Case Report

Diagnosis of a Uterine Arteriovenous Malformation by Duplex Sonography

HEIDRUN EBERHARDT, RDMS, DALE R. CYR, RDMS, THOMAS R. EASTERNLING, MD, DAVID A. NYBERG, MD, LAURENCE A. MACK, MD

Hypervascularity of the uterine wall during pregnancy has previously been described in both the sonographic and gynecologic literature. We report an arteriovenous malformation (AVM) presenting as an intervening mass separating the gestation from the cervix. By real-time imaging this mass appeared to be paruterine hypervascularity, but prenatal and postpartum duplex examinations were instrumental in the diagnosis of AVM, which was ultimately confirmed by postpartum arteriogram and pathologic examination.

CASE REPORT

A 22-year-old woman, gravida 4, para 2, abortion 1, was referred to our institution because a uterine mass was palpated. Sonography demonstrated a 12-week gestation with numerous serpiginous cystic structures adjacent to the lower uterine segment and cervix bilaterally. Duplex examination confirmed venous flow in these structures. At approximately 16 weeks' gestation the patient experienced severe cramping and vaginal bleeding. A repeat sonogram showed an abnormally elongated and thickened lower uterine segment (Fig. 1). The markedly hypervascular uterine walls and massively dilated uterine segmental veins were again noted, and flow was confirmed by duplex scanning. Additional findings included an anterior placenta previa with numerous large placental lakes and echogenic material in the posterior portion of the uterine cavity suggesting a small hematoma. Miscarriage was deemed inevitable, and labor was augmented with vaginal prostaglandin E2. The fetus was delivered spontaneously, but the placenta was retained. An uncomplicated dilatation and curettage was performed.

After 4 weeks a follow-up sonogram still demonstrated a moderately enlarged uterus measuring 12 cm in length and 6 cm in anteroposterior dimension with very prominent vessels involving the myometrium. A duplex examination at this time identified a large feeding vessel, which seemed to arise from the right iliac artery. Diffuse high-velocity arterial and venous signals (approaching 20 kHz) were recorded in the area, strongly suggestive of an arteriovenous malformation involving the uterine vasculature (Fig. 2). Two months after delivery an arteriogram confirmed a large AVM in the pelvis being primarily supplied by the anterior division of the internal iliacs bilaterally (Fig. 3). Successful placement of several oculding spring emboli coils into the iliac branches, minimizing the flow through the malformation. Because of the size of the malformation, which was contiguous with the uterus, and the patient's symptoms were significant, hysterectomy was performed. Pathologic examination revealed a large AVM involving the myometrium, cervix, and parametrium.

From the Departments of Radiology and Obstetrics and Gynecology, University of Washington, Seattle, Washington.
Correspondence: Heidrun Eberhardt, RDMS, Department of Radiology, University of Washington, 58-65, Seattle, WA 98195.
**DISCUSSION**

AVMs are defined as vascular fistulas that allow direct communication between arteries and veins. They are classified as being either congenital or acquired. AVMs of the uterus are very rare, with an etiology that is not entirely understood. The most common vessels in uterine/pelvic AVMs are the uterine and ovarian vessels, but multiple vessel involvement from surrounding structures is not uncommon.

Congenital AVMs are believed to be due to the embryologic failure of the common capillary plexus to differentiate into distinct arteries and veins. Other terms have been used to describe congenital AVMs, such as cirrhotic aneurysm, pulsating angioma, and congenital arteriovenous aneurysm. Congenital AVMs may be detected at any age, although most have been reported in the child-bearing years.

Acquired AVMs are most commonly caused by trauma. The most frequent causes are penetrating injuries, but surgical problems, malignant disease in the pelvis, trophoblastic disease, and radiation therapy have been reported to cause AVMs. One case demonstrated that necrosis of the placental chorionic villi caused direct arterial-venous communication.

Clinically, patients with uterine/pelvic AVMs usually present with pelvic pain, vaginal bleeding, or dyspareunia. Treatment for these lesions varies, with surgical intervention such as hysterectomy, salpingo-oophorectomy, or specific ligation of the involved vessels. Angiographically, intraarterial embolization of the malformation has also proven to be very effective. A combination of tech-
niques as in our case, requiring intraarterial embolization and surgery, is not uncommon.9

Prominent vasculature of considerable diameter surrounding the gravid uterus is not an uncommon finding. Hadlock et al report a 20% occurrence. This appearance is most striking between 18 and 32 weeks and is thought to represent a combination of choriod decidual sinuses and large draining uterine veins.1

Hypervascularity involving the gravid uterus could be mistaken for similar-appearing abnormalities such as degenerating fibroids, hydatidiform mole with coexisting fetus, or abruptio placentae.10 The sonographic observer should also be aware of the normal “subplacental complex,” which represents vascular channels at the placental-myometrial border. This is visualized more commonly in posterior placentas because of near-field artifacts obscuring anterior placental borders.11

Sonographic diagnosis of uterine/pelvic AVM has been previously reported.12,13 The findings in these cases correlated with the real-time studies in our case, which was a parauterine mass of multiple cystic, serpiginous structures that extended into the adnexa. The most striking entity of these cases, including the present case, was the mass effect the lesions caused by displacing surrounding structures. It has been our experience that normal parauterine vasculature does not cause a mass effect displacing other anatomic substructures.

The use of duplex sonography can aid the sonographic observer in detecting vascular flow to rule out nonvasculature etiologies. In this particular case, the ability to demonstrate abnormal blood flow allowed the proper diagnosis to be made.

We believe this is the first reported case of a uterine AVM diagnosed by duplex sonography. AVMs should be considered when exceedingly large parauterine vessels are noted that may or may not be causing a mass effect upon the uterus or surrounding structures.

REFERENCES