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Case Report

Acquired uterine arteriovenous fistula following dilatation and curettage: an uncommon cause of vaginal bleeding

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ABSTRACT

Dysfunctional uterine bleeding is a common presentation of women in the emergency department. We describe the case of a 33-year-old female who presented with intermittent spotting due to an acquired uterine AVF. The patient underwent a transvaginal pelvic ultrasound as well as a CT angiogram. The patient was treated conservatively and elected to undergo uterine artery embolization in an effort to preserve fertility. She successfully delivered a healthy baby boy at 39-week gestation via an emergent caesarian section due to a prolapsed umbilical cord 17 months after undergoing the uterine artery embolization.

Introduction

A 33-year-old female ultrasound technologist presented to the emergency department after experiencing intermittent spotting. She complained of 3/10 pelvic pain accompanied by a constant throbbing sensation. She noted the intermittent spotting to be worse with intercourse and running. The patient had four prior spontaneous miscarriages, two of which required subsequent dilatation and curettage. Her most recent D&C was approximately 1 week prior to her presentation to the emergency department. Due to her symptoms and line of work, she allowed her students to practice by performing a transvaginal ultrasound on her. At that time, an abnormality was noted and the patient went to the ER for further evaluation.

Upon her presentation to the ER, the patient was in no acute distress and her triage vital signs were stable. She denied any heavy vaginal bleeding, nausea, vomiting, chest pain, dizziness, or shortness of breath. Her laboratory studies revealed mild anemia with a hemoglobin level of 11.5 g/dL and a hematocrit level of 33.9%.

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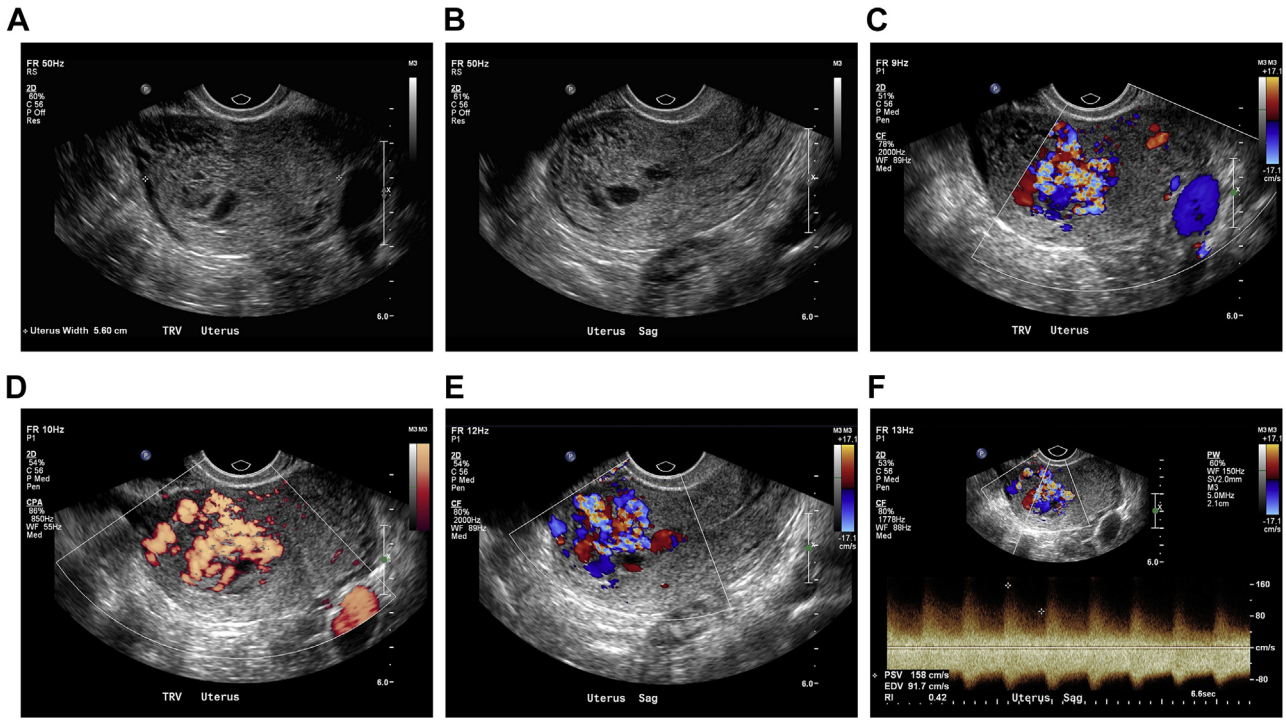

Fig. 1 – Transverse (A) and longitudinal (B) gray scale sonographic images of the uterus demonstrate a focal collection of tubular, anechoic to hypoechoic echoes in the right uterine. No discrete soft tissue mass or mass effect was appreciated. Corresponding transverse color Doppler (C) and power Doppler (D) images as well as a longitudinal color Doppler (E) image demonstrate evidence of increased blood flow within the tubular spaces and adjacent myometrium. Spectral Doppler (F) image exhibits areas of high velocity, low-resistance flow within the area of concern.

Imaging findings

A transvaginal ultrasound was performed (Fig. 1). The gray scale images demonstrated a focal collection of round to tubular anechoic spaces within the myometrium. Color and power Doppler imaging demonstrated blood flow within the anechoic spaces and adjacent myometrium. Spectral Doppler ultrasound images demonstrated findings consistent with high-velocity, low-resistance blood flow.

After a multidisciplinary discussion between emergency room physician, the patient's gynecologist, and the consulting interventional radiologist, the patient was discharged due to the fact that she was hemodynamically stable. The patient was followed closely on an outpatient basis. The intermittent spotting continued as did her complaint of a constant throbbing sensation.

One month following her presentation to the ER, she underwent a CT angiogram of the abdomen and pelvis (Fig. 2) which included a subsequent volume rendered 3D reconstruction (Fig. 3). This study revealed a tangle of vessels in the right uterine fundus with early filling of the right arcuate and iliac veins. After a full discussion of the potential risks, benefits, and alternatives, the patient elected to undergo uterine artery embolization as opposed to a hysterectomy in an effort to preserve her fertility. Diagnostic angiograms of the bilateral internal iliac arteries followed by selective angiograms of the bilateral uterine arteries were performed. A tangle of vessels in the right hemipelvis was again demonstrated with a dominant arterial supply from the right uterine artery (Fig. 4A). Bilateral uterine artery embolization was performed utilizing 500–700 μm Embospheres (Merit Medical Systems, South Jordan, UT). The procedure was technically successful (Fig. 4B). The patient complained of mild pelvic cramping following the procedure which resolved with the use of Tylenol. Her symptoms of intermittent spotting and the constant throbbing sensation abruptly ceased. She successfully delivered a healthy baby boy at 39-week gestation via an emergent caesarian section due to a prolapsed umbilical cord 17 months after undergoing the uterine artery embolization.

Discussion

Uterine arteriovenous malformations (AVMs) are an uncommon, however, potentially fatal cause of vaginal bleeding. The reported incidence is among patients who present with menorrhagia. The true incidence is unknown, but there are fewer than 100 cases reported [1–3]. O'Brien et al. [4] proposed a rough incidence of 4.5%, making it an important diagnosis to be considered in women with unexplained vaginal bleeding. The presentation of our patient was

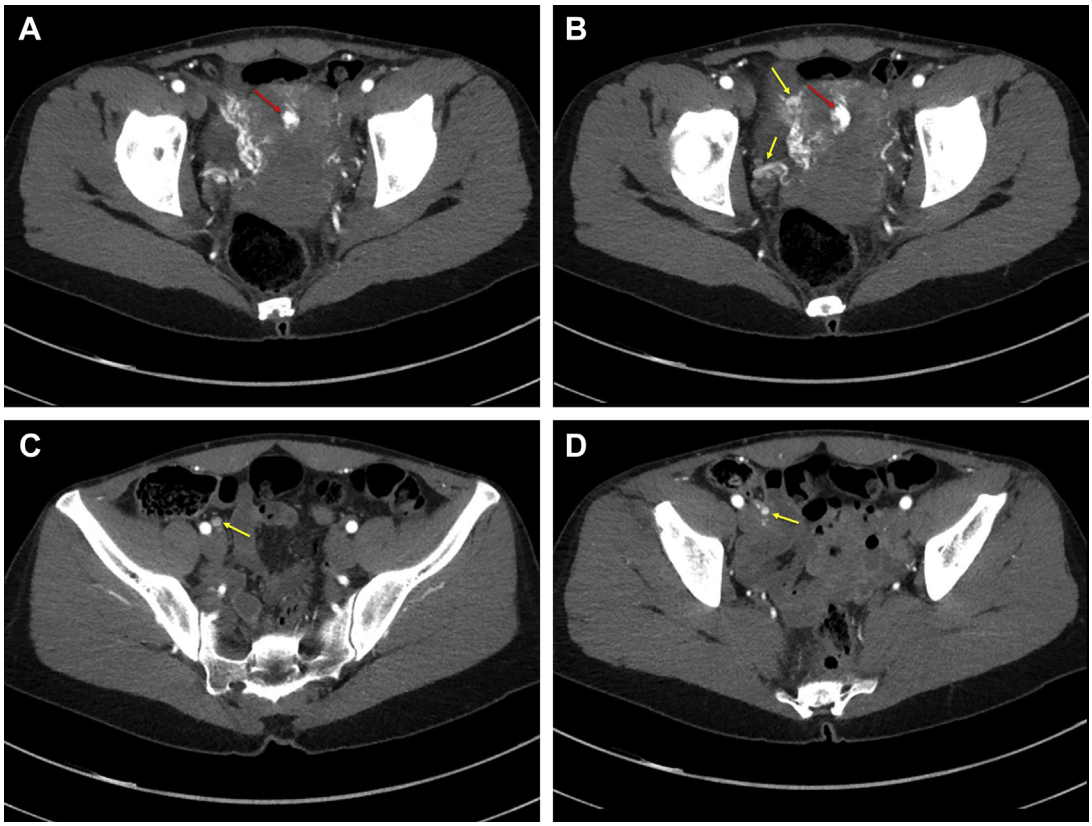

Fig. 2 – Axial images from a CT angiogram of the pelvis demonstrate a serpiginous collection of enlarged vessels in the right uterine fundus (A and B, red arrows) which appear isodense to adjacent arterial structures. There is evidence of early opacification of the myometrial venous plexus on the right (B and C, yellow arrows) and right iliac veins (C and D, yellow arrows) which is not comparatively seen in the left hemipelvis.

atypical in that she presented with a chief complaint of light spotting accompanied by pelvic pain and a persistent throbbing sensation with physical activity as opposed to menorrhagia.

Uterine AVMs can be congenital or acquired [5–7]. Acquired uterine AVMs have been attributed to a variety of causes, including pelvic trauma, surgery (curettage and cesarean section), neoplasm, or inflammation/infection [9]. The majority of reported cases are acquired secondary to dilation and curettage [8]. There is a pathophysiological difference between a congenital as opposed to an acquired uterine AVM. Congenital uterine AVMs are characterized by multiple feeding arteries, a central nidus consisting of vessels with characteristics of both arteries and veins, and multiple large draining veins [5]. In contrast, acquired uterine AVMs consist of one or more arteriovenous fistulas between intramural arterial branches and the myometrial venous plexus [5]. Patient history is therefore of utmost importance in distinguishing between a congenital uterine AVM and what is more accurately described to be an acquired uterine AVF as their radiologic features are similar.

The most common presenting symptom is menorrhagia. Uterine bleeding is thought to occur when vessels of AVMs are exposed from sloughing of the endometrium during the menstrual cycle or iatrogenically during a dilation and curettage. Dilatation and curettage in the setting of an AVM may lead to a catastrophic outcome.

Sonography is the study of choice in these patients and reveals multiple tortuous anechoic spaces in the myometrium without mass effect [4]. Myometrial inhomogeneity, an intramural mass mimicking a fibroid, and a large bulky cervix that mimics a cervical fibroid or carcinoma are other gray scale features. Color Doppler ultrasonography shows the tangle of vessels as serpiginous/tubular anechoic structures within the myometrium with a low-resistance and high-velocity flow pattern [4,6,7,10].

Pelvic angiography remains the gold standard for diagnosis of a uterine AVM [2]. Other imaging modalities of importance include pelvic magnetic resonance imaging and computed tomography angiography [6]. Both of these modalities are noninvasive examinations that allow one to confirm the diagnosis of uterine AVM. Contrast enhanced examinations will demonstrate enhancement of the collection of serpentine vessels which will enhance as intensely as normal vessels in the arterial phase. These findings will be accompanied by evidence of early venous return which is signified by opacification of venous structures on the arterial phase images.

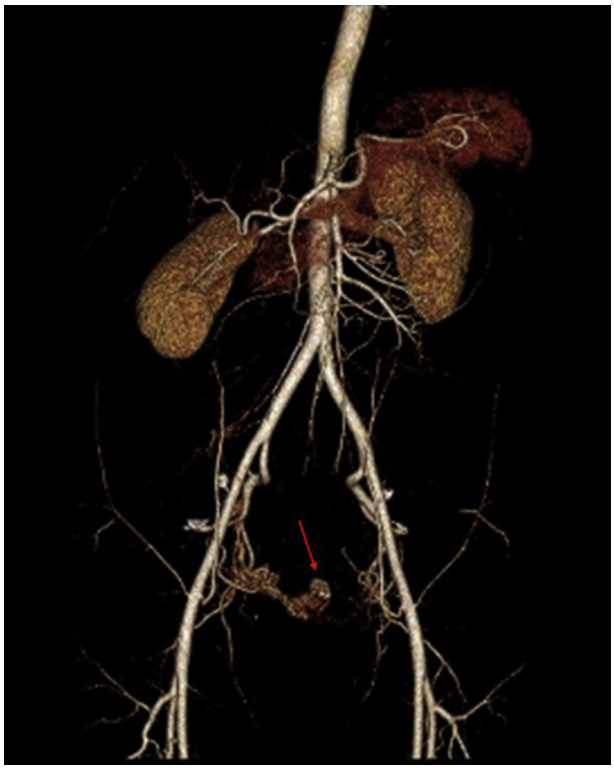

Fig. 3 – Volume rendered 3D reconstruction from a computed tomography (CT) angiogram depicts the uterine arteriovenous fistula (AVF) as a serpiginous collection of enlarged vessels in the right hemipelvis (red arrow).

Management of uterine AVMs depends on many factors including the patient's hemodynamic status, age, and desire for future fertility. Prior to the advent of embolotherapy, hysterectomy was the therapy of choice. Since the first description of a successful embolization treatment for uterine AVM in 1986, embolotherapy has become a well-recognized alternative to surgical intervention for uterine AVMs, with the major advantage of maintaining childbearing capacity [11–14]. Various embolic materials have been utilized in successfully preserving reproductive capability [1,2,4,18]. Despite advances in therapeutic techniques and embolic agents, pregnancy following successful embolization of uterine AVMs remains rare [2–4,7,9]. Decreased vascularization of the placenta has been proposed as being the main cause of adverse pregnancy outcomes following embolotherapy [8]. Superselective embolization of the nidus of the AVM with preservation of additional pelvic arteries and branches allows for collateral circulation to develop and probably accounts for cases of successful pregnancies in these cases [1,7,8].

A retrospective review by Ghai et al. reported 15 patients who underwent pelvic arterial embolization for traumatic uterine AVMs at their institution over a 10-year span. Embolization agents used varied and depended on operator preference and expertise. The technical success rate was 100%, and the clinical success rate was 93%. The authors did not believe the type of embolization agent used influenced the outcome. Four of the 15 patients had a total of five uneventful intrauterine pregnancies carried to term. The authors concluded that percutaneous embolotherapy is a safe and effective treatment for traumatic uterine AVMs while preserving the possibility of future pregnancy [11,12,14,15].

Patients with one episode of bleeding and hemodynamic stability can also be treated with other conservative measures such as oral contraceptive pills [6,16]. Timmerman et al. presented 10 cases that demonstrated uterine AVM features by color Doppler ultrasound; of these, spontaneous resolution of six of the cases occurred following oral contraceptive pill use or expectant management making this a viable option [17].

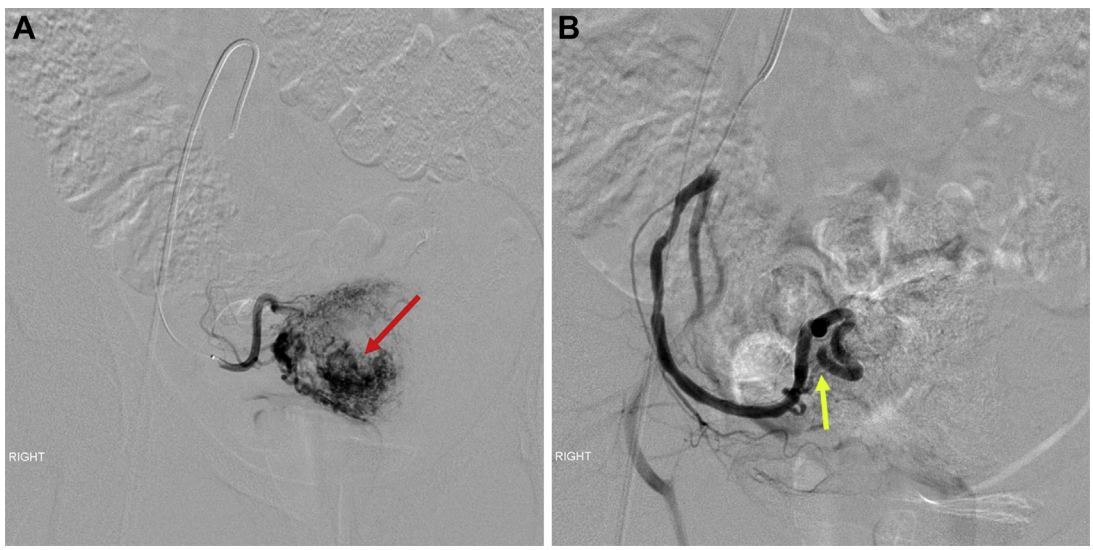

Fig. 4 – Subselective angiogram of the right uterine artery demonstrates a tangle of vessels in the right hemipelvis consistent with an AVF (A, red arrow). Angiogram of the right uterine artery demonstrates absence of the previously demonstrated AVF with a truncated appearance of the distal right uterine artery (B, yellow arrow) consistent with successful embolization.

REFERENCES

[1] Poppe W, Van Assche FA, Wilms G, Favril A, Baert A. Pregnancy after transcatheter embolization of a uterine arteriovenous malformation. Am J Obstet Gynecol 1987;156:1179–80.

[2] Fleming H, Ostor AG, Picket H, Fortune DW. Arteriovenous malformation of the uterus. Obstet Gynecol 1989;73:209–14.

[3] Hoffman MK, Meilstrup JW, Shackelford DP, Kaminski PF. Arteriovenous malformations of the uterus: an uncommon cause of vaginal bleeding. Obstet Gynecol 1997;52:736–40.

[4] O'brien P, Neyastani A, Buckley AR, Chang SD, Legiehn GM. Uterine arteriovenous malformations: from diagnosis to treatment. J Ultrasound Med 2006;25(11):1387–92.

[5] Cura M, Martinez N, Cura A, Dalsaso TJ, Elmerhi F. Arteriovenous malformations of the uterus. Acta Radiol 2009;50(7):823–9.

[6] Grivell RM, Reid KM, Mellor A. Uterine arteriovenous malformations: a review of the current literature. Obstet Gynecol Surv 2005;60(11):761–7.

[7] Polat P, Suma S, Kantarcý M, Alper F, Levent A. Color Doppler US in the evaluation of uterine vascular abnormalitiesc. Radiographics 2002;22(1):47–53.

[8] Chia YN, Yap CS, Tan BS. Pregnancy following embolisation of uterine arteriovenous aalformation—a case report. Ann Acad Med Singap 2003;32:658–60.

[9] Peitsidis P, Manolakos E, Tsekoura V, Kreienberg R, Schwentner L. Uterine arteriovenous malformations induced after diagnostic curettage: a systematic review. Arch Gynecol Obstet 2011;284:1137–51.

[10] Huang MW, Muradali D, Thurston WA, Burns FN, Wilson SR. Uterine arteriovenous malformation: gray scale and Doppler US features with MR imaging correlation. Radiology 1998;206:115–23.

[11] Bonduki CE, Feldner PC, Silva JD, Castro RA, Sartori MG, Girão MJ. Pregnancy after uterine arterial embolization. Clinics (Sao Paulo) 2011;66(5):807–10.

[12] Ghai S, Rajan DK, Asch MR, Muradali D, Simons ME, TerBrugge KG. Efficacy of embolization in traumatic uterine vascular malformations. J Vasc Interv Radiol 2003;14:1401–8.

[13] Ghosh TK. Arteriovenous malformation of the uterus and pelvis. Obstet Gynecol 1986;68(3 Suppl):40S–3S.

[14] Kwon JH, Kim GS. Obstetric iatrogenic arterial injuries of the uterus: diagnosis with US and treatment with transcatheter arterial embolization. Radiographics 2002;22(1):35–46.

[15] Wilms GE, Favril A, Baert AL, Poppe W, Van Assche FA. Transcatheter embolization of uterine arteriovenous malformations. Cardiovasc Intervent Radiol 1986;9(2):61–4.

[16] Selby S, Haughney M. Uterine arteriovenous malformation with sudden heavy vaginal hemorrhage. West J Emerg Med 2013;14(5):411–4.

[17] Timmerman D, Van den Bosch T, Peeraer K, Debrouwere E, Van Schoubroeck D, Stockx L, et al. Vascular malformations in the uterus: ultrasonographic diagnosis and conservative management. Eur J Obstet Gynecol Reprod Biol 2000;92:171–8.

[18] Markoff G, Quagluarello J, Rosen RJ, Beckman EM. Uterine arteriovenous malformation successfully embolised with a liquid polymer, isobutyl 2-cyanocrylate. Am J Obstet Gynecol 1986;55:659–60.