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Case Report

Uterine arteriovenous malformation mimicking retained products Of conception – treated with embolization ☆

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ABSTRACT

Uterine arteriovenous malformations are uncommon but pose the risk of potentially life-threatening hemorrhage. A 29-year-old pregnant female presented with vaginal spotting, after which ultrasound diagnosis of missed miscarriage was made and medical management undertaken. Vaginal spotting continued post-treatment which led to repeat pelvic ultrasound and subsequent magnetic resonance imaging which confirmed a uterine arteriovenous malformation masquerading as retained products of conception. Uterine instrumentation with dilatation and curettage could have been potentially devastating. The patient was successfully treated with uterine artery embolization.

Introduction

Uterine arteriovenous malformations are uncommon but potentially life-threatening lesions characterized by abnormal arteriovenous connections within the uterus, without an intervening capillary network. Uterine arteriovenous malformations can be classified as either congenital or acquired, with the latter being more common and secondary to previous pelvic surgery, infection, gynecologic malignancies, gestational trophoblastic disease, or exposure to diethylstilboestrol. Diagnosis is made using color Doppler ultrasound and magnetic resonance Imaging with hysterectomy and embolization remaining the primary treatment options.

Case report

We present the case of a healthy 29-year-old female gravida 4, para 2+0 (G4P2+0) who presented with a positive pregnancy test and vaginal spotting. There was no history of previous caesarean section, dilatation and curettage or pelvic surgery. An ultrasound scan was performed which revealed a missed

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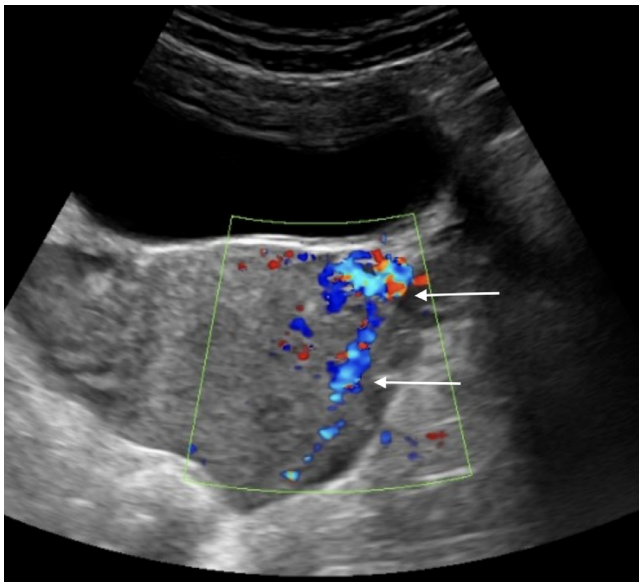

Fig. 1 – Transabdominal pelvic ultrasound in the transverse plane with colour flow Doppler mapping, demonstrating serpiginous vessels in the myometrium within the uterine corpus.

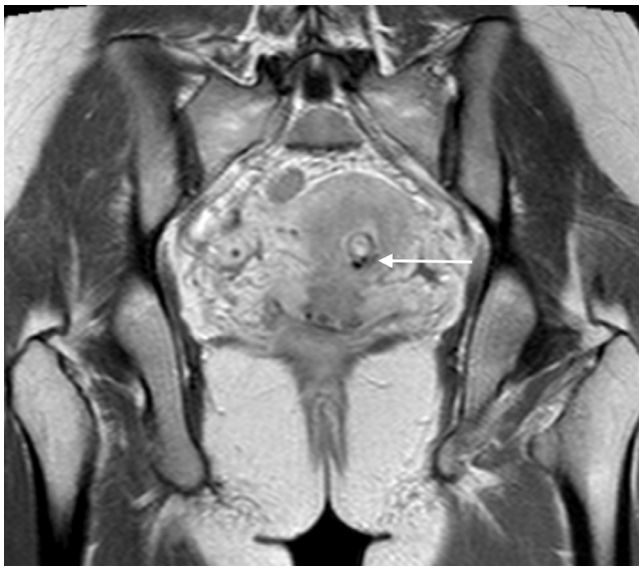

Fig. 2 – Coronal MRI pelvis T1W postintravenous gadolinium administration showing serpiginous low signal flow voids in the myometrium and endometrial cavity, consistent with a uterine AVM.

miscarriage. Medical management of the missed miscarriage was undertaken with misoprostol. The patient continued to spot post-treatment which prompted further investigation. A second ultrasound scan revealed a uterine arteriovenous malformation (AVM) which was confirmed with Magnetic Resonance Imaging (Figs. 1–4).

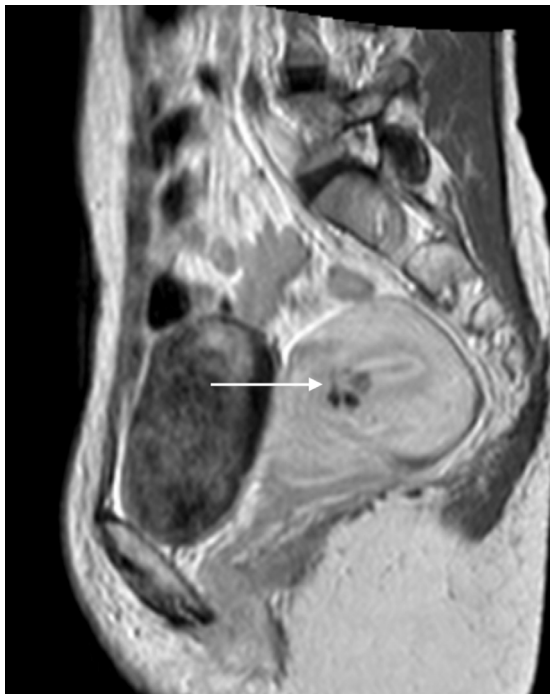

Fig. 3 – MRI pelvis T1W postintravenous gadolinium in sagittal plane showing endometrial and myometrial flow voids consistent with a uterine arteriovenous malformation (AVM).

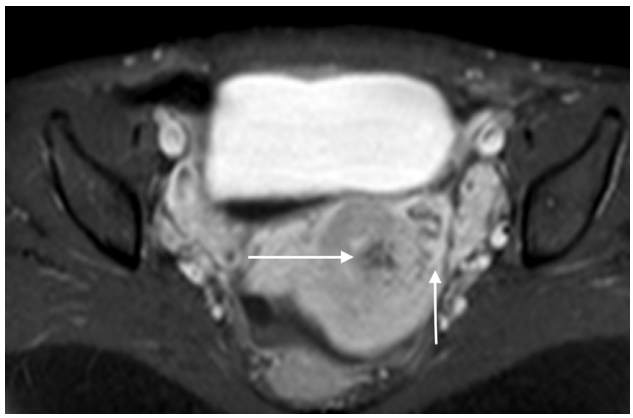

Fig. 4 – MRI pelvis T1W postintravenous gadolinium in the transverse plane revealing flow voids within the uterine parenchyma (horizontal arrow) and in the left adnexa (vertical arrow).

Uterine AVM imaging

Treatment options were discussed with the patient and she elected to have a uterine sparing embolization procedure rather than hysterectomy. She was successfully treated by uterine artery embolization and interval scan 2 months later demonstrated complete resolution of the AVM. She has remained asymptomatic to date.

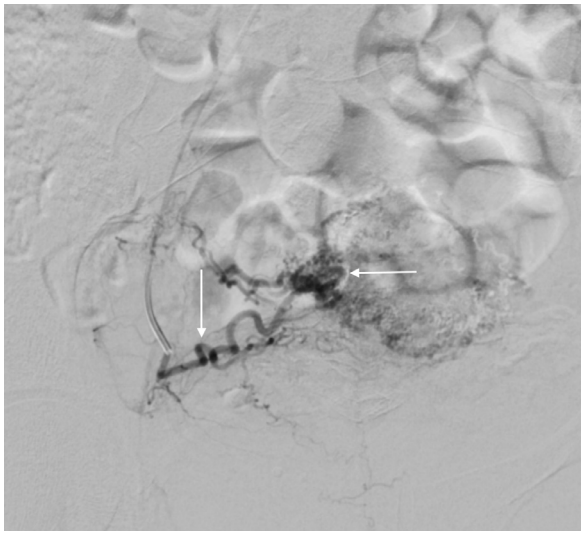

Fig. 5 – Selective angiogram of the right uterine artery (vertical arrow) demonstrating filling of the uterine AVM (horizontal arrow).

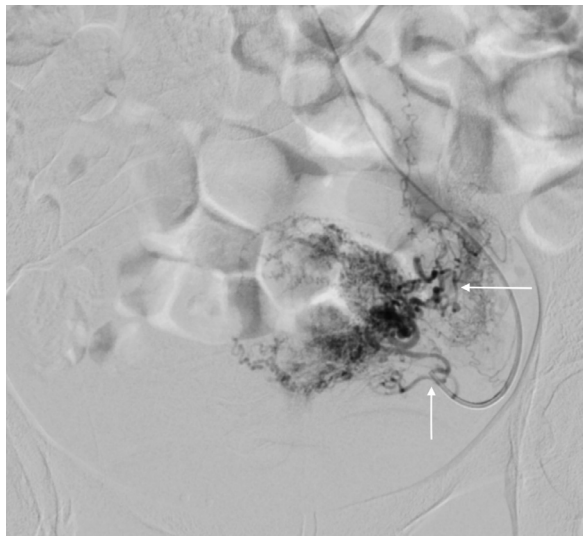

Fig. 6 – Selective left uterine artery angiogram (vertical arrow) demonstrating filling of the uterine AVM (horizontal arrow).

Embolization procedure

Informed consent was sought and obtained. Under aseptic technique an ultrasound-guided puncture of the left brachial artery was performed using a 21-gauge needle. Vascular access was obtained and a 4-French vascular sheath placed. After obtaining a non-selective pelvic angiogram, the right uterine artery was selectively catheterised using a 4-French sheath, a 100 cm 4-French Cobra Glidecatheter and a 0.035-inch Terumo Glidewire. A right uterine artery angiogram was then performed and embolization of the right uterine artery done with polyvinyl alcohol particles, measuring 500-700 micrometres, until stasis of flow was achieved. This was followed by absorbable gelatin powder slurry. The left uterine artery was then selectively catheterized, and additional polyvinyl alcohol particles were injected until vessel occlusion was achieved, followed once more by gelatin powder slurry. After bilateral uterine artery embolization, a completion pelvic angiogram was performed to confirm post embolization stasis of flow in the uterine arteries. The left brachial artery access sheath was then removed and manual compression applied to the left antecubital fossa. The patient was kept for overnight monitoring and given intravenous fluids, anti-inflammatory drugs, analgesics and antibiotics. There were no access site complications and the patient was discharged on the following day with oral analgesics and antibiotics (Figs. 5–9).

Follow-up Imaging

Figure 9.

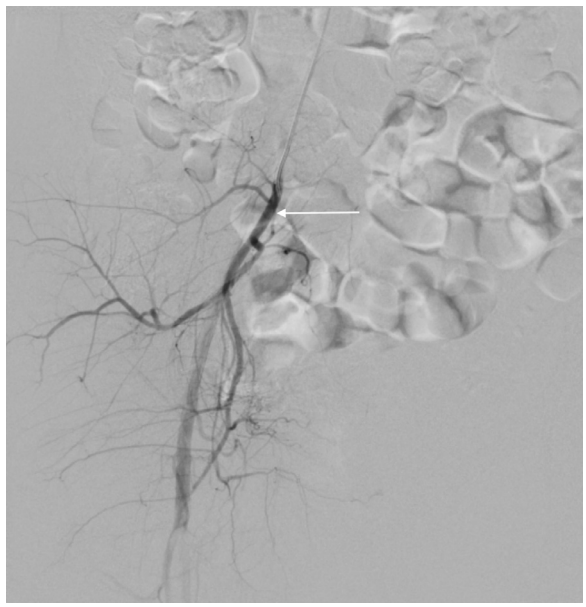

Fig. 7 – Completion digital subtraction angiogram (DSA) of the right internal iliac artery (horizontal arrow) demonstrating no filling of the uterine AVM.

Discussion

An AVM is an abnormal communication between arteries and veins that bypasses the capillary system. These anomalies are potentially fatal as the high-pressure arterial flow can compromise the integrity of the venous walls and result in hemorrhage [1]. AVMs can be found anywhere in the vascular system, including the uterus [2].

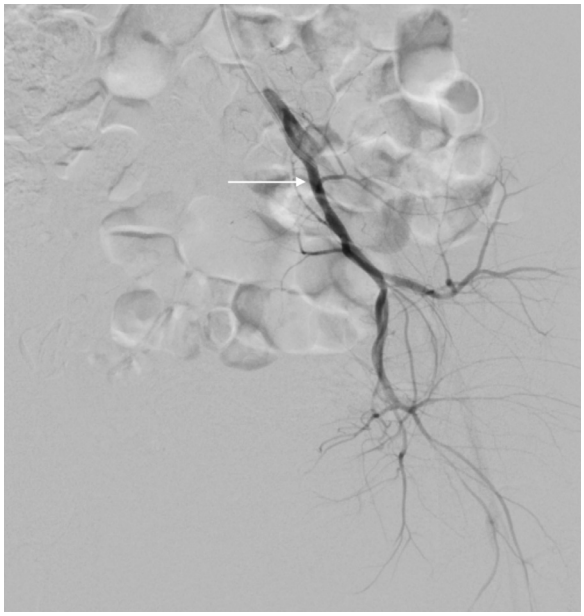

Fig. 8 – Completion digital subtraction angiogram (DSA) of the left internal iliac artery also demonstrating no filling of the uterine AVM.

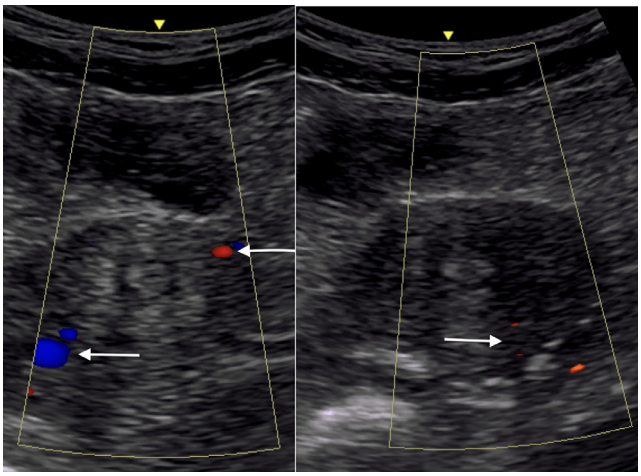

Fig. 9 – Post embolization ultrasound with color flow mapping of uterine body revealing normal myometrial flow (arrows), and absence of flow in the previously noted endometrial and myometrial serpiginous vessels.

Uterine AVMs (UAVMs) may be congenital or acquired. Congenital UAVMs are rare, tend to have multiple vascular connections and invade the surrounding structures. Acquired UAVMs are confined to the myometrium and/or the endometrium and show a direct communication between the intramural branches of the uterine artery and the myometrial veins [3]. Acquired UAVM is more common and often follows a history of curettage procedures, caesarean section, or pelvic surgery. Acquired UAVM is also associated with infection, retained products of conception, gynaecologic malignancies, gestational trophoblastic disease, and exposure to diethylstilboestrol [4].

Acquired UAVMs are usually identified in symptomatic, multiparous women of childbearing age. It is unusual to find an acquired UAVM in nulliparous women without a history of gynecological surgery or uterine instrumentation [5]. The most common presentation of an acquired UAVM is abnormal uterine bleeding.

The association of pregnancy and worsening symptoms suggests a hormonal mechanism. Pregnancy and associated hormonal changes, such as elevated human chorionic gonadotropin, may play a role in the proliferation of otherwise latent AVMs through an unclear mechanism. Thus abnormal uterine bleeding after miscarriage may be due to retained product of conceptions, nonobliteration and subinvolution of the blood vessels of the placental bed, and secondary to UAVM formation. These all share similar radiologic findings which can prove to be a diagnostic challenge [5].

The current primary treatment options are embolization and hysterectomy. In recent times embolization has evolved as the treatment option of choice in women of reproductive age desirous of future pregnancies [6] while hysterectomy is considered in postmenopausal women and emergency situations.

Informed Consent

Written informed consent has been obtained from the patient to use her information in the manuscript.

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