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Acquired Uterine Arterovenous Malformation after Termination of Pregnancy: Sonographic-Pathologic Correlation and Description of Findings: A Case Report

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Abstract

A case of acquired uterine Artero-Venous malformation (AVM) is reported aiming at describing the correlation between findings at transvaginal sonography and pathologic examination of the uterus.

Keywords: Transvaginal ultrasound; Doppler; Arterovenous malformation; Uterus

Introduction

Uterine AVMs constitute a rare and life-threatening cause of uterine bleeding. A correct diagnosis is paramount as AVMs may be confused with a retained product of conception or a molar degeneration of the trophoblast; transvaginal sonography coupled with Doppler evaluation of the uterine vessels is useful in the differential diagnosis. We report a detailed correlation between preoperative transvaginal sonography and histopathologic examination of the uterine vascular circuit describing the structural rearrangement of the arterial and venous walls.

Case Report

A 29-years-old woman (gravida 4, para 3, aborta 1) was admitted to our Department for heavy vaginal bleeding and a negative beta-hCG value two months after being submitted to termination of pregnancy at 12 weeks of gestation. The patients had a history of three term vaginal deliveries and no other remarkable event.

Transvaginal sonography (TVS) showed the presence of a slightly enlarged uterus; in the context of the myometrium of the anterior uterine wall an inhomogeneous mass measuring 26 × 30 × 28 mm was found, in close proximity to the endometrium. Such mass had ill-defined outer borders and showed a mixture of anechoic and hypoechoic areas (Figure 1). A nest of conglomerated densely packed blood vessels were seen at power Doppler within the mass (Figure 2) with an irregular, tortuous direction. At pulsed Doppler the blood flow demonstrated a very high velocity and low resistance (PSV: 84 cm/sec, RI: 0.49), suggestive for the presence of a uterine arterovenous malformation (MAV). At TVS the endometrial stripe appeared thin (5 mm maximum thickness) on a longitudinal section. Haemoglobin level was low (9.5 gr/dl) with no other remarkable findings. Due to the persistence of the heavy vaginal bleeding and the TVS findings an urgent laparotomic hysterectomy was performed under general anesthesia. At surgery the uterus appeared slightly enlarged with normal consistence and colour. The final histologic diagnosis was acquired arterovenous malformation of the anterior uterine wall. At pathologic examination the uterus showed a normal shape, but increased volume measuring 10x8x5 cm. The endometrium was thin and finely hemorrhagic. The anterior myometrium presented a medium thickness of 3.5 cm with a prominent vascular component (Figure 3). This corresponded histologically to a convoluted; randomly distribute network of arteries and veins of intermediate diameter occupying the whole thickness of the anterior uterine wall. The arteries were mainly of muscular type, thick-walled, with a variable degree of elastic fibers disruption and focal intimal proliferation with myxoid changes (Figure 4). The veins showed a thinner wall than the arteries. The blood vessels were located within the myometrial smooth muscle with no interposed stroma. Staining for CD10, a marker of endometrial stroma, was negative. Inflammatory infiltrate was absent except for a few scattered lymphocytes.

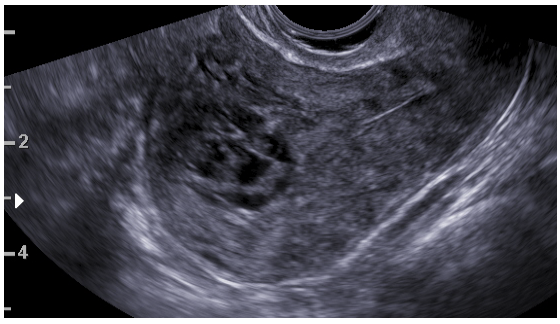

Figure 1: Transvaginal sonographic longitudinal section of the uterine corpus, showing the presence of an inhomogeneous mass located in the anterior uterine wall.

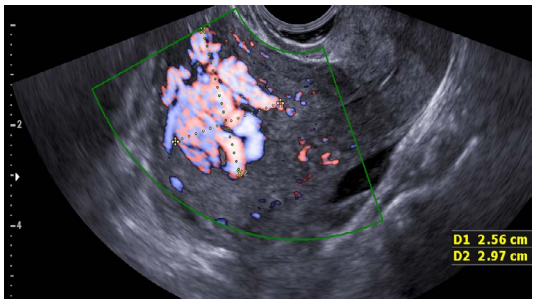

Figure 2: Transvaginal sonographic longitudinal section of the uterine corpus. Power Doppler depicts the presence of many irregularly dispersed blood vessels within the mass (callipers), creating multiple unions between arteries and veins.

Discussion

Uterine AVMs are rare and the exact incidence is yet to be

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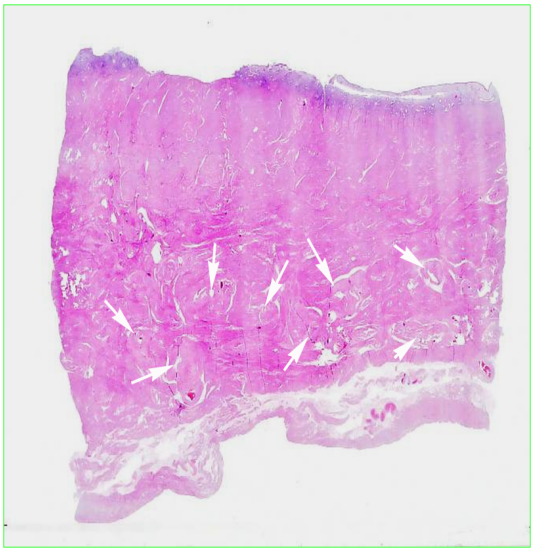

Figure 3: The uterine wall contains multiple convoluted arteries and veins of intermediate diameter. The arteries are of muscular type, thick-walled, with a variable degree of elastic fibers disruption and focal intimal proliferation with myxoid changes.

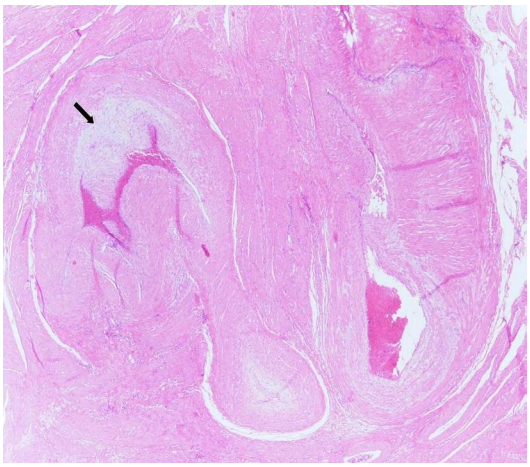

Figure 4: Histologic specimen of the uterus (Hematoxilin & Eosin, 2HPF) showing thick-walled and tortuous arteries within the myometrium with no stromal interposition. Focally, there is intimal proliferation with myxoid changes (black arrow).

determined. They can be congenital or acquired occurring after pregnancy, abortion or curettage [1]. Blood loss due to rupture of AVMs may be so heavy that a transfusion or even an urgent hysterectomy may be required.

AVM is generally defined as an entanglement of blood vessels with an abnormal connection between arteries and veins lacking a capillary bed, which normally regulates pressure and resistance [2]. This may explain the pulsed Doppler findings of high velocity in blood flow and low resistance, which favours a condition of turbulence and therefore easy bleeding. A correct diagnosis is paramount as AVMs may be confused with a retained product of conception or a molar degeneration of the trophoblast. This may lead to unnecessary and risky surgical treatment, such as uterine curettage, causing disruption of the abnormal blood vessels and massive bleeding. However, the majority of AVMs tends to be treated conservatively [3] and will disappear spontaneously in a variable length of time. Few patients, with heavy persistent bleeding, require active management and may be referred for angiography and embolization [1].

In our case, AVM occurred after termination of pregnancy and uterine curettage. Due to heavy bleeding and no desire for future pregnancies, hysterectomy was the elective treatment given the opportunity to evaluate sonographic and pathologic findings. Since AVMs' treatment is mainly conservative, detailed histopathology description has been reported in few papers only [4-6] and a clear sonographic-pathologic correlation is still lacking in the scientific literature. It is known that a nest of thick-walled arteries and dilated veins constitute the bulk of the newly-formed blood vessels. We noted in the arterial wall aspects of vascular remodelling resembling those found in hypertension: intimal proliferation, myxoid changes and elastic fibers disruption. In the other case reports, similar vascular alterations were mentioned such as: "intimal fibrosis" [5], "parietal fibrosis, intimal proliferation and elastic fibers disruption" [4] and "intimal thickening and elastic fibers fragmentation or duplication" [6]. On the other hand, these findings were not correlated to a possible damage induced by a constant high velocity blood flow. Arteries are high resistance vessels and respond to hemodynamic forces with a structural rearrangement of the wall involving the intima and the media [7]. In AVMs, further evidence of high pressure and low resistance, due to the absence of a capillary network, was that the veins were dilated with a thickened wall. This high pressure circuit is then captured by pulsed Doppler imaging. Peitsidis et al [8] extensively reviewed most of acquired AVMs after uterine diagnostic curettage. Among 29 AVMs treated with hysterectomy, histological description was present in less than 10 cases with a scarce accuracy and dubious findings. Our case provides a complete study of an acquired AVM with ultrasound and pulsed Doppler findings followed by a comprehensive pathological examination. The latter revealed the vascular effects caused by a high-pressure circuit lacking a counteracting resistance comparable to hypertensive injuries. Hence, a better understanding of AVMs pathophysiology may be helpful in choosing the correct management since imaging and pathology are closely associated. We have described such correlation with the aim of resolving the lack of diagnostic criteria in the existing pathologic and sonographic literature. Authors declare no financial relationship with any sponsoring organization; moreover they have full control of all primary data and they agree to allow the Journal to review their data if requested. Authors have no conflict of interest.

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