Radiological findings of uterine arteriovenous malformation: a case report of an unusual and life-threatening cause of abnormal vaginal bleeding

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). Such condition represents about 1–2% of all genital and intraperitoneal hemorrhages (2).

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 (3). 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 (4,5). The association of the clinical history with imaging findings is useful in the differentiation between congenital and acquired presentations.

CASE REPORT

An 18-year-old patient found out to be pregnant and, one month later, presented with abnormal genital bleeding. Ultrasonography (US) results were suggestive of GTD. The patient underwent three curettages, maintaining high beta-HCG levels (34,818.00), with diagnosis of GTD. Transvaginal US demonstrated the presence of myometrial arteriovenous fistulas and tamponade of the communication between the uterine cavity and the serosa (a sequela from perforation during a previous curettage).

Five months after the symptoms onset, the patients remained with high beta-HCG levels, so treatment with methotrexate was initiated. The patients underwent a new US whose findings were similar to those observed on the previous study (Figures 1, 2 and 3). Pelvic magnetic resonance imaging (MRI) confirmed the presence of arteriovenous fistulas (Figure 4).
A good response to monochemotherapy was observed, and the patient presented negative serum beta-HCG levels within seven months. Follow-up US after the treatment demonstrated the uterus with normal volume, presence of small amount of fluid in the uterine cavity and hematosalpinx. The arteriovenous fistulas did not exist anymore.

Three months after the negative beta-HCG results, the patient presented increased serum beta-HCG levels again and polychemotherapy was initiated, but she died because of septic complications.

DISCUSSION

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 extraterine or interpo-
position of a vascular plexus. Causes include curettage and
GTD, and AVMs persist in 10–15% of cases of GTD in re-
mission after chemotherapy.

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

Historically, the diagnosis was made after laparotomy.
Subsequently, angiography became the gold standard. Curr-
rently, transvaginal Doppler US is the most utilized method,
and angiography is reserved for patients submitted to surgi-
cal treatment or therapeutic embolization.

US findings include heterogeneous, ill-defined mass,
with multiple, hypoechoic cystic or tubuliform structures vary-
ing in size and focal or asymmetrical endometrial and myo-
metrial 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.

Although Doppler US can strongly suggest the presence
of AVM, its ability to accurately determine the lesion extent
in the pelvis may be limited. MRI is an excellent noninvasive
method to determine the disease extent and aid to confirm
the diagnosis. Findings include voluminous uterus, ill-
defined mass, focal or diffuse interruption of the junctional
zone and prominent parametrial vessels.

Differential diagnoses with similar sonographic findings
include GTD and other hypervascular lesions such as retained
conception products and abnormal placentation. Such a
differentiation is critical, considering that curettage is not
the appropriate therapy in cases of AVM and might exacer-
bate the bleeding. Stable patients may be conservatively
-treated, with spontaneous lesion regression. Therapeutic
embolization is indicated in cases of anemic or hemodynam-
cally instable patients.

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 es-
sential, despite its rarity.

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