



UTERINE ARTERIOVENOUS MALFORMATION TRANSVAGINAL DOPPLER ULTRASONOGRAPHY: CASE REPORT

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ABSTRACT

Uterine arteriovenous malformation (AVM) is a rare condition, with fewer than 100 cases reported in the literature. Despite it being rare, it is a potentially life-threatening condition. This case report describes a healthy 29-year-old patient, nulligravida, with an unremarkable medical history, came from gynaecologist for ultrasound due to complain of irregular heavy PV bleeding. Transvaginal Doppler ultrasonography is a widely available, noninvasive and excellent diagnostic method. Transvaginal ultrasound (TVS) of the pelvis showed increased vascularity with multidirectional flow of the uterus and a prominent vessel, located on the posterior wall.

KEYWORDS: *Arteriovenous malformation, Primary infertility, Transvaginal ultrasound.*

INTRODUCTION

Uterine arteriovenous malformation (AVM) is a rare vascular condition, with less than 100 cases reported in the literature. It is a dilatation of the intervillous space deep inside the myometrium, allowing a direct flow from the arterial system towards the venous system, without participation of capillary vessels^[1].

Uterine AVMs may be either congenital or acquired. The congenital presentation is rarely found, resulting from abnormal embryonic development of the primitive vascular structures which determine multiple abnormal communications between arteries and veins^[2]. However, in most cases such malformation is acquired, with a great variety of causes, including gestational trophoblastic disease (GTD), pelvic trauma, surgical procedures (cesarean section, curettage), cervical or endometrial carcinoma, infection and exposure to diethylstilbestrol^[3,4]. The association of the clinical history with imaging findings is useful in the differentiation between congenital and acquired presentations.

CASE REPORT

A healthy 29-year-old patient, nulligravida, with an unremarkable medical history, previously investigated for primary infertility came from gynaecologist for complain of irregular PV bleeding to our clinic for ultrasound examination. Her Human chorionic gonadotropin hormone (β -HCG) levels were 125 mU/ml. Previous history of 3 incomplete miscarriages occurred in between 6 to 8 weeks of gestation and therefore curettage was carried out.

Transvaginal US of the pelvis showed uterus measuring 5.7 cm \times 3.9 cm \times 4.8 cm with an endometrial thickness of 8 mm. Gray-scale sonography reveals serpiginous/tubular anechoic structures within the myometrium of posterior wall of uterus (Figure 1). There was increased vascularity of the uterus with a prominent vessel seen in posterior wall. Spectral Doppler US showed a peak systolic velocity (PSV) of 51 cm/s and resistive index (RI) of 0.4 (Figure 2). The spectral Doppler wave form of arterialized venous waveform is also seen (Figure 3).

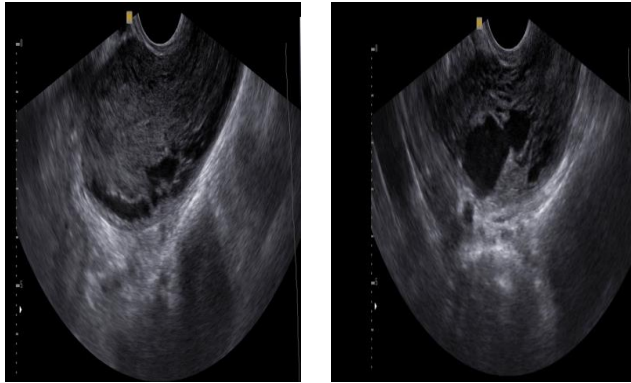


Figure 1. Gray scale transvaginal US demonstrating multiple tubular anechoic structures within the myometrium of posterior wall of uterus.

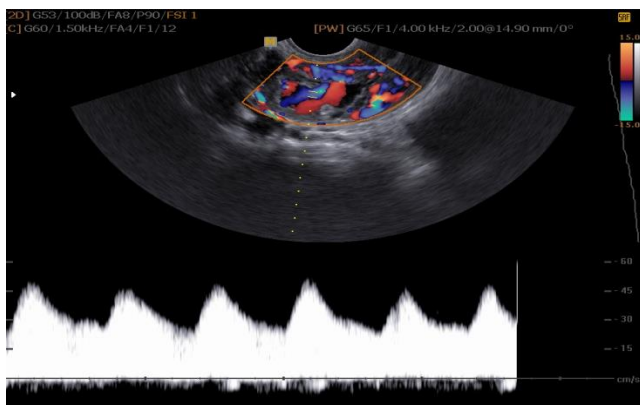


Figure 2. Spectral Doppler Transvaginal US demonstrating peak systolic velocity (PSV) of 51 cm/s and resistive index (RI) of 0.4.

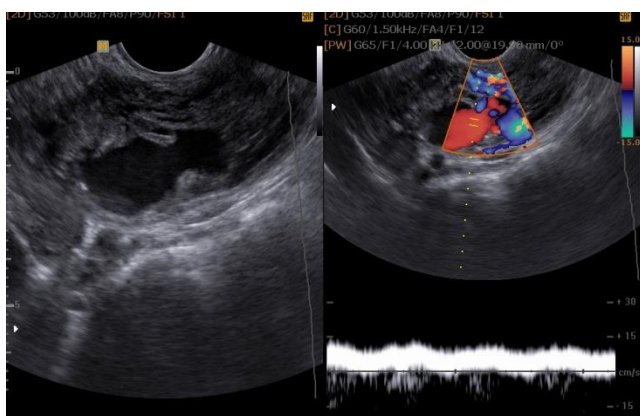


Figure 3. Spectral Doppler Transvaginal US demonstrating arterialized venous waveform.

DISCUSSION

A UAVM consists of a proliferation of vascular channels with fistula formation and an admixture of small, capillary-like channels. The size of these vessels can vary considerably. They are classified as congenital or acquired. The latter are more common and is often described as a uterine arteriovenous fistula.

Congenital UAVMs tend to have multiple feeding arteries, a central nidus (a tangle of vessels with histologic characteristics of both arteries and veins), and numerous large draining veins^[5]. **Acquired or traumatic uterine AVMs** represent multiple small arteriovenous fistulas between intramural arterial branches and the myometrial venous plexus^[5]. They typically represent a single artery joining a simple vein.

Acquired uterine AVMs are abnormal communications between intramural branches of the uterine artery and the myometrial venous plexus, deep inside the myometrium and endometrium. They may be supplied by one or both uterine arteries, without blood supply from extrauterine or interposition of a vascular plexus. Causes include curettage and GTD, and AVMs persist in 10–15% of cases of GTD in remission after chemotherapy.

Generally, such lesions occur in women at childbearing age, with either acute or chronic symptoms^[6,7]. The most common symptom is menorrhagia or metrorrhagia. Other symptoms include recurrent spontaneous miscarriages, low abdominal pain, dyspareunia and anemia secondary to blood loss. Pelvic assessment can demonstrate a pulsatile mass^[6,7]. It is believed that the bleeding occurs as the malformation vessels become exposed due to the endometrial desquamation during menstruation, or iatrogenically during dilatation and curettage^[7].

Historically, the diagnosis was made after laparotomy. Subsequently, angiography became the gold standard. Currently, transvaginal Doppler US is the most utilized method, and angiography is reserved for patients submitted to surgical treatment or therapeutic embolization^[4].

US findings include heterogeneous, ill-defined mass, with multiple, hypoechoic cystic or tubuliform structures varying in size and focal or asymmetrical endometrial and myometrial thickening. Doppler US demonstrates arteriovenous shunt with low-resistance and high-velocity flow. Spectral analysis may predict the degree of the vascular lesion arterializations and aid in the definition of the treatment^[2].

Differential diagnoses with similar sonographic findings include GTD and other hypervascular lesions such as retained conception products and abnormal placentation^[6]. Such a differentiation is critical, considering that curettage is not the appropriate therapy in cases of AVM and might exacerbate the bleeding^[7]. Stable patients may be conservatively treated, with spontaneous lesion regression. Therapeutic embolization is indicated in cases of anemic or hemodynamically unstable patients^[6,7].

CONCLUSION

Uterine AVMs are uncommon lesions, but may be cause of severe genital bleeding. Such a diagnosis should be considered in patients at childbearing age with history of uterine instrumentation or other risk factors (such as GTD) who present with abnormal genital bleeding. Doppler US is an excellent noninvasive and widely available diagnostic method, but the knowledge about this clinical entity is essential, despite its rarity.

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