Case study

Title: A Rare Case of Uterine Arteriovenous Malformation after Dilatation and Evacuation

Abstract- Uterine arteriovenous malformation (AVM) is a rare cause of heavy and sometimes life-threatening vaginal bleeding. As name implies, uterine AVMs result from one or more sites of abnormal direct communication between an artery and a vein without an intervening capillary bed resulting in increased pressure and high-velocity flow, as well as marked vascular enlargement, in the venous system. The treatment of choice depends on the symptoms, age, desire for future fertility, localization, and size. This case report highlights our experience with a patient having acquired uterine AVM after dilatation and evacuation which was done in our institute. Transvaginal scan of pelvis showed evidence of heteroechoic echogenic contents within the uterine cavity with few cystic spaces with increased vascularity on color Doppler. CT angiogram confirmed the diagnosis of AVM in endometrium and posterior myometrium with two arterial feeders and draining veins. Embolization of AVM was performed successfully and patient became asymptomatic post procedure and stopped bleeding PV.

Keywords- Uterine AVM, Embolization, Arteriovenous malformation

Introduction

Uterine arteriovenous malformation (AVM) is a rare condition, with fewer than 100 cases reported in the literature. It is a potentially life-threatening condition, as patients may present with profuse bleeding. Colour Doppler ultrasound (US) provides a non invasive method for initially diagnosing this rare condition and confirmation can be made using diagnostic angiography [8]. pical symptom is vaginal bleeding; however, some patients may present with life-threatening massive bleeding [9].

Case Report- A 29 years old female P1L1A1 was presented to our facility with complaint of vaginal bleeding on and off for one month. She had history of spontaneous abortion of 3 months pregnancy followed by dilatation and evacuation one and half month before in our institute. She had one full term normal vaginal delivery 2 years back. There is no other history of prior curettage. On examination she was hemodynamically stable but anemic. Her abdomen was soft. On vaginal examination os was closed and minimal bleeding per vaginum was there. She was hospitalized and 2 units of packed RBC were transfused. On ultrasound pelvis, there was evidence of heteroechoic echogenic contents measuring 1.5*2.3 cm within the uterine cavity with few cystic spaces. The lesion showed increased vascularity on color Doppler with peak systolic velocity 60 cm/s and no evidence of extension into myometrium. Her beta HCG level was 184.7 miu/ml.

As there was possibility of uterine AVM so CT angiogram (CTA) and digital spectral angiogram of pelvis was performed. On CT angiogram, a heterogeneously hypodense lesion measuring 3.2*2 cm was seen involving uterine endometrium and posterior myometrium. Uterus was bulky. On arterial phase, a nidus of tangled vascular channels was seen predominantly involving posterior myometrium with extension into endometrium. There were two arterial feeders, arising from aorta at 1 o'clock at L2-L3 level (left ovarian artery) and another arising from right internal iliac artery (right uterine artery). Early contrast opacification is also seen in some of the draining veins in arterial phase with perisistence in venous phase. These draining veins are seen draining into bilateral internal iliac veins.

Thus, uterine artery embolization was planned after explaining all risks and benefits as patient desired future fertility. Left uterine artery, left ovarian artery and right uterine artery were cannulated separately and embolized by 500-micron polyvinyl alcohol particles by interventional radiologist. Post procedure angiogram did not show any contrast blush in the region of uterus. Patient was asymptomatic post procedure and stopped bleeding PV. Hence, she was discharged. She was followed up in our facility after one week. Her beta HCG level decreased and transabdominal ultrasound did not show any vascular lesion. Then she was followed up weekly by serum beta HCG level which gradually decreased till zero. Patient remained asymptomatic throughout.

Discussion- Uterine Arteriovenous malformation is a potentially life-threatening disorder in which patient present with vaginal bleeding that may be profuse and cause hemodynamic instability. Dubreuil and Loubat reported the first case of uterine AVM in 1926 [1].

There are two types of AVM, congenital and acquired. Abnormal embryologic differentiation of primitive vascular structures is the suspected etiology of congenital AVM. This leads to inadequate capillary bed formation and resultant arteriovenous connections. [2] Acquired AVM forms after a disruption and defective healing of the uterine vasculature. This abnormal connection may form after a surgical procedure such as endometrial curettage or conditions such as endometritis, gestational trophoblastic disease, endometriosis or cesarean section. [2,3]

Pelvic sonography with color doppler is often the initial screening test performed in the setting of suspected uterine AVM. Spectral Doppler demonstrating elevated flow velocities within low-resistance vessels indicates the presence of an arteriovenous communication. Either CT or MRI can be used to better delineate the anatomy of uterine AVM. Digital subtraction angiography (DSA) remains the gold standard for diagnosis of uterine and pelvic AVMs. The defining feature of AVMs on DSA is brisk early filling of numerous enlarged veins emerging from a nidus of abnormal vessels after arterial contrast injection.

The treatment depends on the age, desire for future fertility, localization, and size of the lesion. The mainstay for management of uterine AVM has been hysterectomy or the embolization of uterine arteries. However, the uterine artery embolization (UAE) remains the first choice of treatment in women at reproductive age having expectation of future fertility [4]. Whether this procedure is safe for women desiring future fertility is controversial; however, women who become pregnant after UAE are at risk of malpresentation, caesarean delivery, preterm birth, and postpartum hemorrhage [5]. Other treatment methods mentioned in literature are use of methylergonovine maleate, gonadotropin releasing hormone analogues, and danazol generally for the treatment of patients with mild hemorrhage with uterine AVM. However, there is currently no clear agreement on the treatment of asymptomatic uterine AVMs.

The literature suggests that transcatheter embolization has high rates of success in treating pelvic and uterine AVMs. One series of 42 patients who underwent Gelfoam embolization for postobstetric uterine AVMs demonstrated successful definitive endovascular treatment in 88% of patients, with the remaining 12% requiring hysterectomy; 13 of these patients became pregnant, culminating in an 80% successful delivery rate with eight term deliveries, two spontaneous abortions, and three elective terminations.[6] Another study of 15 patients with uterine AVMs demonstrated an endovascular therapy success rate of 93%, with only one patient requiring hysterectomy. [7]

Conclusion- Uterine and pelvic AVMs are a rare cause of excessive vaginal bleeding; however, asymptomatic or mildly symptomatic uterine AVMs may be more common than previously thought as detection increases with the use of routine imaging. This case report highlights the importance of CT angiogram and DSA for diagnosis of uterine AVM and role of Uterine artery embolization as treatment modality because of higher success rate and minimally invasive in nature. Uterine AVMs refractory to embolization can be cured with hysterectomy. Conservative management may be appropriate for many patients with uterine AVMs, particularly those with asymptomatic and/or lower-flow lesions. Close clinical follow-up with imaging is still recommended.

Declaration:

Written informed consent of patient was taken for case report along with picture publication.

References-

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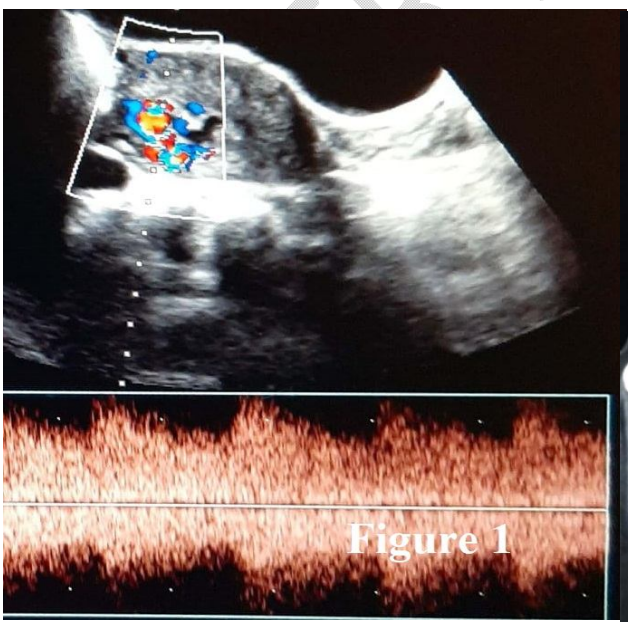


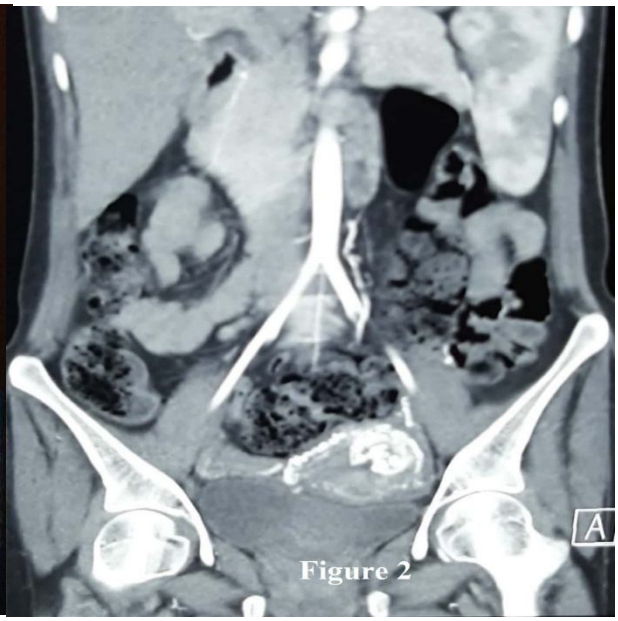
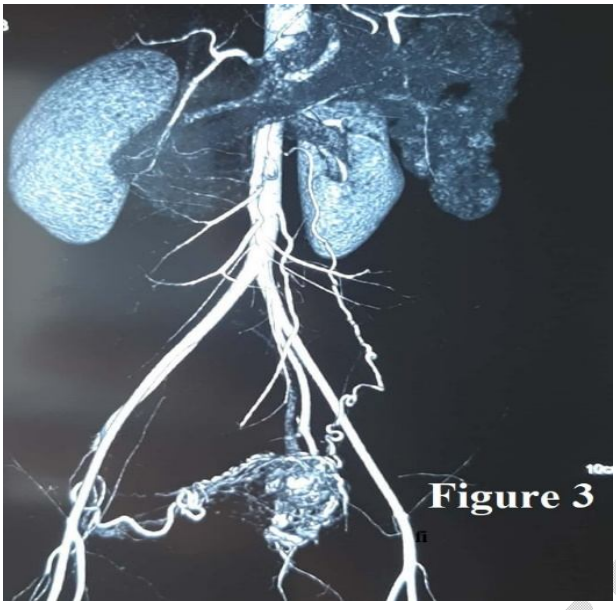


Figure 1. Ultrasound showing bulky uterus with prominent vascularity in posterior wall.

Figure 2. CT angiogram of pelvis showed contrast blush in Uterus in posterior wall.



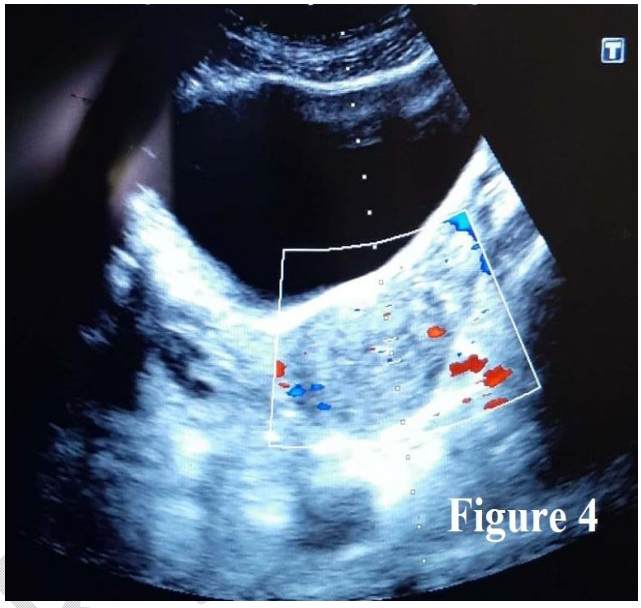


Figure 3. On DSA AVM feeder arising from aorta and right internal iliac artery i.e. left ovarian artery and right uterine artery.

Figure 4. Post procedure follow up transabdominal ultrasound did not show any vascularity within uterus.