

## Postpartum haemorrhage

SI Mohamed<sup>1</sup>, MBBS, MRad, BJJ Abdullah<sup>1,\*</sup>, MBBS, FRCR, SZ Omar<sup>2</sup>, MBBS, MRad

<sup>1</sup> Department of Biomedical Imaging (Radiology), Faculty of Medicine, University of Malaya, Kuala Lumpur, Malaysia

<sup>2</sup> Department of Obstetric and Gynaecology, Faculty of Medicine, University of Malaya, Kuala Lumpur, Malaysia

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### CASE REPORT

A 30-year-old Malay female presented with massive secondary postpartum haemorrhage three weeks after a complicated vaginal delivery. There was difficulty in the delivery of the placenta at delivery and it was only delivered completely after 30 minutes by controlled cord traction. She was also treated with 40 units oxytocin infusion to prevent postpartum haemorrhage. Blood loss was minimal and estimated at 250 ml. She also had a previous history of a pregnancy being complicated by retained placenta where manual removal of the placenta under general anaesthesia was required. Her menarche was at 14 years and before that pregnancy she had a normal menstrual pattern.

Physical examination revealed a mildly pale young woman with blood pressure of 100/60 mmHg and pulse rate of 90 beats per minute. Gynaecological examination revealed a slightly enlarged, retroverted, mobile and non-pulsatile uterus. There were large blood clots in the vagina and nominal active vaginal bleeding from a normal-appearing cervical os. No cervical or vaginal lacerations were observed. Laboratory evaluation revealed mild anaemia with haemoglobin count of 10.0 g/L and a normal platelet count of 178,000. Coagulation profile and serum beta-HCG level were within normal limits.

In view of bulky uterus, transvaginal ultrasonography pelvis was performed to rule out intrauterine pathology (Figure 1).



**Figure 1** Transvaginal colour Doppler ultrasound (printed in grey scale) of the uterus in the sagittal plane

### QUESTIONS

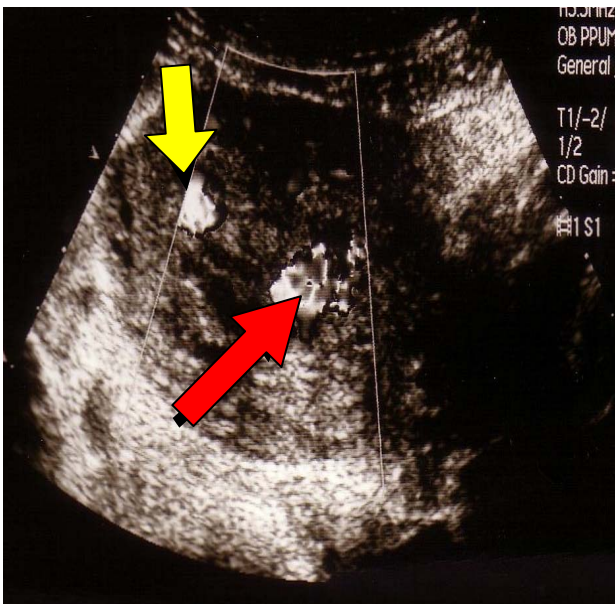
1. What does the ultrasound image show?
2. What is the underlying pathological process?
3. What are the known causes of this abnormality?
4. What are the other clinical presentations?
5. What other imaging modalities can be used for diagnosis?
6. What treatment options are available?

\* Corresponding author. Present address: Department of Biomedical Imaging (Radiology), University of Malaya, 50603 Kuala Lumpur, Malaysia. Tel.: +603-79502069; Fax.: +603-79581973; E-mail: basrij@ummc.edu.my (Basri J.J. Abdullah).

# Arteriovenous malformation of the uterus: a rare cause of secondary postpartum haemorrhage

## Answer 1

Ultrasonography reveals mixed echogenicities within the uterine cavity with multiple anechoic spaces within the myometrium at the uterine fundus and body. These cystic spaces demonstrated colour signals and high velocity flow with a low resistive index at colour Doppler ultrasonography consistent with a diagnosis of uterine arteriovenous malformation (AVM) (Figure 1).



**Figure 1** Transvaginal colour Doppler ultrasound (printed in grey scale) of the uterus in the sagittal plane demonstrates colour signals with high velocity blood flow within the myometrium at the uterine fundus (yellow arrow) and body (red arrow).

## Answer 2

AVMs of the uterus are rare and were first described by Dubreuil and Loubat in 1926 [1]. An AVM consists of a proliferation of arterial and venous channels with fistula formation and an admixture of small, capillary-like channels. The size of these vessels varies considerably, which probably accounts for the variety of descriptive terms found in the literature [1,2]. The true incidence of uterine AVMs is unknown since to date only case reports or small case series exist, making it impossible to provide a true incidence [3]. It is known that AVMs are more common in women and have a predilection for the pelvic blood vessels. Since then, several terms have been used to refer to these lesions, including cavernous haemangioma, cirroid aneurysm, racemose aneurysm, arteriovenous aneurysm, pulsatile angioma, and arteriovenous fistula.

## Answer 3

Uterine AVMs may be congenital or acquired. In the congenital variety, there is failure of differentiation into artery and vein with the ultimate presence of multiple anastomoses between the two as well as involving the surrounding muscles, skin or viscera [4]. Even though it is generally accepted that acquired malformation may be due to previous uterine trauma (prior pelvic operation, curettages), intrauterine contraceptive devices, pathologic pregnancy-related events, and the treatment of endometrial carcinoma and gestational trophoblastic disease [5] resulting in an abnormal communication between adjacent artery and vein, it has been suggested that these procedures may only cause a clinically silent congenital malformation to manifest. In addition, the effect of hormones on AVMs further confuses the issue. Nevertheless in view of the patient's history of previous pregnancy complicated by retained placenta, this may indicate an acquired aetiology. In addition, the absence of symptoms prior to this, the presence of a single communication between artery and vein, plus the absence of investment of surrounding tissues makes an acquired aetiology highly likely.

Doppler and magnetic resonance (MR) imaging features of uterine AVMs may overlap with other causes of arteriovenous shunting, including abnormal placentation and gestational trophoblastic disease (GTD). These can be differentiated with serum beta human chorionic gonadotropin (HCG) tests results (negative with AVM, positive with GTD) [6].

## Answer 4

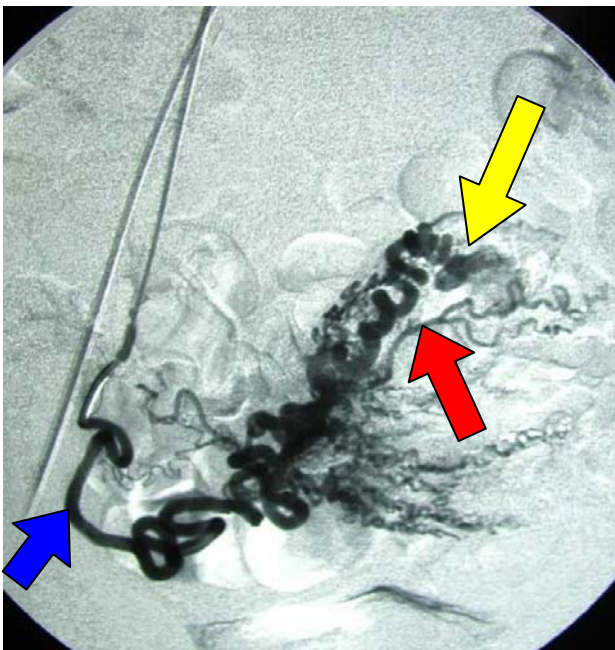
Even though the clinical presentation of uterine AVMs is highly variable, the most common presenting complaint is excessive vaginal haemorrhage or menorrhagia, which requires blood transfusions in 30% of reported cases [6,7]. Other uncommon presentations include postpartum haemorrhage like in this patient, postmenopausal bleeding and congestive heart failure secondary to a vascular steal syndrome [2,3]. Additionally, a number of cases of asymptomatic uterine AVMs have been found incidentally at the time of hysterectomy [1]. Age and prior pregnancy have little value in making a diagnosis. The presence of a palpable arterial pulsation on bimanual examination or the presence of a pulsating mass on hysteroscopy should increase the clinician's suspicion for AVMs.

## Answer 5

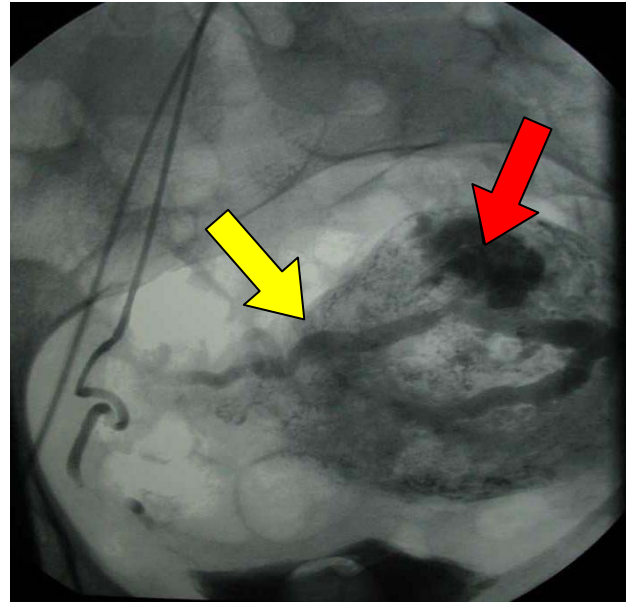
Uterine AVMs can be diagnosed by using ultrasonography, contrast-enhanced computed

tomography, MR imaging and angiography [7]. The greyscale ultrasonography (US) may demonstrate prominent parametrial vessels, a normal appearing endometrium and multiple distinct anechoic spaces in the myometrium without mass effect. On colour Doppler US, these cystic spaces generate colour signals in a mosaic pattern representing turbulent flow. These appearances were seen in this patient. Spectral analysis of the arterial vessels within the lesion shows high-velocity flow with a low resistive index (approximately 0.51-0.65). Spectral analysis of venous flow demonstrates a similar pattern [2,6]. However, the ability of Doppler US to depict the precise extent of the lesion within the pelvis may be limited even with transvesical or endovaginal ultrasound. Doppler US however is the initial method of choice and the preferred method for following up patients after treatment [6].

MR imaging may be performed to confirm a diagnosis and delineate the extent of AVM [6]. MR imaging may show a bulky uterus, a focal uterine mass, disruption of the junctional zones, serpiginous flow-related signal voids, and prominent parametrial vessels [6]. The definitive diagnosis has been traditionally made using angiography and it is well documented in the literature [1,3-5,7]. Pelvic angiography in this patient revealed serpiginous, dilated and tortuous vessels in the uterus, supplied primarily by the right uterine artery with a small contribution from the left uterine artery (Figure 2). Early large draining veins and nidus of the AVM were noted at the end of the arterial phase (Figure 3).



**Figure 2** Selective catheterization of right uterine artery (blue arrow) demonstrates dilated, tortuous and serpiginous vessels supplying the fundus (yellow arrow) and body of the uterus (red arrow).



**Figure 3** Large early draining veins (yellow arrow) and nidus (red arrow) of the uterine arteriovenous malformation are noted at the end of the arterial phase.

#### Answer 6

The treatment of uterine AVM depends on the severity of vaginal bleeding, the patient's age and her future fertility desires. If the patient is asymptomatic, no treatment is required [7]. Acute treatment consists of haemodynamic stabilization and management of active bleeding. Ultimate treatment depends on the patient's reproductive desires [2,3]. In the past treatment has been confined to hysterectomy, but this carries significant morbidity and in women of reproductive years is associated with considerable psychological distress. Recent reports have described successful treatment by embolisation, with subsequent pregnancies indicating preservation of uterine function [3, 8] though this is still controversial. Embolisation performed simultaneously with angiography can provide both diagnosis and treatment during the same procedure.

In the case presented, hysterectomy was discussed as definitive therapy but the patient expressed a desire to retain the uterus if at all possible to attempt future pregnancy. The patient was transfused with three units of whole blood and after haemodynamic stabilisation; percutaneous transfemoral pelvic angiography was performed with a view for embolisation. Both uterine arteries were selectively embolised using a 5F Roberts Uterine Curve catheter (Cook Incorporated, Bloomington, USA) with one vial 300-500  $\mu$ m polyvinyl alcohol particles (Cook Incorporated, Bloomington, USA) (Figure 4). After the procedure, no arterial flow could be demonstrated, and the post procedure course in ward was uneventful. During a follow-up period of six months after the embolisation, the patient has had normal menses without excessive bleeding and no pelvic complaints.

Embolic agents, which can be used for uterine vessels, include polyvinyl alcohol (PVA) particles, absorbable gelatine sponge (Gelfoam; Pharmacia and

Upjohn, Kalamazoo, MI), metallic coils, and glue (N-butyl cyanoacrylate, Histoacryl; B. Braun, Melsungen, Germany). The basis of embolic agent selection depends largely on operator preference and expertise; in those with expertise with the use of glue as an embolic agent for neural AVMs prefers it to other agents. Conversely, PVA particles and absorbable gelatine sponge are preferred by the others and is indicated in cases in those patients where selective embolisation of nidus feeding vessels is not technically possible. Micro-catheters are used when standard angiographic catheters cannot be advanced into the distal uterine artery or branches of the uterine artery as a result of vessel size, tortuosity, and/or spasm.



**Figure 4** The large uterine arteriovenous malformation is successfully embolised using polyvinyl alcohol particles (PVA).

## CONCLUSION

Uterine AVM should be included in the differential diagnosis in patients with excessive postpartum haemorrhage. Colour and spectral flow Doppler US can aid diagnosis and clinical management. Conservative treatment by uterine artery embolisation should also be considered in the treatment of these lesions as it appears safe and effective [8].

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