A 27-year-old woman who was gravida 3 para 2 presented to an obstetrics and gynecology clinic with complaints of persistent pelvic pain and vaginal bleeding following medical termination of pregnancy with methotrexate (without dilation and curettage [D&C]) about 4 weeks earlier. Physical exam at that time was notable for a 12-week–sized markedly tender uterus. Pelvic ultrasound revealed a large, hypervascular uterus with a large uterine arteriovenous malformation (AVM) measuring at least 3.5 cm X 1 cm in the sagittal plane (Figure 1).

The patient had a past medical history significant for systemic lupus erythematosus, arthritis, genital herpes, and cervicitis. Her most recent prior pelvic ultrasound was unremarkable (about 2 months prior to presentation). The AVM malformation was confirmed with a pelvic CTA, which also demonstrated dilated vessels within the endometrium, raising the concern of retained products of conception (Figure 2). Because the patient desired future fertility, she was referred to the interventional radiology department for a possible uterine AVM embolization.

**PROCEDURE**

The embolization was performed as an outpatient procedure, utilizing a right groin access that was achieved under direct ultrasound guidance. A 4-F Omni™ Flush catheter (AngioDynamics) was advanced through a 5-F Pinnacle® introducer sheath (Terumo Interventional Systems) into the distal abdominal aorta, with subsequent aortoiliac angiography demonstrating patent vessels with filling of a large uterine AVM, predominantly supplied by the left uterine artery (Figure 3).

Figure 1. Sagittal ultrasound of the uterus showed a 3.5-cm X 1-cm uterine AVM at the time of initial presentation.

Figure 2. Pelvic CTA showing axial, coronal, and sagittal views of approximately 5-cm X 5-cm X 1.5-cm dilated disorganized vascular structures encompassing the anterior and lateral uterine walls and fundus.

Figure 3. Arterial (A) and venous (B) phase frontal aortoiliac angiogram demonstrated filling of a large uterine AVM, supplied by the left uterine artery without evidence of feeders from the right uterine artery (B).
the left uterine artery (Figure 3). The catheter was then manipulated into the left hypogastric artery (Figure 4). Subsequently, a straight 0.021-inch 130-cm Renegade® STC-18 Microcatheter (Boston Scientific Corporation) was then advanced into the distal left uterine artery over a Fathom®-16 Guidewire (Boston Scientific Corporation). Additional images were obtained (Figure 5) before delivering four vials of 700-μm Embozene™ Microspheres (Boston Scientific Corporation). Due to a small amount of residual flow to the AVM, a gelfoam slurry was then used for further embolization, after which, near stasis was achieved with no filling of the AVM (Figure 6).

Interrogation of the right internal iliac artery was also performed and demonstrated no feeding vessels to the AVM. Because the patient desired future fertility and no flow to the malformation was identified, the decision was made to stop the procedure at this point.

RESULTS
Approximately 3 hours after the procedure, the patient began experiencing profuse vaginal bleeding followed by tachycardia and hypotension. The patient was sent for emergent repeat angiography (Figure 7). Because the right side was not embolized, the repeat bleeding was thought to come from that source, but was negative. Left uterine artery evaluation also revealed no radiographic evidence of bleeding. Ovarian artery interrogation was also negative. Products of conception were of primary concern after reviewing the imaging. An emergent D&C was then performed, revealing remaining products of conception as the source. Bleeding ceased following the procedure. The patient stabilized and the rest of her hospital course was unremarkable aside from mild pelvic pain. A 3-month follow-up pelvic ultrasound revealed complete resolution of the uterine AVM with a normal-appearing uterus and endometrium (Figure 8).
DISCUSSION

Uterine AVMs are almost always acquired and rarely congenital. They are typically seen in premenopausal women, and risk factors include multiple gestations, D&C, intrauterine devices, cesarean section, and infection. Uterine AVMs result from failed development of a primitive capillary plexus resulting in multiple intra- and extrauterine feeding arteries and large draining veins with an intervening vascular nidus. Acquired uterine AVMs result from abnormal communications between the intramural arterial branches and the myometrial venous plexus without an intervening vascular nidus. Acquired uterine AVMs are typically easier to treat with transarterial embolization due to a lack of extrauterine arterial supply with only one or both uterine arteries supplying one or two feeding vessels.

Uterine AVMs have the potential for life-threatening vaginal bleeding, mandating early diagnosis and treatment. It is important to note that treatment for uterine bleeding with D&C is contraindicated in the setting of a uterine AVM, as it can paradoxically worsen the bleeding. Depending on the severity of symptoms, uterine AVM treatment options range from watchful waiting and medical management to endovascular or surgical intervention. If asymptomatic, monitoring is preferred; spontaneous resolution is common. If symptoms are minimal, medical treatment with oral contraceptives or danazol is a potential option, with the reasoning that decreased blood flow to the uterus may permit the AVM to thrombose. For stable to unstable patients with acute to subacute bleeding, the patient’s desire for future fertility must be considered when determining surgical versus endovascular intervention. During instances of acute massive bleeding that require emergent intervention or if future fertility is no longer desired, hysterectomy should be considered as a definitive option. Catheter-directed uterine artery embolization is preferred when future fertility is desired.

When performing uterine AVM embolization, angiography of bilateral uterine arteries must be performed to evaluate for cross filling of feeder vessels. Furthermore, cross filling may not be apparent on initial angiography. Embolization can be performed with gelfoam, polyvinyl alcohol particles, microspheres, or glue. Complications of uterine artery embolization include pelvic pain, perianal skin sloughing, uterovaginal or rectovaginal fistulas, and lower extremity neurologic deficits. Retained products of conception can potentially result in profuse postprocedural bleeding under the guise of a failed intervention. Following a negative repeat angiogram, retained products of conception should be reconsidered as the source, especially in the setting of recent pregnancy.

Juan Francisco Morales-Leon, MD
Department of Radiology
Tulane University School of Medicine
New Orleans, Louisiana
Disclosures: None.

Robert J. Raymond, MD
Department of Radiology
Tulane University School of Medicine
New Orleans, Louisiana
Disclosures: None.

Mary Brookes Ezell, MD
Assistant Professor, Department of Radiology
Section Chief, Interventional Radiology
Tulane University School of Medicine
New Orleans, Louisiana
Disclosures: None.

Bruce Bordlee, MD
Associate Residency Program Director
Vascular & Interventional Radiology
Tulane University School of Medicine
New Orleans, Louisiana
Disclosures: None.

James Caridi, MD, FSIR
Chairman, Department of Radiology
Professor of Vascular & Interventional Radiology
Tulane University School of Medicine
New Orleans, Louisiana
Disclosures: None.