Uterine AVM
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Citation

Abstract
A uterine arteriovenous malformation (AVM) is a rare cause of uterine bleeding. The reason has been variably described as cirsoid aneurysm, arteriovenous aneurysm, pulsating angioma or cavernous angioma. Earlier the diagnosis was usually made by angiography laparotomy or pathology. With the advent of newer techniques such as Colour Doppler sonography, Computed tomography Magnetic resonance imaging (MRI) the detection of this entity has become easier and smell AVMS can also be detected [1]. Clinical findings in such cases are unreliable, uterine curettage is not therapeutic and even aggravates it [2]. A high index of clinical suspicion is hence required to diagnose this condition [3].

CASE
A 24 year old with no live issue and a history of two previous abortion who had undergone dilatation and curettage 6 months before presented with complaints of sudden episodic profuse vaginal bleeding. The haemorrhage to for the last 3 days was life threatening and required multiple blood transfusions. Per vaginal examination revealed a mildly bulky uterus with bilateral free fornices. Pelvic gray scale ultrasound showed multiple tortuous tubular and serpiginous anechoic spaces throughout the myometrium most marked in the uterine fundus (Fig. 1).

Figure 1
Figure 1: Gray scale pelvic ultrasound showing multiple anechoic spaces throughout the uterine myometrium, most prominent in the fundus.

Colour Doppler US scan showed intense flow with colour aliasing and apparent flow reversals (Figure 2).

Figure 2
Figure 2: Colour Doppler US scan showing intense colour fill within the anechoic spaces. There is evidence of colour aliasing and apparent flow reversals.

Prominent parametrial vessels were also seen (Figure 3).
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Figure 3
Figure 3: Colour Doppler US showing prominent parametrial vessels.

Spectral Doppler revealed pulsatile high velocity venous waveforms with differentiation between arterial and venous waveform difficult as signals are perceived simultaneously above and below the baseline because of proximity of the vessels (Figure 4).

Figure 4
Figure 4: Spectral colour Doppler showing, pulsatile high velocity venous waveforms.

Figure 5
Figure 5: TVS image better depicting the anechoic spaces within the myometrium.

TVS images better depicted the anechoic spaces within the myometrium and the adnexal region (Fig 5) with intense colour fill in on TVS colour Doppler (Fig 6).

Figure 6
Figure 6: Colour TVS image depicting the intense colour fill in the anechoic spaces.

TVS Images depicted colour in the anechoic spaces and colour within the adnexal region (Figure 7,8)
Figure 7
Figure 7: TVS image depicting the anechoic spaces within the adnexal region.

Figure 8
Figure 8: Colour TVS image depicting the intense colour fill in in the adnexal region

TVS spectral Doppler showed low pulsatile arterial waveform.(Fig 9)

Figure 9
Figure 9: Spectral Doppler TVS image showing low pulsatile arterial waveform.

Based on the ultrasound findings a diagnosis of uterine arteriovenous malformation was considered. Choriocarcinoma/gestational trophoblastic disease was considered in the differential diagnosis.

The patient was taken for a MR examination Axial T₁ spin echo MR image showed a bulky uterine fundus with multiple tortuous flow related signal voids. The flow voids were also seen involving the parametrial and adnexal regions (Figure 10).

Figure 10
Figure 10: Axial TW spin echo MR image showing a bulky uterine fundus with multiple tortuous flow related signal voids within the myometrium. The flow voids were also seen involving the parametrium and the adnexal regions.
Sagittal T2W MR image revealed multiple flow voids in the entire myometrium with disruption of the junctional zone. The endometrium was normal (Figure 11).

**Figure 11**
Figure 11: Sagittal TW MR image showing multiple flow voids in the myometrium with disruption of the junctional anatomy. The endometrium is normal with no mass seen. The cervix is also normal.

Postcontrast MR images reveals intense opacification of the channels within the uterine myometrium the parametrium and the pelvic cavity (Figure 12,13).

**Figure 12**
Figure 12,13: Postcontrast axial MR images showing intense enhancement of the vessels in the myometrium the parametrium and the pelvis.

MR angiogram revealed feeder arteries from the bilateral internal iliac arteries and early visualization of the pelvic veins with opacification of the right common iliac vein and then the IVC (Fig.14,15).

Thus the MRI and MRA findings suggested a large arteriovenous malformation involving the uterus with feeder arteries and draining veins.
The patient was taken up for uterine artery embolization and is under follow up.

**Figure 14**
Figure 14,15: MR angiogram image showing the arteriovenous malformation with feeders from the internal iliac artery and early opacification of the right common iliac vein and the IVC.

**DISCUSSION**
Arteriovenous malformations are composed of a tangle of vessels of different sizes with the histologic characteristics of both arteries and veins but without evidence of an intervening capillary network [4].

Uterine AVMs can be congenital or acquired and have been reported in women aged 18-72 years. Congenital uterine AVMs arise from anomalous differentiation in the primitive capillary plexus which results in abnormal communication between arteries and veins. Most commonly uterine AVMs are acquired on a traumatic basis and there is a history of spontaneous abortion followed by dilatation and curettage, therapeutic abortion, endometrial carcinoma or gestational trophoblastic disease [1,5]. Other pelvic trauma such as hysterectomy and caesarean section have also been implicated because of the subsequent development of fistulous communication between uterine arteries and veins. Diethylstilbestrol or DES exposure is also attributed as a cause. Patient history is important in distinguishing between congenital and acquired AVMs as their angiographic features are similar.

The most common symptom is menorrhagia or menometrorrhagia which usually requires blood transfusions in 30% of reported cases [6]. Uterine bleeding is thought to occur when vessels of AVM are exposed from sloughing of the endometrium during the menstrual cycle or iatrogenically during dilatation and curettage. A correct diagnosis is imperative as dilatation and curettage may lead to catastrophic results.

Ultrasound in these patients reveals multiple tortuous anechoic spaces in the myometrium without mass effect. Other gray scale features that have been described include myometrial inhomogenity, an intramural mass mimicking a fibroid and a large bulky cervix that mimicks a cervical fibroid or carcinoma [7]. The abnormalities depicted at colour Doppler US are usually more extensive than those depicted at gray scale US. Colour Doppler reveals intense colour fill in with juxtaposed reds and blues owing to the insonation of overlapping vessels of varifying orientation with different flow directions as they cross the scan plane [7]. The spectral Doppler reveals the classic features of arteriovenous shunting i.e. high PSV low pulsatility of arterial waveform, pulsatile high velocity venous waveforms with little variations in systolic – diastolic velocities [7,8]. The arteriovenous shunting in an AVM needs to be differentiated from malignant arteriovenous shunting seen in neoplasia. Malignant AV shunts tend to have low volume high velocity flow in contrast to low resistance high volume flow that characterizes an AVM [9]. Arteries within a malignant lesion usually drain into a confined venous space whereas the AVM drains into a large, low pressure venous
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MR imaging is helpful in delineating the extent of the lesion. As with AVMs elsewhere in the body, the characteristic features with spin echo sequences is the presence of multiple flow related signal voids within the lesion. The flow related signal voids at MR imaging correspond at Doppler interrogation to the tangle of vessels that compose the uterine AVM. Extension of uterine AVM into the pelvis is easily appreciated at MR imaging.

Gestational trophoblastic neoplasms are another group of hypervascular lesions that manifest with serpentine vessels in the myometrium or in association with a prominent uterine venous plexus [10]. Because the imaging appearance of dilated vessels in GTD may resemble that of uterine AVM, diagnosis depends on patient history and increased titres of human chorionic gonadotropin (HCG) in blood serum.

Choice of management is dictated by the site and size of the lesion. Large lesions (involving the subendometrial tissue) usually require surgical intervention while others respond to conservative management. More recently, uterine AVMs have been treated successfully by intra-arterial embolization with particulate matter. Substances for embolization include gel foam, microfibrillar collagen, isobutyl cyanoacrylate and steel coil spring occludes.

Pregnancy following conservative medical management of AVM and even after successful embolization have been reported in literature [11].

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References
