PVA injections

Case 4: A 30-year-old woman (P3L3), with NVD and LCB 2.5 years prior, presented with HMB for 2 months, and the serum β-hCG was 55.64mIU/ml. USG with Doppler identified a uterine AVM with a feeding vessel, and MRI of the pelvis showed a posterior myometrial AVM (2.6 × 3.2 cm) with a small hematometra (10 cc), supplied by both uterine arteries. Bilateral UAE was performed via the right CFA retrograde access with a 6Fr sheath, pigtail, and vert catheter, followed by selective uterine artery cannulation using a Progreat microcatheter. PVA particles (500–750 μm) mixed with lipiodol were injected into the right uterine artery until reduced flow was observed, followed by gel foam injection [Figure 8]. The same steps were repeated for the left uterine artery. The 45-minute procedure achieved complete occlusion, and the bleeding stopped. At 4 weeks, she developed amenorrhoea, and at 6 months follow-up, USG showed a full recovery and no residual AVM with normal periods.

Figure 7: Digital subtraction angiography Rt uterine artery AVM

Figure 8: Fluoroscopic image showing microcatheter placement and embolisation of the Rt uterine artery

Case 5: A 34-year-old woman (P2L2A1) with NVD and LCB 3 years prior presented with HMB for 4 months, and the serum β-hCG was 64.04mIU/ml. USG with Doppler demonstrated a uterine AVM with a feeding vessel, and MRI of the pelvis confirmed a posterior myometrial AVM (3.4 × 2.2 cm) with a 10 cc hematometra, supplied by both uterine arteries. Bilateral UAE was performed via right CFA retrograde access under LA using a 6Fr sheath, 6Fr pigtail catheter, and 5Fr vert catheter over a 0.035 wire. Selective cannulation of the uterine arteries was achieved using a Progreat microcatheter. PVA particles (500–750 μm) mixed with lipiodol were injected into the right uterine artery until reduced flow was noted, followed by gel foam injection

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[Figure 10]. The same procedure was performed on the left uterine artery, and the entire procedure took 45 min. An angiogram confirmed the occlusion, and the bleeding stopped. At 4 weeks, she developed amenorrhoea, and at 6 months, USG showed no residual AVM with normal cycles and complete recovery.

Figure 9: Fluoroscopic angiography with a catheter.

Table 1: Summary of all cases

Case	Age	Antecedent Event	Hb (g/dL)	PRBC Units	Imaging	Size (cm)	Embolic Agent	Outcome	Follow-up
1	24	Post LSCS and LCB before 10 months	8.8	1	USG + CT	3.6×2.5	PVA+Gel foam	Bleeding stopped	16 months, normal
2	33	Pre NVD + MTP + TAT before 1.5 months	7.9	2	USG + MRI + CT	6.2×3.9	PVA+Gel foam	Bleeding stopped	10 months, normal
3	28	Pre-NVD and abortion before 2 months	7.5	2	USG + CT	2.5×2	PVA+Gel foam	Bleeding stopped	6 months, normal
4	30	Pre NVD + LCB before 2.5 years	9.5	NA	USG + MRI	2.6×3.2	PVA+Gel foam	Bleeding stopped	6 months, normal
5	34	Pre NVD + LCB before 3 years	9.8	NA	USG + MRI	3.4×2.2	PVA+Gel foam	Bleeding stopped	6 months, normal

DISCUSSION

UAVMs are uncommon but clinically important causes of severe uterine bleeding in women of reproductive age. Acquired lesions often follow uterine trauma, pregnancy-related procedures, or instrumentation, and they may present with sudden HMB that requires imaging and intervention.[7] Studies suggest that the most severe UAVMs are acquired rather than congenital, and prior uterine procedures are the main causative reasons.2 In our series, all five patients presented with heavy menstrual bleeding and had recent obstetric or gynaecologic antecedents (LSCS, MTP, NVD, or recent abortion). Transvaginal or transabdominal grayscale USG with colour and spectral Doppler is the first-line investigation because it is rapid, commonly available, and useful when a hypervascular "mosaic" pattern and low-resistance, high-velocity flow are seen.[2] CT angiography and MRI/MR angiography provide additional anatomic detail (extent of myometrial involvement, feeding arteries, and draining veins) and are useful when USG is inconclusive or when procedural planning is required. DSA is the diagnostic gold standard and simultaneously allows therapeutic embolisation.[8] A similar diagnostic pathway was used in our patients, which led to an early and accurate diagnosis.

Mildly elevated β-hCG levels can also indicate conditions such as retained products of conception (RPOC) or gestational trophoblastic disease (GTD). However, in RPOC, USG usually reveals an echogenic endometrial mass with vascularity, while GTD is characterised by continuously rising β-hCG.[9] In contrast, patients in our study with β-hCG values of 55–64 mIU/mL had imaging suggestive of AVM, and had a declining β-hCG on follow-up, and their angiography confirmed pure vascular shunting without retained tissue, thereby excluding RPOC and GTD. UAE is an established fertility-sparing option for symptomatic UAVMs. A range of embolic agents, including PVA particles, gelatin sponges (Gelfoam), tris-acryl microspheres, and liquid adhesives (e.g, N-butyl cyanoacrylate/Onyx), have been successfully employed.[10] The choice of agent is influenced by lesion anatomy, operator preference, availability, and the urgency of haemorrhage control.[11] In our case series, we used 500–750 μm PVA particles with lipiodol and adjunctive gel foam for temporary occlusion of smaller channels; this technique achieved immediate haemostasis in all five patients.

Complications of UAE include post-embolisation syndrome, non-target embolisation, access-site events, and rare vascular injury. A study suggested that there are no major effects on ovarian function after selective uterine embolisation when performed carefully, particularly in women under 40.[12] All of our patients were counselled about these possible risks, but none of them experienced any major complications, non-target embolisation, or clinical

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signs of ovarian failure during the follow-up period. Structured clinical and imaging follow-up after embolisation enables the early detection of residual or recurrent vascular shunting and assessment of menstrual and reproductive recovery.[2] Therefore, we followed patients at 4 weeks and at intervals of up to 16 months, and all showed radiological resolution and restoration of menses. The global success rate of embolisation treatment was 88.4%, and the rate of adverse outcomes was 1.8%.[13] However, early diagnosis, precise management, and proper follow-up in all our patients led to 100% success with no complications in all five cases.

CONCLUSION

Uterine artery embolisation is a safe, minimally invasive, and fertility-preserving treatment for UAVM. USG with colour Doppler is the initial diagnostic tool, supported by CT or MR angiography, with DSA as the gold standard. UAE provides effective haemorrhage control, achieves radiological resolution, restores normal menstruation, and has a low risk of complications, making it a reliable alternative to hysterectomy in women of reproductive age. However, these findings are based on a limited number of cases, and larger studies are required to validate long-term outcomes, particularly with respect to fertility and reproductive potential.

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