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# Enhanced Myometrial Vascularity – A Rare Cause of Abnormal Uterine Bleeding

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**Abstract**

We present a case of a woman who was diagnosed with enhanced myometrial vascularity (EMV) following a uterine aspiration due to retained products of conception (RPOC) following a first-trimester miscarriage. EMV is a rare condition characterized by a transient increase in blood flow within the myometrium, which is almost exclusively observed in the context of recent pregnancy. The clinical spectrum of EMV can range from asymptomatic to life-threatening bleeding. Vaginal ultrasonography with color Doppler is the initial diagnostic procedure for EMV, although it lacks specificity, necessitating a detailed clinical context for accurate diagnosis. Digital subtraction angiography is considered the gold standard for the diagnosis of EMV, but due to its invasive nature, it is reserved for patients requiring embolization. The optimal treatment plan for each patient depends on their specific symptoms and may involve a combination of expectant and surgical management. However, the most effective and appropriate approach remains to be determined. It is crucial to be cognizant of this condition to facilitate an expedient diagnosis and to potentially circumvent invasive therapeutic interventions.

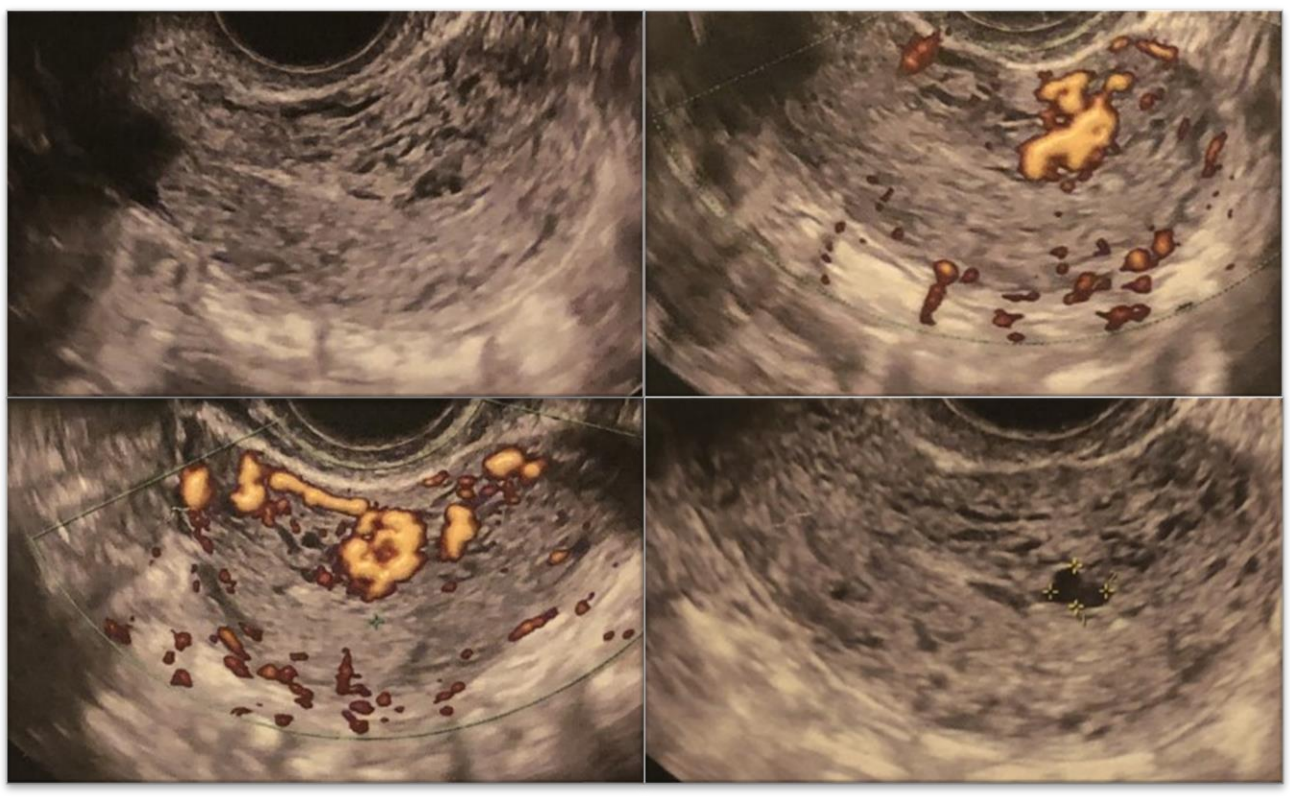
## Clinical Case

A 30-year-old nulliparous healthy woman, with no history of smoking or regular medication intake, and with no relevant family history, presented with an early pregnancy loss in January 2022 which needed uterine aspiration due to retained products of conception (RPOC).

In the pre-conception blood tests, she had a hemoglobin (Hb) of 15g/dL and a negative study for Von Willebrand Disease, which was requested based on a history of heavy menstrual bleeding in adolescence.

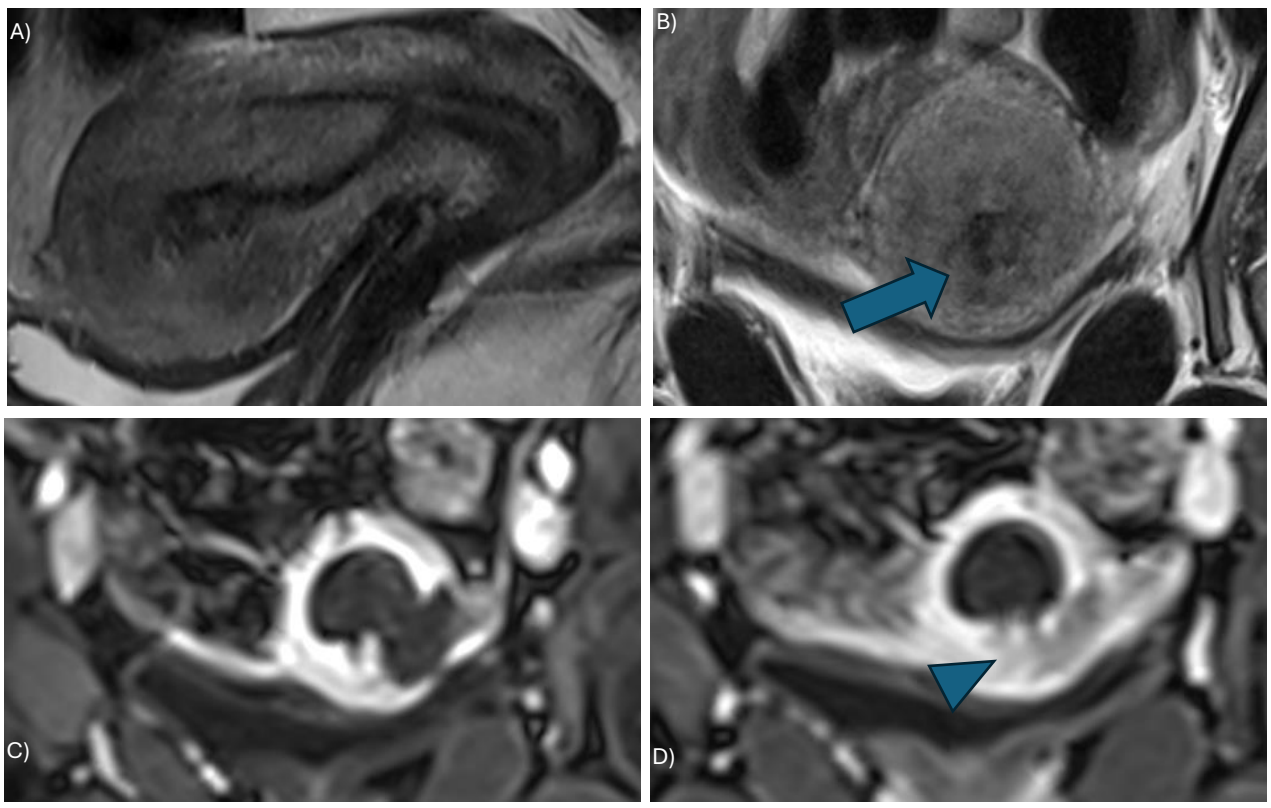
Two months after the surgical procedure, she was asymptomatic with oral combined contraceptives and had normal menstrual bleeding. An ultrasound was performed **(Fig.1)** showing hypervascularized myometrium, more exuberant on the anterior wall, and a sub-endometrial anterior lacunar vascular lesion measuring 6x4mm was identified (arterio-venous malformation (AVM) could not be excluded. The endometrium was thin, and poorly defined, with no evidence of RPOC. At this point, due to the absence of symptoms, expectant management was decided, with ultrasound reassessment scheduled.

**Figure 1 Ultrasound Revealing a Vascularized Myometrium and A Vascular Sub-Endometrial Lesion.**



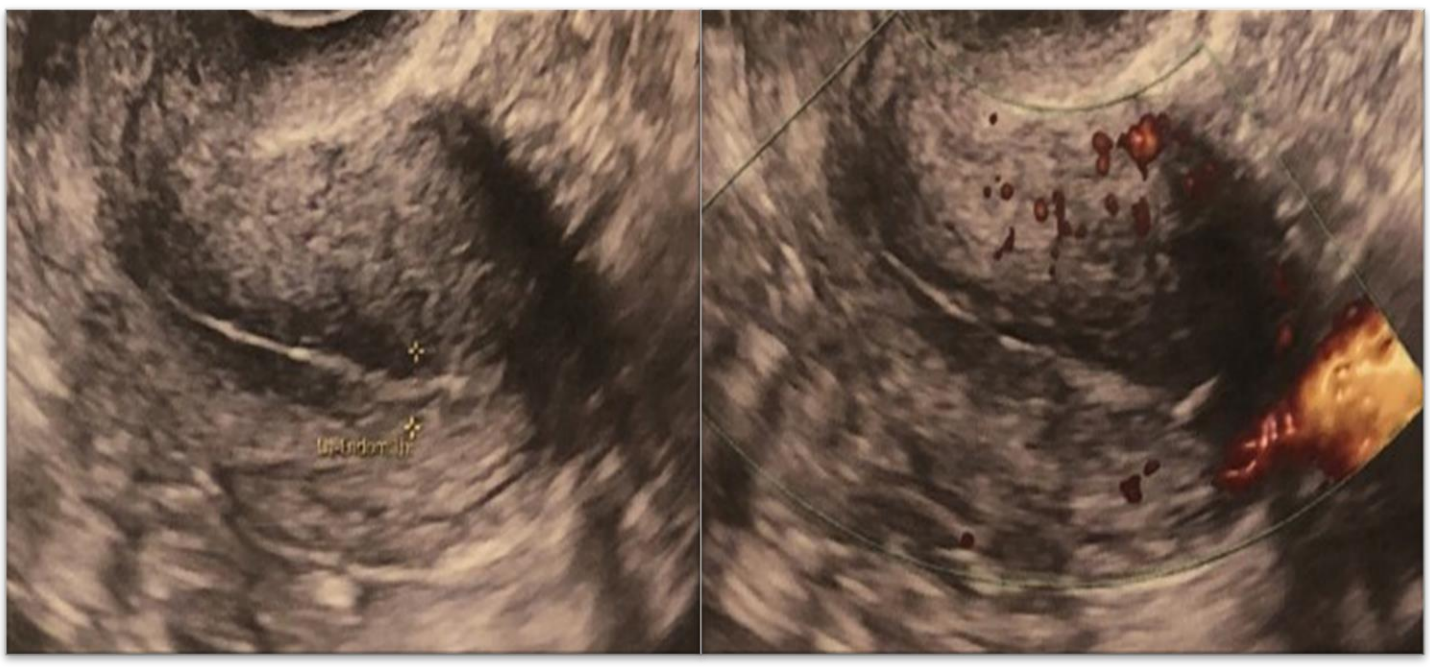
However, two weeks later the patient presented to the emergency department with a heavy uterine bleeding accompanied by the presence of clots. The patient was hemodynamically stable, with a hemoglobin level of 13.5 g/dL, normal coagulation, and a negative HCG. The patient was administered medical therapy comprising misoprostol 800 mcg and intravenous (IV) tranexamic acid, which resulted in effective control of the bleeding. Given the patient's stability, a pelvic magnetic resonance imaging (MRI) with angiography and dynamic contrast enhancement was performed. The exam revealed an endometrial cavity distended by a blood clot and a sub-endometrial 4cm irregular lesion with mild hypersignal on T1 and T2, with progressive enhancement, suggestive of a probable acquired AVM/EMV in the context of RPOC/uterine instrumentation **(Fig. 2)**.

**Figure 2 Pelvic MRI and MRI angiography with Dynamic contrast-enhanced (DCE). A) Sagittal T2WI, B) Coronal T2WI C) and D) MR angiography, DCE T1WI, show nodular heterogeneous image in anterior myometrium with is signal T2WI and vascular central signal voids (arrow); late contrast enhancement on DCE (arrowhead) and communication with an endometrial cavity. No vascular fistule or early venous filling was observed on MRI.**




The patient was discharged home with a high-dose estrogenic treatment regimen comprising a combined contraceptive pill with 30ug of estrogens, administered 6/6h for 48h, 8/8h for the next 48h, 12/12h for the following 48h, and then 1 pill/day. Additionally, oral tranexamic acid, 500mg, was administered 8/8h. At this juncture, the therapeutic options were deliberated, and a hysteroscopy was scheduled. One week later, the operative hysteroscopy was performed. It was possible to identify a 4 cm clot and an irregular material occupying the anterior and right walls and fundus, suggestive of RPOC. A resectoscopy was performed, during which the total clot and lesions were resected. Despite the use of loop coagulation, uterine massage, misoprostol 800mcg, and intravenous tranexamic acid, the hemostasis was challenging to achieve. The estimated blood loss was 600ml, with fluid intravasation amounting to 3000ml. Due to the development of postoperative anemia (hemoglobin concentration of 10 g/dL), a dose of 500 mg of ferric carboxymaltose was administered. The patient was discharged home on the same day, with instructions to take oral tranexamic acid and an oral combined contraceptive pill. One month after surgery, she was asymptomatic and the ultrasound revealed a normal uterus, with no evidence of EMV or RPOC **(Fig. 3)**. The histological examination was consistent with RPOC.

**Figure 3 Pos-Operative Normal Ultrasound**



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## Literature Review

The term "enhanced myometrial vascularity" (EMV) is used to describe the presence of transiently increased blood flow within the myometrium. It is important to note that this phenomenon does not represent a true arteriovenous malformation. Instead, it is either the result of normal peri trophoblastic flow of spiral arteries or placental bed involution/subinvolution, which results in focal areas of marked tortuous endometrial vascularity extending into the myometrium. **[2,4,6,11]**

This condition is almost exclusively seen in the context of recent pregnancy, typically secondary to retained products of conception in the early postpartum period, or following a first-trimester miscarriage or termination of pregnancy. Additionally, it may be associated with gestational trophoblastic disease or a cesarean scar pregnancy.**[2,5,8,9,11]** Less common causes of increased myometrium blood flow include uterine procedures (curettage, cesarean section, myomectomy, etc.), polyps, fibroids, endometrial or cervical carcinoma, endometritis, and endometriosis.**[5,8,10,11]**

The true incidence of EMV is unknown due to its rarity and also because the term AVM is often used interchangeably in the literature. **[2,8]**

EMV can be asymptomatic, but most of the patients present with heavy or irregular vaginal bleeding. **[8,11]**

Transvaginal ultrasound scanning with color Doppler is the primary tool of choice for the diagnosis of EMV. The ultrasonographic characteristics are nonspecific and include the presence of irregular hypoechogenic, tortuous, tubular structures within the myometrium. RPOC are frequently present. On the color Doppler there is a turbulent pattern with multiple flow reversals, which demonstrates low impedance flow with a high peak systolic velocity (PSV) ≥20 cm/s and low arterial waveform pulsatility. Although some studies consider that a high PSV (PSV >60 cm/s) confers a greater hemorrhagic risk, others have shown that PSV values do not correlate with the hemorrhagic risk. **[6,7,8,11]**

Due to the lack of specificity of the ultrasonographic findings, the clinical context is essential for the diagnosis of EMV by ultrasound.**[11]**

Digital subtraction angiography is considered the gold standard since is the only exam that can distinguish EMV from MAV. EMV appears as a hypervascular lesion without early venous filling. Nevertheless, it is seldom employed as a standalone diagnostic tool due to its invasive nature and is typically reserved for patients who require surgical intervention or embolization. **[2,6,8,11]**

Additional imaging modalities, such as MR angiography and CT, may also assist in the diagnosis. On the first, we can see a bulky uterus with a blurry mass, focal or diffuse disruption of the junctional zone, and abnormal tortuous uterine vessels.**[6,8,11]**

The management of patients with EMV is contingent upon their presenting symptoms.**[10]**

In the absence of symptoms or evidence of heavy bleeding, a conservative approach is advised, given that EMV is typically a transient phenomenon. **[2,11]** The follow-up evaluation should be done with serial β-hCG and vaginal ultrasound. Spontaneous resolution usually occurs between 1 week to 6 months. **[7,11]** Medication such as tranexamic acid, uterotonic agents (e.g. misoprostol), or gonadotropin-releasing hormone agonists may be used. Dilation and curettage (D&C) remain an acceptable alternative in the context of RPOC. There is sparse evidence supporting that D&C on patients with EMV and RPOC is associated with increased bleeding risk, except in cases of cesarean scar and molar pregnancies. **[3,6,11]**

In the event of significant or prolonged bleeding, surgical management is indicated. **[10,11]**

In patients with significant preoperative bleeding or anemia, uterine artery embolization (UAE) may be a suitable option. However, this procedure entails the use of radiation and the insertion of foreign bodies, which carries the potential risk of complications. Furthermore, it may result in a reduction in ovarian reserve and impaired fertility, although favorable reproductive outcomes have been reported. **[1,3,10]**

In the event of persistent bleeding, more invasive procedures may be required, such as hysteroscopic electrosurgery, uterine or internal iliac artery ligation, or hysterectomy as a last resort.**[11]**

## Conclusion

EMV is a rare condition associated with pregnancy complications. A high index of suspicion is essential for the diagnosis. The optimal management of patients with RPOC and EMV remains to be determined. Each patient requires individualized management based on symptoms and fertility plans. Through early and proper identification of patients with EMV, we may be able to avoid potentially morbid treatments that include transfusion, curettage, UAE, or ultimately, hysterectomy. **[6,8,10]**

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