Menorrhagia is a common disorder during the reproductive age. It often results from myometrial or endometrial lesions. Menorrhagia caused by uterine arteriovenous malformations (AVMs) is a rare but life-threatening event. The major presentation of uterine AVMs is characterized by painless, intermittent, abrupt and profuse vaginal bleeding that is usually refractory to medical treatment [1]. The appropriate management of AVMs relies considerably on accurate diagnosis [2]. In order to achieve a prompt diagnosis, it is essential to raise a clinical suspicion of uterine AVMs in patients with unaccountably intractable vaginal hemorrhage with specific bleeding characteristics.

The patient was a 38-year-old woman, gravida 2, para 2 (via cesarean section twice). Her last cesarean delivery was twelve years prior to this presentation, and her menstrual cycle had been regular without any experience of excessive menstrual flow after the delivery. She came to the emergency department complaining of sudden intermittent, painless and profuse vaginal bleeding with blood clots on the seventh day of her period. Vaginal spotting was the only unusual sign shortly before the event. She denied having any underlying hematologic disorders or drug exposure such as hormonal therapy. Pelvic sonography showed thin endometrium without definite uterine tumors (Figure 1). On pelvic examination, the appearances of the uterine cervix and vagina were grossly normal without traumatic lesions or tumor formations. However, there was intermittent flow of blood gushing out from the endocervical canal, and the amount of blood loss was approximately 500 mL within minutes. Results of coagulation studies were within normal limits, and urine β-hCG test was done. Immediate hemodynamic resuscitation was done, with intravenous fluids supplement and blood component therapy. The hemorrhage was not responsive to intravenous medicines such as transamines (oxytocin) and uterotonic (methylergonovine). Subsequently, uterine tamponade with a Foley balloon was performed, and the bleeding gradually decreased. Eleven hours after the balloon tamponade, she experienced a gush of profuse vaginal bleeding again. As uterine AVMs were suspected, pelvic angiography was recommended for further diagnosis and treatment; however, the patient refused. Finally, the couple opted for surgical hysterectomy to avoid the possible failure of conservative treatment. Gross examination of the uterine cavity revealed one small vascular clump with laceration at the right isthmus (Figure 2A), and a well-defined hemorrhagic lesion was found at the isthmus of the formalin-fixed uterine wall (Figure 2B). Microscopic examination of the right isthmus showed dilated and tortuous vessels with mixed thick and thin vascular walls, which was consistent with uterine AVMs (Figure 3). The hospital course of the patient before the hysterectomy is summarized in the Table. The postoperative course was uneventful, and she was discharged on the fourth postoperative day.

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Accepted: June 22, 2007

Rupture of Uterine Arteriovenous Malformation as a Cause of Severe Menorrhagia

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Figure 1. Transabdominal sonography reveals a well-defined uterus with no tumors and a thin endometrium (blood clots in the vagina).
Menorrhagia from Uterine AVMs Rupture

6 units of packed red blood cells and 6 units of fresh frozen plasma.

AVMs are formed by large and dysplastic feeding arteries, resulting in decreased vascular resistance. Lesions in the uterus are very rare, and they can be congenital or acquired lesions, with the latter often associated with prior uterine surgery or pregnancy. The major manifestation is abrupt, intermittent and profuse vaginal hemorrhage associated with menometrorrhagia and immediate or delayed postpartum hemorrhage [1,3]. Hormonal changes are thought to trigger the bleeding episode of uterine AVMs, such as those seen during menstruation and pregnancy [4]. Because of its life-threatening potential, uterine AVMs should be highly suspected in patients who have experienced an episode of unexplained vaginal hemorrhage with specific bleeding patterns.

Prompt accurate diagnosis and appropriate treatment are important. Uterine AVMs can be diagnosed by using contrast-enhanced computed tomography, magnetic resonance imaging, color Doppler ultrasonography, angiography, and magnetic resonance angiography [5,6]. Angiography is traditionally helpful in making a definitive diagnosis and can determine the vascular supply and guide embolization therapy. As soon as the diagnosis is made, treatment should be started. In the literature, several cases were successfully treated with only medications such as methylergonovine and prostaglandin F2α [7]. But heavy vaginal hemorrhage often fails to respond to medical treatment if rupture of uterine AVMs has occurred. Recently, conservative management with laparoscopic bipolar coagulation of the bilateral uterine vessels has been proposed as an alternative approach [8]. Embolization performed simultaneously with angiography can be a good therapeutic technique, and it is a promising option for preserving future fertility after the procedure [9]. However, failure of embolization may result from the recurrence of uterine AVMs due to abundant collateral circulation and revascularization. Surgical hysterectomy is inevitable and reserved for the cessation of the uncontrollable uterine bleeding. Therefore, the treatment of AVMs depends on the severity of vaginal hemorrhage, the patient’s age, and future reproductive desires. However, we discussed with the patient about the available treatment options in the hospital; she still refused uterus-preserving treatment such as angiography.
Our experience indicates that uterine AVM rupture should be suspected in patients presenting with an episode of intermittent, abrupt and heavy vaginal bleeding without the detection of any source of bleeding such as endometrial hyperplasia, and cervical or vaginal lacerations. Temporary hemodynamic stabilization of uterine AVMs rupture can be achieved by intrauterine balloon tamponade. A Foley catheter is inexpensive, readily available, and easily inserted into the uterine cavity without any special techniques [10]. The procedure is valuable while awaiting opportunity to perform a definitive treatment such as embolization or hysterectomy. In addition, vaginal bleeding from the abnormal vascular structure within the uterine cavity (Figure 2A) may be aggravated by uterine instrument usage such as dilatation and curettage; therefore, it is important to be aware of this uncommon condition in order to prevent additional excess uterine bleeding from inappropriate procedures.

References