

Uterine Arterio-Venous Malformation: A Rare Post Mortem Finding in a Young Female

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ABSTRACT

Uterine arterio-venous malformation is one of the differentials of dysfunctional uterine bleeding that can result in life-threatening emergency with sudden, unexpected massive vaginal bleeding. We describe a case of 20-year old female, who presented with sudden heavy vaginal bleeding and was diagnosed with uterine arterio-venous malformation on post-mortem examination. High index of suspicion is required to make a timely diagnosis for appropriate management and to avoid maternal morbidity and mortality.

Keywords: Uterine arterio-venous malformations, embolization, dysfunctional uterine bleeding.

INTRODUCTION

Vascular lesions of the uterus are rare, among which arterio-venous malformations (AVMs) accounts for an approximate incidence of 4.5%.¹ AVMs are abnormal growth and connection between arteries and veins without an intervening capillary bed, resulting in areas of high and low flow, which are fragile and prone to bleeding.² Uterine AVMs can be congenital or acquired. In recent years, there has been an increasing number of acquired vascular lesions following pregnancy, abortion, caesarean section, and curettage.³ So, it should be kept as one of the possibility in a patient with massive vaginal bleeding, especially in the presence of hemodynamic instability.

CASE REPORT

A hysterectomy specimen was received from the dead body of 20years old female for histopathological examination. The deceased was married and presented in emergency department with sudden massive vaginal bleeding for last 6 hours. She gave

history of light spotting from last 2 days preceding this sudden heavy bleeding. She also had irregular menses ever since a missed abortion 6 months prior. During this episode of sudden heavy vaginal bleeding, she also complained of suprapubic cramping, but no associated nausea, vomiting, dizziness, chest pain, or shortness of breath.

On presentation, the patient's vital signs showed a blood pressure of 90/60 mmHg, pulse of 120, respiratory rate of 28 and temperature of 99.7°F. Her urine pregnancy test was negative. She had normal external genitalia with no signs of trauma. Her vaginal examination revealed copious dark blood and clots, with no trauma noted. Her cervix had no lesions and clots were noted in the os. On bimanual examination, she had a closed cervical os, no cervical motion tenderness, and no adnexal tenderness.

Her laboratory studies, including electrolyte panel, coagulation studies, and thyroid function tests were unremarkable. She had haemoglobin of 8.1 g/dL and

hematocrit of 27.1%. Trans-vaginal ultrasound showed an enlarged endometrial cavity measuring 30 mm transversely. Multiple hypertrophied vessels were noted within the myometrium, at the endomyometrial junction showing low resistance arterial flow, but no active flow within the endometrial cavity suggesting a diagnosis of uterine arteriovenous malformation (AVM). Despite receiving 2 units of packed red blood cells, the patient condition kept on deteriorating with uncontrolled vaginal bleeding. The patient declined surgical intervention and within 2 Hrs of presentation patient expired. Post-mortem was done and uterus with bilateral adnexa was send to pathology department.

Grossly, we received a previously opened up uterus specimen, which measured 10x7x3 cm and weighed 150 gm. External surface was unremarkable. On cut section, cervical canal measured 3.0 cm and endometrial cavity measured 5.0 cm, both were filled with blood clot. The endomyometrial thickness was 0.4 cm and 1.6 cm respectively. There were tiny pin point

haemorrhagic areas in the myometrium. (Figure 1)

On microscopy, endometrium was in proliferative phase and myometrium showed many congested thick-walled vessels of varying calibre reaching upto serosal surface (Figure 2). Bilateral tubes and ovaries were unremarkable grossly as well as microscopically.

The histomorphological features were consistent with uterine AVM.



Figure 1: Gross photograph of hysterectomy specimen revealing pin point haemorrhagic areas in the myometrium.

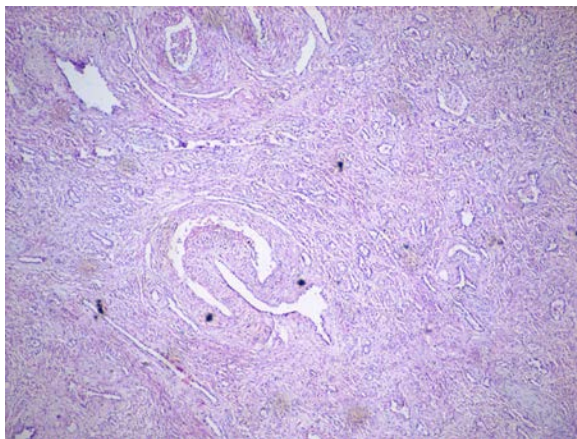


Figure 2

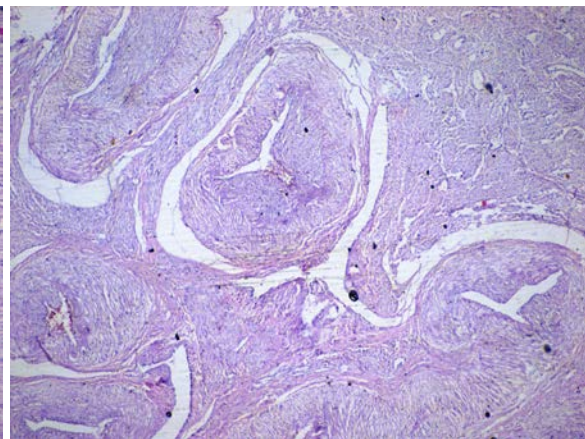


Figure 3

Figure 2 & 3: Ectatic thick-walled vessels in the myometrium (H&E 40x & 100x).

DISCUSSION

AVMs can occur in any organ in the body, including pelvic vasculature. The first case of AVM was reported in 1926.⁴ Uterine AVMs are a rare cause of uterine bleeding and less than 100 cases has been reported in the literature till date.⁵ A study by O'Brien et al showed an incidence of 4.5%, in 464

pelvic sonographic examinations performed for pelvic bleeding.¹

AVM can be congenital or acquired, each type having a distinct angiographic appearance. Congenital AVMs are very rare resulting from defect in differentiation of the primitive capillary plexus during fetal angiogenesis, creating multiple feeding vessels and draining veins

with an intervening nidus.⁶ Acquired AVMs usually have smaller arteriovenous fistulas between intramural arterial branches and the myometrial venous plexus without a nidus. These are usually traumatic, resulting from prior curettage, uterine surgery, direct uterine trauma, and less commonly from endometrial carcinoma, cervical carcinoma, gestational trophoblastic disease, choriocarcinoma, and infection.⁷

Uterine AVMs has been described in patients between 18 and 72 years of age, these patients most commonly present with sudden heavy vaginal bleeding that may even result in sudden collapse of patient. Bleeding results from spontaneous vessel rupture or vessel rupture triggered by curettage. Pregnancy and associated hormonal changes, such as elevated human chorionic gonadotropin (hCG), may play a role in the proliferation of otherwise latent AVMs. Similarly, women undergoing fertility treatments may be at higher risk secondary to elevated estrogen level, causing endothelial proliferation and differentiation of the endometrium.⁸

Earlier, the diagnosis was made after hysterectomy and histopathologic examination. Nowadays, angiography has become the gold standard for diagnosis, with the added benefit of ability to deliver treatment through embolization.⁹ Gray scale ultrasonography often shows nonspecific heterogenous or anechoic tortuous spaces in the myometrium. Color and spectral Doppler ultrasonography shows further detailing of a tangle of vessels producing a “color mosaic” pattern with multidirectional high and low velocity flow.¹⁰ Computed tomography and magnetic resonance imaging may be used to determine the size, extent, vascularity, and involvement of adjacent organs.

In the present case, AVM is of acquired type, contributing factor appeared to be missed abortion that occurred 6 months prior, preliminary diagnosis was made on trans-vaginal ultrasound. However, patient condition deteriorated rapidly with sudden collapse and the final diagnosis was

confirmed on histopathological examination on post-mortem.

Traditionally, uterine AVMs have been treated by uterine artery ligation or hysterectomy. Currently, angiographic arterial embolization is the preferred treatment because it is minimally invasive and has the potential to preserve fertility. In a patient with massive vaginal bleeding, especially in the presence of hemodynamic instability like in our case, it is important to initiate aggressive resuscitation with intravenous fluids and early use of blood products. Temporizing measures, such as intrauterine tamponade with a foley catheter, can be performed in the emergency to treat life-threatening vaginal hemorrhage.¹

CONCLUSION

Dysfunctional uterine bleeding can result in life threatening medical emergency. In patients with sudden and massive vaginal bleeding and a history of prior uterine instrumentation, the diagnosis of uterine arteriovenous malformation should be considered. Color or spectral Doppler ultrasonography should be used to confirm the diagnosis.

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