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Review

Uterine arteriovenous malformations following gestational trophoblastic neoplasia: a systematic review

Omar Touhami [a], Jean Gregoire [a], Patricia Noel [b], Xuan Bich Trinh [a], Marie Plante [a,*]

[a] Gynecologic Oncology Division, Centre Hospitalier Universitaire de Québec L'Hôtel-Dieu de Québec, Laval University, Quebec City, QC, Canada
[b] Radiology Department, Centre Hospitalier Universitaire de Québec L'Hôtel-Dieu de Québec, Laval University, Quebec City, QC, Canada

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ABSTRACT

Uterine arteriovenous malformation (AVM) following gestational trophoblastic neoplasia (GTN) is a rare condition. It can be associated with chronic vaginal bleeding or life-threatening heavy bleeding, even after complete resolution of the tumor following chemotherapy. This analysis aimed to perform an extensive systematic review highlighting clinical symptoms, imaging, management and prognosis of this rare complication of GTN. We also describe an additional case of uterine AVM following GTN. We conducted a literature search using Medline, Embase and Cochrane library to analyze the clinical data of 49 published cases of uterine AVM following GTN. Median age of the women diagnosed with AVM was 29 years (range 15–49). Median gravidity was 2 (range 1–8) and 50% of women were nulligravida. Complete molar pregnancy was the most common initial gestational trophoblastic diagnosis (48%). Overall, 44 patients (88%) were symptomatic and presented with chronic or acute abnormal vaginal bleeding. Only 3 patients had an undetectable HCG level at the time of uterine AVM diagnosis. Hypoechoic space in the myometrium is the most relevant finding on ultrasonography but the gold standard for the definitive diagnosis of AVMs is angiographic examination. Uterine artery embolization was the most common treatment option performed in 82% of the patients and was successful in controlling the bleeding in 85% of cases. We identified 20 pregnancies after successful embolization of uterine AVM following a GTN and 90% of them were successful. Because of the risk of life-threatening heavy bleeding, the diagnosis of uterine AVM should always be considered in patients with a history of recurrent unexplained vaginal bleeding after gestational trophoblastic neoplasia. Angiographic embolization is successful in the majority of cases and does not appear to compromise future pregnancy.

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* Corresponding author at: Gynecologic Oncology Division, Centre Hospitalier Universitaire de Québec, L'Hôtel-Dieu de Québec, 11 côte du Palais, Quebec City, Quebec, Canada, G1R 2J. Tel.: +1 418 691 5392; fax: +1 418 691 5720.
E-mail address: Marie.plante@crhdq.ulaval.ca (M. Plante).

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Introduction

Arteriovenous malformation (AVM) is an abnormal connection between arteries and veins, bypassing the capillary system. There is usually proliferation of vascular channels with formation of numerous small fistulae [1]. Uterine AVM are rare and can occur as a consequence of uterine curettage, cesarean section, gestational trophoblastic neoplasia (GTN), maternal diethylstilbestrol exposure or endometrial, cervical neoplasia [2,3]. Uterine AVM can be found in patients with gestational trophoblastic neoplasia and can cause chronic vaginal bleeding or life threatening hemorrhage, even after complete resolution of the tumor following chemotherapy.

This relationship between GTN and uterine AVM could be explained by a disorganized trophoblastic proliferation, increased angiogenesis caused by high levels of human chorionic gonadotropin [4] and finally by the uterine curettage which is the cornerstone of gestational trophoblastic disease (GTD) treatment. Although ultrasound imaging with pulsed Doppler analysis and MRI are useful for the diagnosis and localization of uterine AVM [5], angiography is essential for precise delineation of feeding arteries and draining veins allowing treatment planning. Uterine AVM is a rare condition, but a potentially life-threatening one that can occur in young women who generally wish to preserve fertility, hence the importance of noninvasive treatment options that preserve fertility. In this article, we report a case of uterine AVM following GTN and we performed a systematic review of the literature on the subject.

Case report

A 28-year-old white female, gravida 1, para 0, was referred to our hospital because of gradual increase of human chorionic gonadotropin (HCG) levels and persistent bleeding 7 weeks after dilatation and evacuation of a complete molar pregnancy. The CT scan showed an endocavitary complex uterine mass vascularized measuring $7 \times 5.8 \times 5.6$ cm without evidence of lung metastasis. She was diagnosed with a persistent non-metastatic gestational trophoblastic neoplasia with a stage I tumor (FIGO anatomic staging 2000) and a WHO prognostic score of 5. She was initially treated by five cycles of actinomycin-D (1.25 mg/m^2 intravenously (IV) bolus dose every 2 weeks), with plateauing of HCG. Then seven cycles of Methotrexate (50 mg/m^2 intramuscular (IM) weekly) were given before she developed resistance. Finally she responded completely to five cycles of EMA-CO (etoposide, methotrexate, actinomycin-D, cyclophosphamide, vincristine) with three cycles of consolidation. Three months after the end of chemotherapy she developed intermittent vaginal bleeding and two episodes of vaginal hemorrhage leading to unconsciousness with a hemoglobin value of 9.0 g/dL. HCG level remained undetectable. Pelvic ultrasound was performed to further evaluate the patient's abnormal and persistent bleeding. The ultrasound showed a heterogeneous mass in the posterior myometrium with an anechoic internal focus measuring $15 \times 10 \times 15$ mm. On color Doppler, a mosaic pattern of color signals due to aliasing associated with high velocity flow was demonstrated. Pulsed Doppler analysis revealed an elevation of the peak systolic velocity (PSV = 100 cm/s) with a low resistive index (RI = 0.39), illustrating a typical high flow, low resistance, blood flow pattern (Fig. 1). MRI also revealed a heterogeneous mass comprising a tubular focus of low signal intensity on *T*1 and *T*2-weighted sequences in keeping with flow void that was enhancing as intensely as normal vessels. This focus was surrounded by non-enhancing high signal intensity areas on both *T*1 and *T*2-weighted sequences in keeping with some myometrial hematoma (Fig. 2). At that point, the diagnosis of uterine AVM was seriously suspected. The patient was counseled to undergo embolization for the management of her suspected AVM. She was advised of the risks and benefits. Given her desire for fertility preservation, she accepted the procedure. Selective right uterine artery angiography performed via the left femoral artery showed a slightly enlarged uterine artery, feeding a vascular nidus with arteriovenous communication and early venous drainage. This was followed by embolization with Gelfoam (*Gelfoam, Pharmacia & Upjohn Co., Kalamazoo, MI*) (Fig. 3). The left uterine artery was then similarly selectively catheterized and embolized. Repeat pelvic arteriogram performed at the completion of the procedure demonstrated persistent minimal filling of the arteriovenous malformation. This required a second procedure 3 weeks later with embolization of the 2 uterine arteries. Pelvic arteriogram at the end of the procedure demonstrated the absence of flow in either uterine artery and no filling of the arteriovenous malformation. The patient developed a small femoral artery pseudoaneurysm, secondary to the femoral catheterization and was successfully treated by ultrasound-guided compression. Oral hormonal contraceptives were prescribed and no recurrence of abnormal bleeding was reported after 11 months of follow-up. Cycles were regular and HCG levels remained undetectable.

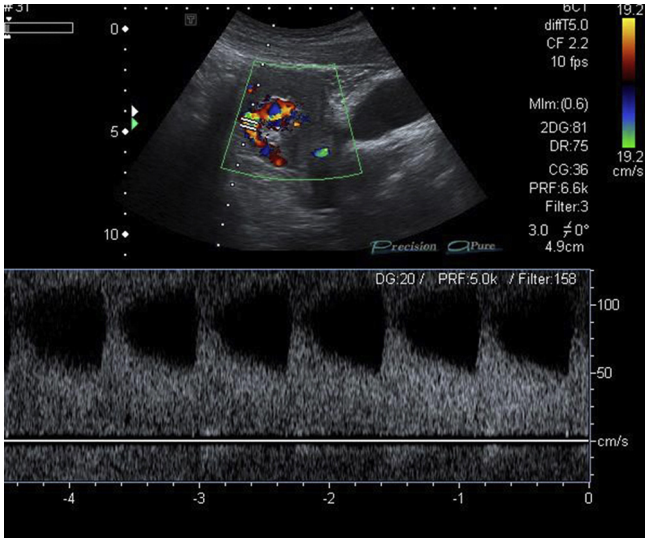

Fig. 1. Pulsed Doppler analysis illustrating a typical high flow, low resistance blood flow pattern in the posterior wall of the uterus.

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