Successful Treatment of Uterine Arteriovenous Malformation with Percutaneous Embolization

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SUMMARY

Objective: Uterine arteriovenous malformation (AVM) is a rare condition and can be life-threatening if not managed properly. We report a case that was diagnosed by typical ultrasound imaging and treated successfully with uterine arterial embolization.

Case Report: A 28-year-old female, gravida 4, para 3, abortus 1, presented with massive vaginal bleeding 19 days after a termination of pregnancy due to fetal anomaly. After a dilatation and curettage 3 years previously, typical ultrasound image findings and a declining pattern of serum β-hCG (human chorionic gonadotrophin), acquired AVM was highly suspected. The patient underwent bilateral uterine arterial embolization. Four weeks later, there was nearly complete resolution of the AVM and the patient's menstrual cycle was restored 6 weeks after embolization.

Conclusion: AVM can be diagnosed at an early stage with the aid of history taking and ultrasound. Percutaneous embolotherapy is a safe and effective treatment for AVM, especially when fertility preservation is desired. [*Taiwanese J Obstet Gynecol* 2007;46(1):60–63]

Key Words: arteriovenous malformation, color Doppler ultrasound, embolization

Introduction

Arteriovenous malformation (AVM) is a tangle of various size vessels with arterial and venous histological characteristics but without evidence of an intervening capillary network [1]. Uterine AVM is classified as either congenital or acquired. Definitive diagnosis has been traditionally made by angiography. With the development of color Doppler and power Doppler ultrasound, diagnosis is now easier. To preserve fertility, uterine arterial embolization is a treatment option. We report a case of acquired AVM that was treated successfully with percutaneous embolotherapy.

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Case Report

A 28-year-old female, gravida 4, para 3, abortus 1, presented with massive vaginal bleeding 19 days after termination of pregnancy due to fetal anomaly. Her first two children had been delivered via vagina uneventfully. No anomalies were noted. Elective dilatation and curettage (D&C) had been performed 3 years previously for personal reasons. The fourth pregnancy was terminated at 22 weeks gestational age due to multiple fetal anomalies. The female baby was born via vagina with an Apgar score of 0 and 0 at 1 minute and 5 minutes, respectively. Multiple anomalies were noticed, including cleft lip, cleft palate, low set ears, nasal bone hypoplasia, and sacral area meningocele, which were compatible with the prenatal sonographic diagnosis. The complete placenta, weighing 156 g, was delivered spontaneously. Chromosomal study of the cord blood and placental tissue showed a normal female 46,XX karyotype.

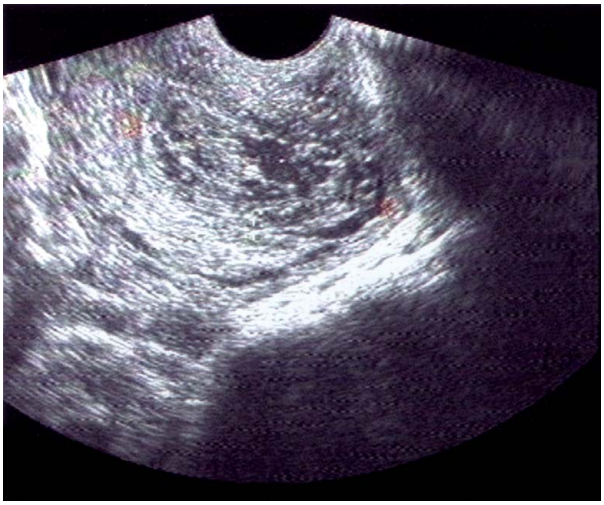

Figure 1. Transvaginal grayscale ultrasonography shows numerous tortuous anechogenic structures over the fundal myometrium of the uterus.

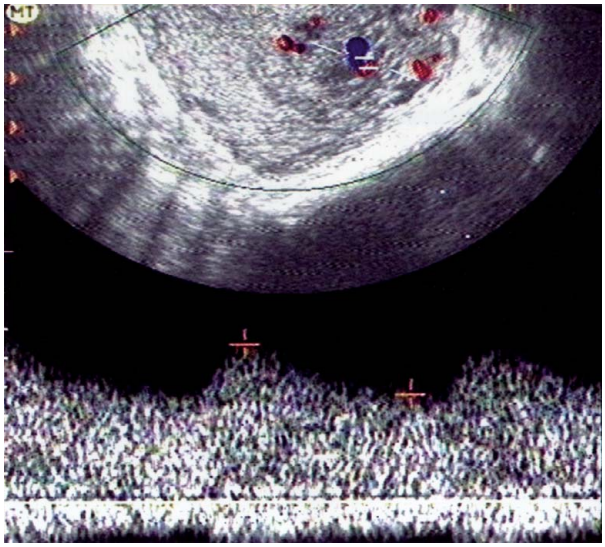

Figure 2. Endovaginal sonogram of the uterus with spectral Doppler tracing obtained within the uterine arteriovenous malformation. Note the characteristic high-velocity peak systolic flow with associated high diastolic flow and low-resistance pattern (resistance index, 0.32).

The postpartum care was uneventful. She was discharged the day after the termination.

During the postpartum period, minimal vaginal bloody discharge was noted. Otherwise there was no other discomfort. However, massive vaginal bleeding developed on day 19 postpartum. She visited our emergency room (ER) where ultrasonography showed intrauterine content, suspected to be retained placenta or blood clot. Her vital signs were stable. Hemoglobin level was 14.0 gm/dL. As uterine bleeding due to retained products of conception was suspected, 0.2 mg of intravenous methylergonovine maleate and 20 units of intravenous oxytocin were administered. She was discharged from the ER after the vaginal bleeding subsided. The following day, she returned to our Outpatient Department, presenting with minimal but persistent vaginal bleeding. Transvaginal sonogram showed an ill-defined area of myometrial inhomogeneity 3–4 cm in diameter at the fundal area of the uterine wall and intrauterine content (Figure 1). Color Doppler ultrasonography revealed high-velocity and multidirectional flow in the myometrium. Spectral Doppler ultrasonography revealed low-resistance flow, and high-velocity arterial and pulsatile venous flow (Figure 2).

To make a differential diagnosis between AVM and gestational trophoblastic disease, serials of serum β-hCG (human chorionic gonadotrophin) were checked and they showed a declining pattern (30.4, 15.0, 16.1, 7.74, and 5.05 mIU/mL). The ultrasound findings revealed progressive slow enlargement of the lesion. During the 1 month follow-up period, the patient presented with minimal vaginal bleeding and no further episodes of massive vaginal bleeding. However, the lesion showed no sign of resolution.

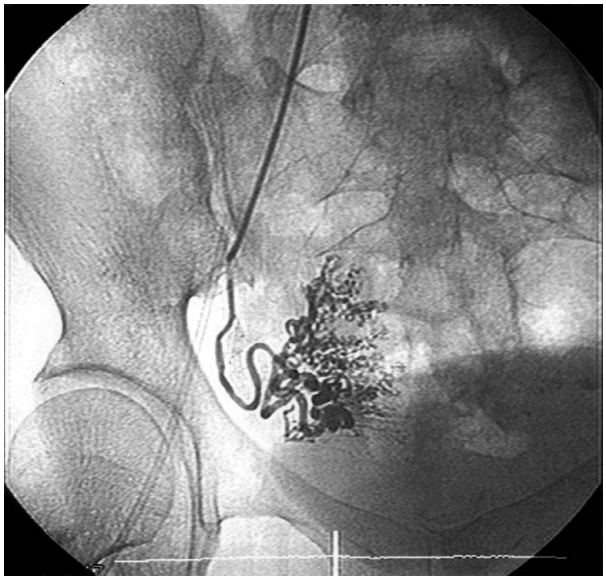

Figure 3. Pelvic angiogram demonstrates enlarged right uterine artery supplying a large vascular mass.

Percutaneous embolotherapy was performed on day 57 postpartum due to suspicion of AVM. Angiography showed enlarged bilateral uterine arteries supplying a large vascular mass conforming to AVM (Figure 3). Uterine arterial embolization was performed using coils and gelform. Dye stasis was noted after embolization (Figure 4). The patient had an uncomplicated postembolization course and was discharged on the third day of hospitalization.

Four weeks after embolization, ultrasonography showed the lesion as almost completely resolved. During

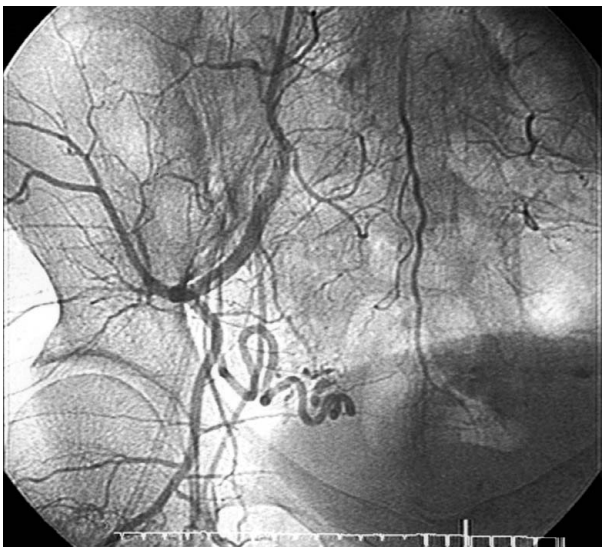

Figure 4. Pelvic angiogram obtained after embolization shows no residual opacification and complete embolization of the arteriovenous malformation.

resolution, the vaginal discharge changed its color to brown. Four weeks after embotherapy, the patient was asymptomatic. Her menstrual cycle restarted 6 weeks after the embotherapy.

Discussion

AVM is a tangle of vessels of different sizes with arterial and venous histological characteristics but without evidence of an intervening capillary network [1]. Although the pelvis is a common site for AVM, only occasionally does it involve the uterus.

Uterine AVM has traditionally been classified as either congenital or acquired. Congenital AVMs are believed to result from an arrest of normal vascular embryological development and tend to extend beyond the uterus into the pelvis, with frequent feeders from pelvic vessels other than the uterine arteries [2]. In contrast, acquired AVMs are usually associated with previous uterine surgery, curettage, carcinoma of the cervix, and endometrial or trophoblastic disease [1].

Varying degrees of vaginal bleeding have been the most common symptom of uterine AVM, often after an invasive procedure such as uterine curettage or therapeutic abortion. Bleeding may occur when the vessels erode through the endometrium and become exposed or shedded [3]. Ghai et al [1] enrolled 15 patients with AVMs on whom 25 embolization procedures were performed. Each patient in the study had a history of D&C and/or uterine surgery performed 1 month to 7 years before the diagnosis of AVM (median 19 months). We assumed that our patient's AVM was related to the elective D&C performed 3 years previously, since neither manual removal of the placenta nor curettage was performed during the recent vaginal delivery. There were no other related risk factors for AVM in this patient. Uterine AVM should be suspected in case of unexplained vaginal hemorrhage in any patient who has a history of therapeutic D&C, abortion, previous uterine surgery, endometrial carcinoma or gestational trophoblastic disease, regardless of how long back such events occurred.

With the development of color Doppler ultrasonography, diagnosis of AVM has become easier. Typical findings are high-velocity and multidirectional flow in the myometrium. Spectral Doppler ultrasonography reveals low-resistance flow and high-velocity arterial and pulsatile venous flow [4,5]. As reported recently by Ghi et al [6], three-dimensional power Doppler sonography provides a clearer description of the vascular lesion, showing tortuous vessels of the grove being imaged in the context of the myometrial layer. Other diagnostic techniques, such as contrast-enhanced computed tomography and magnetic resonance imaging, have also been helpful and provide information about the degree of involvement of adjacent organs [7,8]. However, to distinguish between true and false AVM, X-ray angiography must be performed. True AVM reveals early venous filling, while false AVM shows late venous filling [6]. Moreover, correction with serum β-hCG is important in differentiating vascular malformation from trophoblastic disease or neoplasia.

Although hysterectomy has been used as the ultimate treatment, transcatheter embolization is preferable for the preservation of reproductive capability. Ghai et al [1] had a technical success rate of 100% with embolization. The clinical success rate was 93%: bleeding was controlled in 14 out of 15 patients and one patient underwent a hysterectomy. Therefore, the authors concluded that transcatheter embolization was a safe and effective treatment for traumatic AVMs. For patients with continued desire for fertility, it should be considered as the primary treatment option.

Other methods for noninvasive management of uterine AVMs have been reported, including two case reports regarding methylergonovine maleate [9,10]. In addition, Takeuchi et al [11] reported a woman whose case was managed successfully with danazol (an isoxal derivative of 17-ethinyl testosterone) after failed embolization of AVM. However, reported cases are so few that the effectiveness of methylergonovine maleate or danazol on uterine AVMs has not been proven yet and should be used only in select cases with close clinical follow-up.

Although rare, spontaneous regression of AVM has also been proposed. Mohamed et al [3] reported a 28-year-old female with massive vaginal bleeding post

D&C. Acquired AVM was diagnosed by ultrasound. Since no further episodes of vaginal bleeding occurred, her case was managed by close observation only. Spontaneous complete resolution of AVM and normalization of the menstrual cycle occurred after 2 months. In our case, the patient's case was managed with close observation initially. However, during the subsequent 1 month period, her AVM showed no sign of resolution and vaginal bleeding persisted. She requested that her fertility be preserved and so embolotherapy was performed.

In conclusion, our case illustrates an acquired uterine AVM that most likely resulted from a D&C. Since the time period between D&C or other uterine procedures, and the presence of AVM varies, one has to expect the possibility of acquired AVM as an explanation for persistent abnormal uterine bleeding. Noninvasive management, such as methylergonovine maleate, danazol, or observation with careful follow-up, can only be used for patients with minimal symptoms and good compliance. For those with intractable vaginal bleeding, persistent evidence of AVM, or poor compliance with regular follow-up, surgical intervention should be undertaken. Since transcatheter embolization allows for fertility preservation [12], it should be considered as the primary treatment option.

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