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Case Report

Transcatheter Embolization of Uterine Arteriovenous Malformation: Report of 2 Cases and Review of Literature

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ABSTRACT Arteriovenous malformation (AVM) of the uterus is a rare cause of menorrhagia and may at times lead to life-threatening hemorrhage. The clinical findings may not always be reliable in the diagnosis of uterine AVM, and a high index of suspicion is important because, unlike many other causes of menorrhagia, curettage may paradoxically aggravate the bleeding. Herein are described the cases of 2 patients with uterine AVM with abnormal vaginal bleeding. Both had a history of abortion followed by dilation and curettage. In both patients, the diagnosis of uterine AVM was established at Doppler flow ultrasonography. Treatment using transcatheter embolization was successful, and both patients had normal menstrual cycles at follow-up. One patient delivered a healthy baby 2½ years after transcatheter embolization. Journal of Minimally Invasive Gynecology (2011) 18, 812–819 © 2011 AAGL. All rights reserved.

Keywords: Arteriovenous malformation; Menorrhagia; Transcatheter embolization; Uterus

Uterine arteriovenous malformation (AVM) is a rare cause of menorrhagia that may manifest with life-threatening uterine hemorrhage [1,2]. Unlike other conditions, curettage is not therapeutic and may aggravate the bleeding [3,4]. The clinical diagnosis of uterine AVM is often unreliable. However, correct diagnosis can be reliably made using Doppler flow ultrasonography [5]. Other imaging methods including computed tomography and magnetic resonance imaging (MRI) may be used to complement the diagnosis in difficult cases and to characterize the architectural details of the AVM [6–8]. Herein are presented the cases of 2 patients with uterine AVM successfully treated using transcatheter embolization (TCE), a newer treatment method that has been used in several reported cases [3,9–11]. The literature about uterine AVM is also briefly reviewed.

Case Reports

Case 1

A 30-year-old woman, para 1 with 2 missed abortions, came to our hospital with a history of continuous vaginal bleeding for 3 months that did not respond to hormone therapy. Menarche occurred at age 13 years, and cycles were every 30 days and lasted for 4 days. The patient had 1 living child, 2½ years old, who had been delivered via lower segment cesarean section. In addition, she had 2 missed abortions, at 4 years and 1½ years previously; dilation and evacuation (D&E) was performed both times. Because of continuous bleeding via the vagina, a diagnostic D&E had been performed 1 month previously, and the histopathologic report stated proliferative endometrium. Bleeding had worsened after D&E. Subsequent ultrasonography of the pelvis revealed a hypoechoic area in the posterior myometrium, with internal vascularity. Color flow imaging demonstrated

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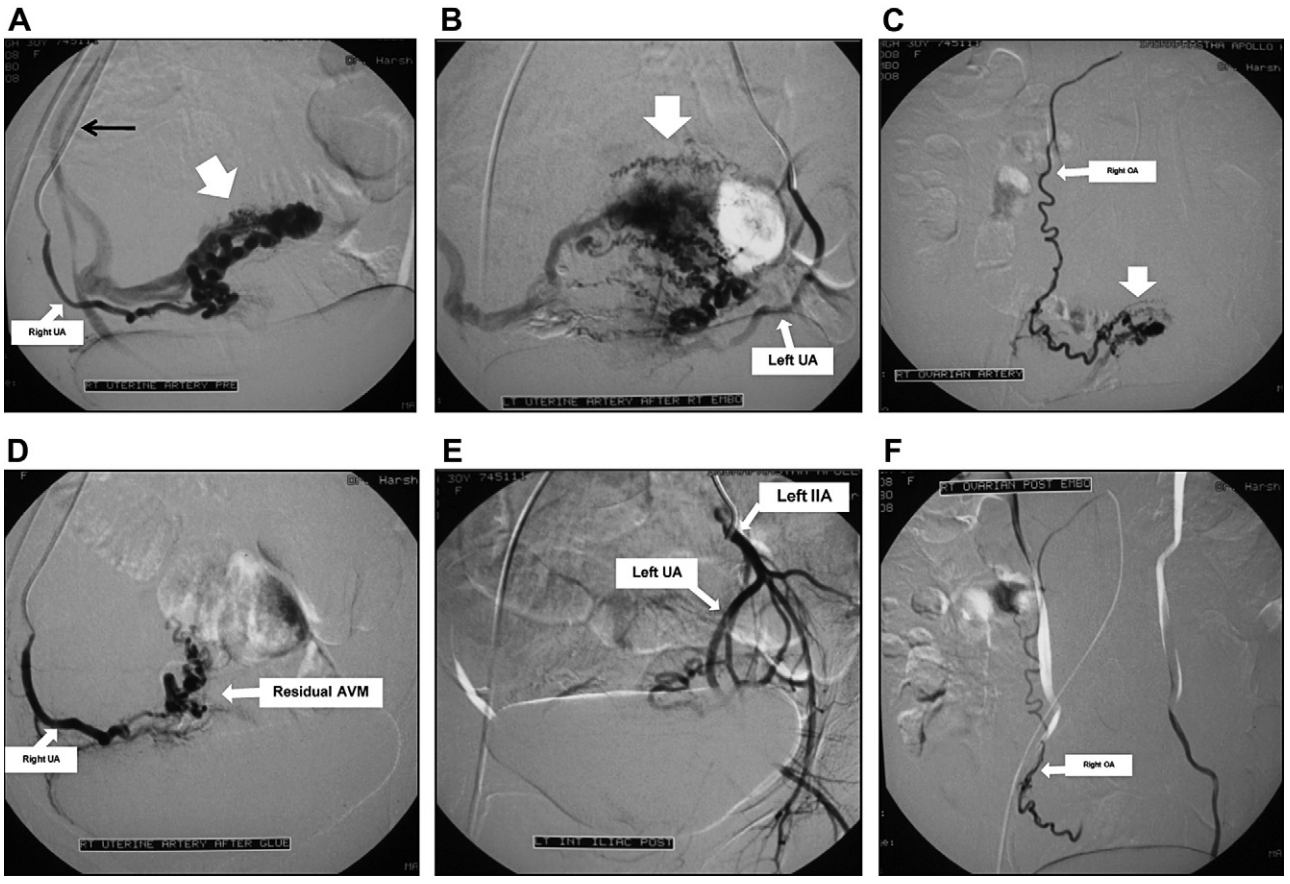
Fig. 1

Case 1. Pelvic angiographic findings. **(A)** Digital subtraction angiogram in anteroposterior view with the angiographic catheter (black arrow) selectively in the right uterine artery, demonstrating a uterine arteriovenous malformation (white arrow). **(B)** Selective digital subtraction angiogram of the left uterine artery in anteroposterior projection, demonstrating uterine AVM (arrow). **(C)** Selective digital subtraction angiogram of the right ovarian artery in anteroposterior view showing feeding of the AVM from the right ovarian artery. **(D)** Post-embolization digital subtraction angiogram of the right uterine artery in anteroposterior view showing obliteration of the AVM after selective embolization of the right uterine artery with glue. A small residual AVM is seen. **(E)** Post-embolization digital subtraction angiogram of the left internal iliac artery in anteroposterior view demonstrating obliteration of the AVM after selective embolization of the left uterine artery using absorbable sponge. **(F)** Post-embolization digital subtraction angiogram of the right ovarian artery in anteroposterior view demonstrating obliteration of the small AVM that was persistently observed after embolization of the right uterine artery. Note that blood flow to the right ovary is preserved. AVM = arteriovenous malformation; OA = ovarian artery; UA = uterine artery.

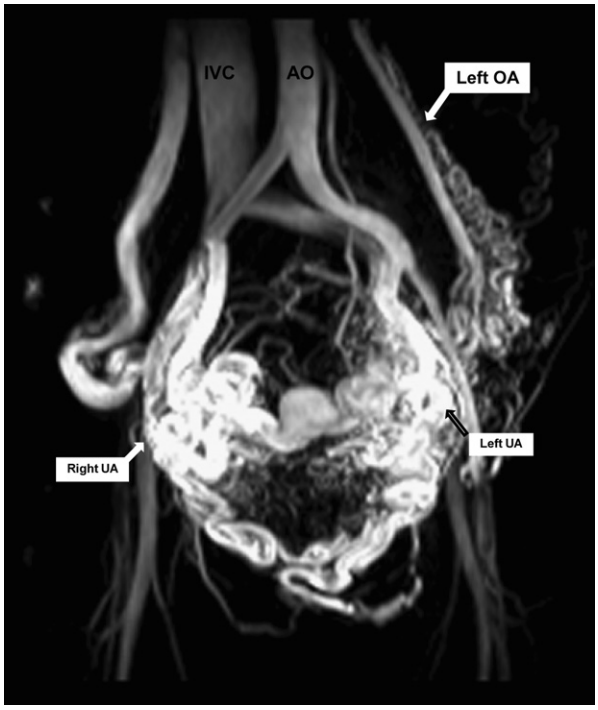


a florid color pattern with mosaic aliasing continuing into the right adnexal vessels and right uterine artery.

At examination, the patient was severely anemic, with normal general and systemic findings. The abdomen was soft, with no organomegaly. Vaginal examination demonstrated active bleeding from a healthy cervix. Uterus was of normal size, anteverted, and freely mobile, and the fornices were clear. Hemoglobin concentration was 7g/dL, and platelet count and coagulation parameters were normal. The provisional diagnosis of a large uterine AVM was made, which was confirmed at pelvic angiography performed before TCE of the AVM. The pelvic angiogram showed a large AVM in the right posterior part of the body and fundus fed by both uterine arteries and the right ovarian artery. The right uterine artery was embolized with glue (n-butyl cyanoacrylate, Samarth Life Sciences Pvt. Ltd., Mumbai, India; and iodized oil [Lipiodol], Guerbet Laboratories, Aulnay-sous-Bois, France). The small residual shunt was treated via embolization of the right ovarian and left uterine arteries using an absorbable gelatin sponge (AbGel; Sri Gopal Krishna Labs Pvt. Ltd., Mumbai, India). The blood supply to the right ovary was preserved (Fig. 1). The procedure and subsequent course were uneventful, and the patient received 1 unit of packed cells. After the procedure, the bleeding via the vagina did not stop immediately; therefore, the patient was given progesterone (Syserone 5 mg once daily) for 21 days. After this cycle, her menstrual periods were normal for 2½ years, when she conceived again. She delivered normally a healthy baby a week after she returned to her homeland.

Fig. 2

Magnetic resonance angiogram demonstrating a large uterine arteriovenous malformation filling from enlarged bilateral uterine arteries and the left ovarian artery. The pelvic veins are also grossly dilated. AO = aorta; IVC = inferior vena cava; OA = ovarian artery; UA = uterine artery.



Case 2

A 45-year-old woman, grava 4, para 0, had a history of irregular periods with heavy bleeding for 5 to 6 years. She had 4 spontaneous early pregnancy losses, followed by D&E each time; the last curettage was performed 6 years previously. Her cycles were every 30 to 60 days, for 4 to 5 days; the last menstrual period was 2 months previously. The patient also had a history of hypertension and hypothyroidism. General and systemic examinations yielded unremarkable findings. Vaginal examination revealed that the cervix was hypertrophied and congested; the uterus was irregular, anteverted, and 14 to 16 weeks of gestation in size; and pulsations were felt in both fornices. The hemoglobin concentration was 9.2 g/dL with normal platelet count and coagulation parameters. Pelvic ultrasound demonstrated a bulky uterus and markedly tortuous and dilated vessels in the myometrium and bilateral adnexae. At pelvic MRI, multiple tortuous flow voids were observed involving the uterus, bilateral adnexae, perivesical region, and pelvic cavity, along with a large venous pouch in the left adnexal region, and dilated and tortuous left ovarian and bilateral uterine arteries (Fig. 2). A pelvic angiogram showed fistulous communication on the left fed by branches of the left ovarian artery and draining into the pelvic veins, with fistulous channels observed near the inferior aspect of the left sacroiliac joint, from both uterine arteries to the pelvic veins (Fig. 3). Uterine artery embolization was performed in 2 stages using bilateral femoral arterial access. The left ovarian arterial feeders were embolized using coils (Cook, Inc., Bloomington, IN), and the left and right uterine arterial feeders were embolized using glue; the large venous pouch on the left side of uterus was embolized using glue after direct puncture under fluoroscopic guidance (Fig. 4). After the procedure, the patient experienced moderately severe pelvic pain (pain score of 5 on a scale of 0 to 10), for which acetaminophen 325 mg with tramadol hydrochloride 37.5 mg (Ultracet) was prescribed twice daily for 5 days, and half that dosage for another 5 days. The pain subsided completely within 10 days. The patient received low-molecular-weight heparin for 3 weeks to minimize the risk of extending thrombosis in the deep pelvic veins. She was well at 1-year follow-up, with regular menstrual periods.

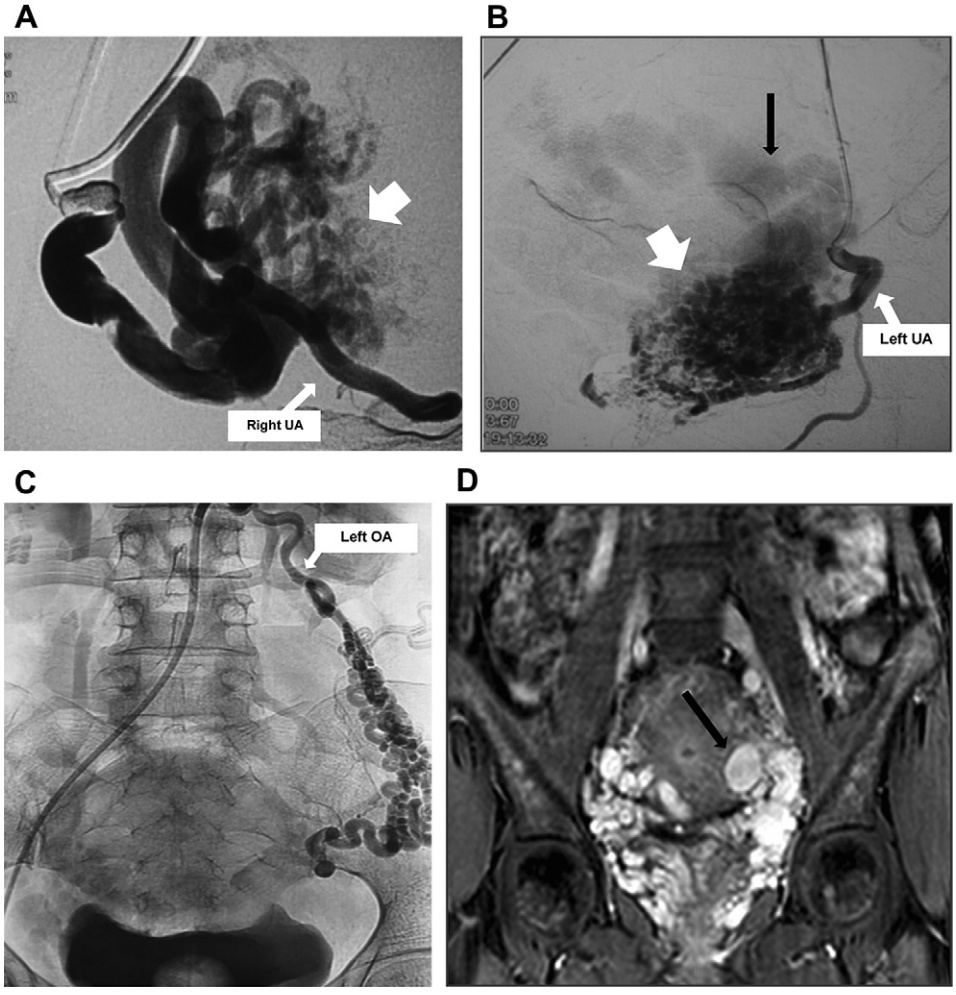
Discussion

Uterine AVM is a rare lesion with considerable risk potential. There are no published reports of the incidence of uterine AVM in the literature. In the series by O'Brien et al [12], uterine AVM was observed in 4.5% of 464 women aged 18 to 41 years in whom a pelvic sonographic examination was performed because of pelvic bleeding. The lesion can be congenital or acquired. Congenital uterine AVMs have multiple feeding arteries, a central tangle of vessels, and numerous large draining veins; these result from abnormal embryologic development of primitive vascular structures and tend to invade the surrounding structures [11,13–15]. Most congenital uterine AVMs are isolated anomalies, but can occur in association with AVMs at other sites [16,17]. Acquired uterine AVM is the predominant type of uterine AVM. It consists of multiple small arteriovenous fistulas between intramural arterial branches and the myometrial venous plexus, tends to have single or bilateral uterine artery feeders without an extrauterine arterial supply, and does not have a characteristic nidus [1,2,18,19]. Uterine AVM has been reported after D&C [20], therapeutic abortion [21], uterine surgery [22,23], cesarean section [24], direct uterine trauma [18], gestational trophoblastic disease [25], and exposure to diethylstilbestrol [26]; uterine AVM has also been reported after procedures such as sterilization [27] and hysterectomy [28]. Both of our patients had a history of D&E.

Patients with uterine AVM may be completely asymptomatic but typically present with intermittent, torrential vaginal bleeding, often in the setting of recurrent spontaneous abortion with normal β-human chorionic gonadotropin (β-hCG) concentration. Menorrhagia can at times be massive and life-threatening, and is thought to occur when the abnormal vessels are exposed from sloughing of the endometrium during menstruation or D&E [20,29]. Other symptoms may include throbbing discomfort in the

Fig. 3

Case 2. Pelvic angiographic findings. **(A)** Digital subtraction angiogram in anteroposterior view demonstrating enlarged right uterine artery feeding a large uterine arteriovenous malformation (AVM; arrow). Large fistulous communications are observed draining into the pelvic veins. **(B)** Digital subtraction angiogram in anteroposterior view showing enlarged left uterine artery feeding the uterine AVM (white arrow). A large venous pouch (black arrow) on the left side is also observed. **(C)** Conventional angiogram in anteroposterior projection showing an enlarged left ovarian artery feeding the uterine AVM. **(D)** Magnetic resonance image of the pelvis showing multiple tortuous flow voids involving the uterus, both adnexae, the perivesical region, and the pelvic cavity. A large venous pouch in the adnexal region (arrow) that was observed on the angiogram of the left uterine artery is also clearly visible in this magnetic resonance image. OA = ovarian artery; UA = uterine artery.

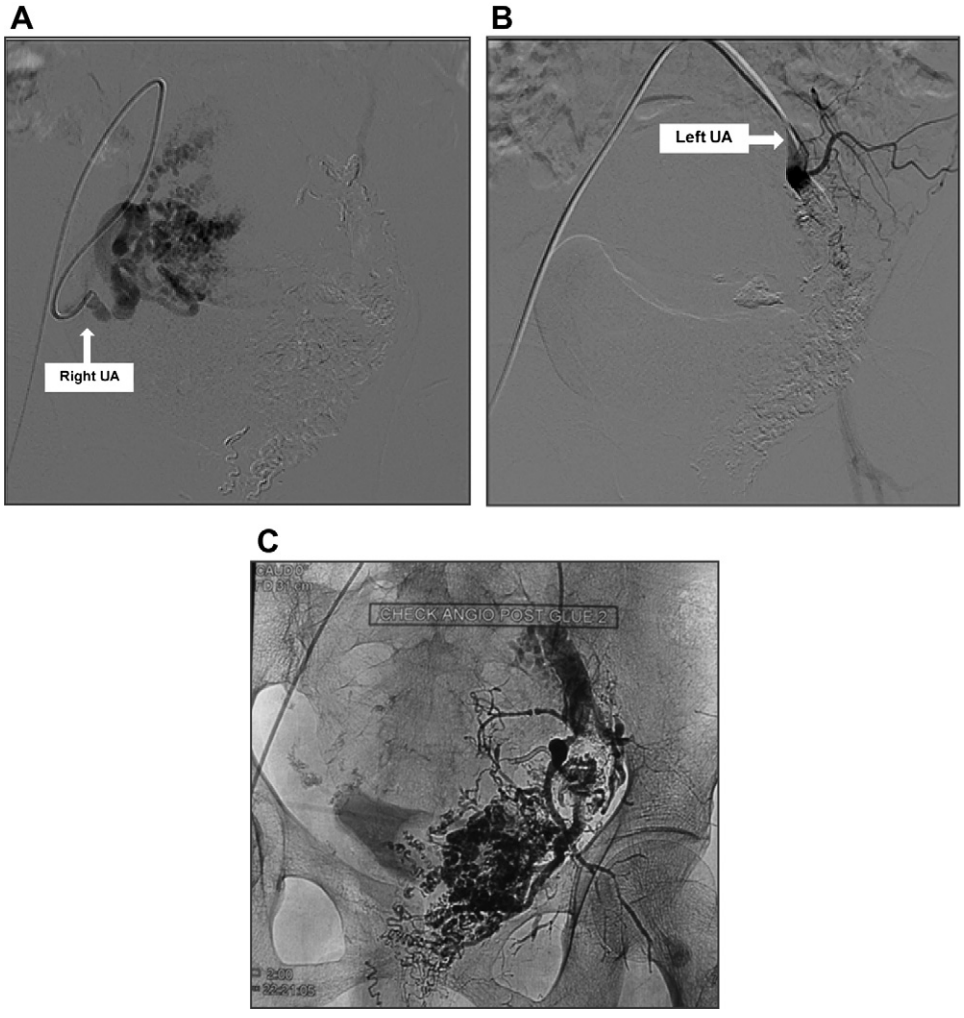


lower abdomen, urinary frequency or incontinence, and dyspareunia [1,30]. Some patients may have strong pelvic pulsations after exercise [5]. Systemic hypotension caused by blood pooling within the AVM and even cardiac failure have been described [11].

Clinical diagnosis of uterine AVM is often difficult, and requires a high index of suspicion. Previously, diagnosis of AVM was usually made at angiography, laparotomy, or pathologic analysis for investigation of abnormal vaginal bleeding. Currently, transvaginal ultrasonography is the imaging method of choice [31]. Typical features at gray-scale ultrasound include tortuous anechoic spaces in the myometrium without mass effect. Other features may include myometrial inhomogeneity, an intramural mass mimicking a myoma, and a large bulky cervix that mimics a cervical myoma or carcinoma [13,32]. However, such findings can also be observed in patients with positive β-hCG including those with retained products of conception, hydatidiform mole, or even intrauterine or ectopic pregnancy, with or without concomitant AVM [6,13,31–34]. Thus, the diagnosis of uterine AVM cannot be made on the basis of gray-scale findings alone, and color and spectral Doppler ultrasound should always be performed. The latter help in differentiating AVM by showing high-flow velocity (typically 4- to 6-fold higher than observed in normal myometrial vessels) with low resistance and mixing of arterial and venous waveforms [6,12,20,21]. The arteriovenous shunting in an AVM must be differentiated from malignant arteriovenous shunting

Fig. 4

(A and **B)** Post-embolization digital subtraction angiograms in anteroposterior view showing obliteration of the arteriovenous malformation (AVM) after selective embolization of the right and left uterine arteries using glue. **(C)** Post-embolization radiograph of the pelvis in anteroposterior view showing the final glue cast of the AVM after successful embolotherapy. See text for details. UA = uterine artery.



observed in neoplasia; neoplasms characteristically have low-volume high-velocity flow with drainage into a confined venous space, whereas AVM drains into a large low-pressure venous pool [35].

While rapid recognition of uterine AVM as the cause of bleeding is critical because uterine instrumentation may aggravate the condition [23], it is important not to overdiagnose the entity in the postpartum and post-abortion periods because many so-called uterine AVMs have spontaneously resolved at follow-up imaging [36]; further, withholding curettage for fear of heavy bleeding related to a possible AVM may result in unnecessary blood loss due to the presence of retained products of conception. In selected cases (as in our second patient), other imaging methods such as computed tomography and MRI may be used to establish the diagnosis. These imaging methods will also help to determine the size, extent, and vascularity of the lesion as well as involvement of adjacent organs [6–8]. Spin-echo MRI sequences typically demonstrate multiple flow-related signal voids within the lesion, corresponding to the tangle of vessels seen at Doppler interrogation [7,37]. Although contrast medium–enhanced angiography has been the conventional criterion standard diagnostic test, it is rarely used at the present time except when embolization therapy is being performed.

Contemporary management of uterine AVMs is dictated by the clinical status of the patient, age of the patient, site and size of the lesion, and future pregnancy aspirations. Isolated episodes of bleeding in a hemodynamically stable patient may be safely treated conservatively because many of these remain asymptomatic and may regress spontaneously [38]. In the series reported by Timmerman et al [37],

of 9 patients with uterine AVMs diagnosed at ultrasonography, 6 experienced spontaneous resolution, 2 patients with hydatidiform mole required chemotherapy, after which the AVMs resolved, and only 1 required embolization. Congenital AVMs do not regress spontaneously [16]. The medical management of AVMs may include therapy using estrogens, methylergonovine, danazol, and prostaglandins [1,29,39,40]. The potential mechanisms by which pharmacotherapy may be helpful have not been elucidated, and have been postulated to involve covering the bleeding vessels with proliferative endometrium facilitated by estrogen therapy and reduction in the blood flow to and collapse of the AVM using methylergonovine maleate [41]. Pregnancy after conservative medical management of AVM has been reported [38,41].

Large AVMs, especially those involving the subendometrial tissue, usually require surgical intervention. The commonly used surgical treatments include ligation of feeding vessels, resection of the uterine lesion, and oversewing of the lesion at laparotomy. Coagulation of the AVM under hysteroscopic guidance or of uterine vessels during laparoscopy has also been used to treat uterine AVMs [42,43]. Currently, hysterectomy to treat AVM is performed only in women who do not need or wish fertility preservation. Transcatheter arterial embolization is a minimally invasive treatment option with potential to preserve fertility because it does not seem to interfere with the menstrual cycle or pregnancy [3,9,10]. The need for angiography and TCE is not accurately predicted by the size of the AVM at imaging, and are generally performed if the patient has anemia, is hemodynamically unstable, or has recurrent bleeding. The procedure involves selective cannulation of the uterine arteries, usually from femoral arterial access at the groin with the patient under local anesthesia. The standard agent used for percutaneous treatment of AVMs is n-butyl cyanoacrylate, an agent that enables controlled and permanent obliteration of the AVM. We used glue to embolize the right uterine artery in our first patient, and the bilateral uterine arteries in our second patient because these were the main arterial feeders of the AVMs. Likewise, the venous pouch on the left side in our second patient was also embolized using glue because of its fairly large size. Other agents such as absorbable gelatin sponges, microfibrillar collagen, and polyvinyl alcohol have also been successfully used for embolization, especially when the target of embolization is more distal and further catheter advancement is difficult or impossible. Stainless steel coils are also commonly used for arterial embolization, but have the disadvantage that often several such coils are needed for obliteration of the AVM, and the necessity for repeated introduction of coils increases the procedure time and the risk of air introduction.

The technical success rate for TCE has been reported to be 79% to greater than 90% [1,10,44]. In some patients, multiple sessions of embolization may be needed (as in our second patient). Embolization failure may be attributed in part to the presence of adenomyosis. but is more often due to development of collateral vessels. Failure of embolization therapy can be managed with hysterectomy [18,42] or with uterine artery and ovarian ligament ligation when uterine preservation is desired [43]. Przybojewski and Sadler [45] reported a novel image-guided management of a uterine AVM after failed TCE in which they directly injected embolization material into the nidus of the AVM under ultrasound guidance and fluoroscopy after exposing the uterus surgically; the patient had a successful term pregnancy afterward. When embolization must be postponed, therapy with gonadotropin-releasing hormone agonists may be used to reduce the size of the AVM; such adjunctive therapy may increase the chances of successful TCE [46].

After successful embolization of a uterine AVM, hypovascularity of involved areas could, in theory, affect placentation and fetal growth; yet, several successful intrauterine pregnancies after TCE of uterine AVMs have been reported [19,47,48] including a successful twin pregnancy [49], which suggests that adequate collateral blood supply can develop to support a full-term pregnancy. Normal placental blood flow has been documented after previous TCE to treat uterine AVM [19]. In the study by O'Brien et al [12], normal menstrual cycles returned 2 months after TCE, and 5 patients became pregnant. Both of our patients had menses within 2 months of TCE therapy, and the first patient also delivered a healthy baby.

With experienced operators, TCE is generally safe. Minor complications including hematoma, urinary tract infection, retention of urine, and vessel or nerve injury at the vascular puncture site are common and require only mild supportive care or careful observation [50]. Varying degrees of pelvic pain are also common in the immediate postembolization period. Pain after TCE is probably due to ischemia produced by the embolization procedure, usually peaks on the first day after the procedure, responds well to analgesic therapy, and resolves in about a week [51]. Severe persistent pain necessitating administration of narcotic therapy may be due to ischemia or infarction of viable uterine tissue [52,53]. The moderate pain after TCE of the uterine arteries in our second patient could have been due to the large area of ischemia produced in a 16-week gestation uterus size. As in other arterial embolization cases, a "postembolization syndrome" consisting of pelvic pain, fever, leukocytosis (without left shift), nausea, vomiting, and malaise may sometimes develop after uterine artery embolization, and often requires hospitalization. Patients who develop or continue to have symptoms consistent with postembolization syndrome with foul-smelling vaginal discharge beyond 1 week after TCE should be evaluated for intrauterine infection. More serious complications after TCE of the uterine arteries are rare. Pelvic infection occurs sporadically after TCE, has been more often reported in cases of TCE performed to treat uterine myomas, is associated with significant morbidity, and may require emergency hysterectomy

[54–56]; fatal sepsis after TCE has also been reported [57]. Uterine necrosis is another rare life-threatening complication that mandates prompt treatment with antibiotics and hysterectomy. Loss of ovarian function can rarely develop after uterine artery embolization, in particular in women older than 45 years because of their more abundant uterine-ovarian arterial anastomoses compared with younger women [58,59]. Other serious potential complications of TCE may include perianal skin sloughing, uterovaginal and rectovaginal fistulas, neurologic deficits in the lower extremities, deep venous thrombosis, and pulmonary embolism [49,52,60,61]. There were no serious complications in our patients.

Conclusion

Uterine AVM is a rare but potentially serious cause of abnormal vaginal bleeding. Clinical findings in such cases are unreliable, and uterine curettage is not therapeutic and may even aggravate it at times; therefore, a high index of clinical suspicion is required. Diagnosis should be considered in all patients of reproductive age who have abnormal vaginal bleeding and negative β-hCG test results. Endovaginal ultrasonography is the imaging method of choice, and color and spectral Doppler imaging should be used routinely to enable the correct diagnosis. Transcatheter embolization is an excellent treatment option with a high success rate, low complication rate, and ability to preserve fertility.

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