CASE REPORT

Sonographic Features of Uterine Arteriovenous Malformations: Two and Three dimensional Findings

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Uterine arteriovenous malformations (AVMs) are uncommon but occasionally serious and even life-threatening vascular lesions within the myometrium. They are detectable with traditional two-dimensional (2D) ultrasonography and color Doppler mapping. The diagnosis is made by visualizing a hypervascular region or mass within the myometrium. We report a case of uterine AVMs that was diagnosed with 2D and three-dimensional (3D) ultrasonography and confirmed by post-hysterectomy histology. When women have abnormal uterine bleeding with a heterogeneous myometrial mass, even in pregnancy, uterine AVMs should be considered.

KEY WORDS — arteriovenous malformations, ultrasonography

Introduction

Uterine arteriovenous malformations (AVMs) are rare disorders that can pose significant risks to patients. The clinical symptoms are highly variable, and can range from episodes of menorrhagia to life-threatening vaginal bleeding. AVMs occur most often after uterine trauma, surgery, endometrial carcinoma, cervical carcinoma, or gestational trophoblastic disease (GTD) [1–5]. Pelvic angiography has been the “gold standard” for confirmation of uterine AVMs [6–9], but it is invasive and thus is rarely performed for purely diagnostic procedures. Ultrasonography remains the most commonly used tool for investigating abnormal uterine masses. We present a case of uterine AVMs diagnosed with 2D and 3D ultrasonography.

Case Report

A 36-year-old female, gravida 4, para 1, visited our outpatient clinic complaining of intermittent vaginal bleeding for 5 months. Her medical history revealed that she had a dilation and curettage (D&C) due to a delayed miscarriage about 2 years beforehand. After the surgery of D&C, she became pregnant again and had an uneventful course until...
the second trimester, when intermittent antepartum hemorrhaging began. In the third trimester, a progressively enlarged hypervascular fundal tumor was found. In spite of medication and hospitalization, her hemorrhaging persisted. At term, a female infant was born without incident, but then the mother developed postpartum hemorrhaging. The bleeding was controlled with conservative treatment, including oxytocin and blood component therapy. After this, the patient had several episodes of moderate painless uterine bleeding unrelated to her menstrual periods.

At our hospital, 2D ultrasonography revealed a heterogeneous mass over the fundal area of the myometrium. With color Doppler mapping, a tangle of vessels with high-velocity flow was found (Fig. 1). Also 3D ultrasonography with power Doppler mapping was used to delineate the uterine mass. A commercially available GE Voluson 730 Expert Ultrasonography System Scanner (GE Medical Systems, Milwaukee, WI) equipped with a multifrequency transvaginal volumetric probe (3–9 megahertz, or MHz) was used.

The 3D ultrasonography and power Doppler imaging method used to visualize the uterine fundal mass was similar to that described in previous studies [10]. We set the color area of the 2D power Doppler to cover the area of the uterine mass. The volume angle was set at 65 degrees. During data acquisition, the transducer was not moved and the patient was asked to hold her breath for 5 to 7 seconds. The 3D ultrasonography with surface rendering mode revealed a tangle of tortuous vessels. The Virtual Organ Computer-aided Analysis (VOCAL) program was also applied (Fig. 2). The measured vascularization index (VI) was 73.684; the flow index (FI) was 70.23; and the vascularization-flow index (VFI) was 51.748. The value of VI represented the blood vessels within the tissue, expressed as a percentage. The value of FI showed the average blood flow intensity. The VFI is the average color value of all the grey and color regions, representing both blood flow and vascularization. These results indicated high blood flow and moderate amounts of vascularization in the uterine AVM lesion. These 3D ultrasonography findings increased our confidence in the diagnosis of AVM (Fig. 3).

To exclude the possibility of GTD, we measured the patient’s serum human chorionic gonadotropin (hCG) level. The serum hCG level was less than 1.2 mIU/mL, and an AVM was strongly suspected. Magnetic resonance imaging (MRI) showed massively dilated vessels on the fundal region with an early venous return from a markedly dilated left renal vein, which also suggested an AVM. Transcatheter arterial embolization (TAE) was suggested, but the patient declined the procedure and requested a hysterectomy instead. Subsequent pathologic examination proved the diagnosis of uterine AVMs (Fig. 4).
Discussion

All uterine AVMs are the result of abnormal communication between arteries and veins. Large numbers of morphologically abnormal vessels secondary to aberrant angiogenesis are usually detected by pathologic examination [2,11]. Uterine AVMs are infrequent, and the true incidence is still unknown [6–8]. When O’Brien et al [9] performed pelvic ultrasonography in 464 patients with abnormal uterine bleeding, they found 21 uterine AVMs, an approximate incidence of 4.5%.

The most common clinical presentation of AVMs is bleeding. However, the bleeding is widely varied, ranging from menometrorrhagia to severe and even life-threatening bleeding. The causes can be congenital or acquired. Acquired malformations are mainly due to previous uterine trauma or surgery, such as curettage or pelvic surgery, or are caused by endometrial and cervical carcinoma in situ, maternal diethylstilbestrol (DES) exposure, or GTD [1–5].

In our case, although a D&C was performed 2 years before the AVMs occurred, we felt the D&C was the cause of the subsequent AVMs. One possible explanation for the long interval between injury and symptoms was that the first symptom of the patient’s AVMs was not severe enough to cause her to seek medical help. It was only after the patient had her antenatal examinations that the hypervascular mass of AVMs was detected on ultrasonography.

Few reports of uterine AVMs in pregnancy can be found in the literature [12–15]. In some cases, pregnant patients had profuse uterine bleeding and required hysterectomy or other kinds of surgical interventions. Other asymptomatic patients may have had preterm deliveries. We speculate that the uterine AVMs in our case developed before our patient’s last pregnancy and that her subsequent postpartum hemorrhage may have been highly related to uterine AVMs. A comprehensive review of her history and 2D and 3D ultrasonography gave us an earlier, more definitive diagnosis. Using this approach enables us to inform patients earlier and can help lower the complications of uterine AVMs.

Ultrasonography is the tool most commonly used for investigating abnormal uterine bleeding. One diagnostic characteristic of uterine AVMs is subtle myometrial heterogeneity seen on gray-scale ultrasonography. With color and spectral Doppler ultrasonography, a tangle of vessels can be seen as arteriovenous shunting with high-velocity, low-resistance flow [2,16,17]. Castro-Aragon, Timmerman, Ghi and their colleagues [12,16,17] recommend that color and spectral Doppler imaging studies should be performed on all females who had heavy vaginal bleeding, regardless of findings on gray-scale ultrasonography. A similar sonographic picture can be seen in patients with positive beta-hCG
findings. When this sonographic finding and positive beta-hCG results are present, the diagnosis is usually pregnancy-related, including intrauterine pregnancy, ectopic pregnancy, retained products of conception, or GTD [17].

Treatment of uterine AVMs is based on the clinical status of the patient. Patients with uterine AVMs who are hemodynamically stable can be treated conservatively [2,7]. Conservative management of uterine AVMs includes use of combined oral contraceptives, oral methylergonovine maleate, Danazol (Ladogal) and gonadotropin-releasing hormone (GnRH) agonists [18–21]. These medications can reduce estrogen levels and subsequently shrink uterine AVMs. If patients are anemic or hemodynamically unstable, interventional therapy may be necessary.

The most popular method of intervention cited in the literature is transarterial embolization (TAE) [7–9]. However, because of the possibility of embolization to other sites and varying success rates with TAE, other treatment options can also be used to manage uterine AVMs. Hysterectomy was the most common method used to treat uterine AVMs before TAE developed. Other less radical surgeries, including removal of the AVM lesion, laparoscopic bipolar coagulation of the uterine vessels, and unilateral ligation of uterine artery and ovarian ligaments have been reported [22–23]. In our case, the patient preferred a hysterectomy because she had experienced repeated episodes of vaginal bleeding in spite of conservative treatment.

Hata et al [24] recommend that 3D ultrasonography with power Doppler mapping can be used to clearly depict the vascular lesion with tangled vessels in the myometrial layer. Ghi et al [17] also recommend using this kind of imaging modality. Both groups propose that 2D and 3D power Doppler ultrasonography with negative findings of serum hCG can be used in the differential diagnosis of uterine AVMs and other hypervascular lesions within the myometrium. Our findings were also compatible with these previous conclusions. Furthermore, we also noted high VI, FI, and VFI measurements in the uterine AVMs under 3D color power Doppler imaging. These data helped make the diagnosis of AVMs because of the highly vascular nature within the myometrial mass. To the best of our knowledge, our case is the first case diagnosed with 2D and 3D ultrasound and actually confirmed by pathologic examination. Before this, nearly all cases were confirmed by TAE not pathologic examination.

In conclusion, if women have abnormal uterine bleeding with a heterogeneous myometrial mass, even in pregnancy, uterine AVMs should be considered. Using 2D and 3D power Doppler ultrasonography can help us correctly make the diagnosis of uterine AVMs.

References


