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Seminars in
ULTRASOUND
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Uterine Arteriovenous Malformations

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Uterine arteriovenous malformations are rare but may represent a life-threatening cause of vaginal bleeding. The typical patient affected is a multiparous woman during her thirties. The origin can be congenital or acquired, with the latter being more common after uterine surgery and presenting mainly as arteriovenous fistulous connections into the myometrium supplied by uterine arteries. The correct diagnosis of uterine arteriovenous malformations requires imaging findings of tubular and tortuous structures with mixed signal from arterial and venous flows; transvaginal color-Doppler ultrasound is the initial technique applied, then integrated with contrast-enhanced magnetic resonance or computed tomography. Multiple treatment approaches are available, including conservative-medical, endovascular embolization and surgery. Transarterial embolization represents the most applied, preserving childbearing capacity with negligible procedural complications; clinical and technical success rates are elevated, up to 90%. The goal of embolization is to occlude the point of fistula or the nidus and the application of multiple embolizing agents has been reported: despite there is no clear superiority of one over the others, liquids, especially those related to the dymethil-sulfoxide family, present relevant technical advantages. Surgery is nowadays to be considered when the endovascular approach fails and in these cases hysterectomy remains the common recommendation.
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Introduction

An arteriovenous malformation (AVM) is an abnormal connection between arteries and veins, bypassing the capillary system.

They can be found anywhere in the body and according to the localization, AVMs are considered central or peripheral if inside or outside the central nervous system respectively.

Uterus is a rare peripheral localization but uterine AVM can represent a life-threatening condition because of severe hemorrhage, requiring blood transfusion in up to 30% of the cases.[1] The first case of uterine arteriovenous fistula has been described back in 1926 by Dubreuil and Loubat.[2]

They represent 1%-2% of all genital and intraperitoneal hemorrhages,[3,4] but the incidence of acquired AVM might have been gradually rising in recent years because of increase in uterine interventions (those following pregnancy, caesarean section, curettage and abortion).[5,6]

The origin can be congenital or acquired; from an anatomopathological point of view, the latter are arteriovenous fistulas and are more common than congenital lesions that on the other hand should be properly named as proper arteriovenous malformations[7]; however in the clinical practice and in literature both acquired and congenital lesions are collectively called AVMs.

Acquired AVMs are vascular abnormalities occurring after trauma, mainly surgical. Therefore an acquired uterine AVM is characterized by a single direct arteriovenous communication (fistula) between the branches of the uterine artery and the myometrial venous plexus. The most common origin of acquired uterine AVM is iatrogenic after surgery (dilation and curettage, cesarean section, myomectomy etc.), especially in postabortal patients because of hyperestrogenic state, which results in abnormal angiogenesis and vascularity. Also nonsurgical etiologies have been described, as infections, trophoblastic diseases and malignancies involving the uterus.[7]

Instead, congenital AVMs result from a defect in the differentiation of the primitive capillary plexus during fetal angiogenesis[8,9] and present a more complex angioarchitecture

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characterized by multiple arteriovenous communications with an interposing nidus and frequently involvement of extrauterine vessels.

Clinical features

The typical patient affected by a uterine AVM is a multiparous woman during her thirties, symptomatic for menometrorrhagia.[10]

Both acquired and congenital AVMs present with vaginal bleedings, meno-metrorrhagia, and pelvic pain. Pelvic varicocele can be associated as a consequence of the arteriovenous shunting.[11] The estrogens hormonal influence induces vessel proliferation and increased risk of rupture: indeed latent AVMs are frequently diagnosed during pregnancy[12] and fertility treatments.[13]

Congenital AVMs can cause symptoms at any age, especially during the second and third decades while acquired AVMs appear in childbearing age; the anamnesis of previous uterine trauma is essential in suspicion of bleedings from an acquired AVM.

Also, uterine AVM can be recognized because of dyspareunia and repeated miscarriages, due to increased vascularization that prevents embrionc implant.

Urinary symptoms as incontinence, pollachiurya and polyuria are commonly associated.[14]

Bleeding is often intermittent and torrential, consequence of the high vascular flow across the involved lesion due to the differential pressure gradient across the arterial and venous systems[7]; anemia and hypotension may be associated in case of acute bleeding, with the need for blood transfusions. Rarely, congestive heart failure has been described secondary to shunt.[15]

However, as for all AVMs, uterine AVMs can remain asymptomatic and diagnosed incidentally.

Diagnosis

The abovementioned clinical and anamnestic data lead the diagnostic route (Table 1).

Imaging and laboratory data allow to acquire the final AVM diagnosis.

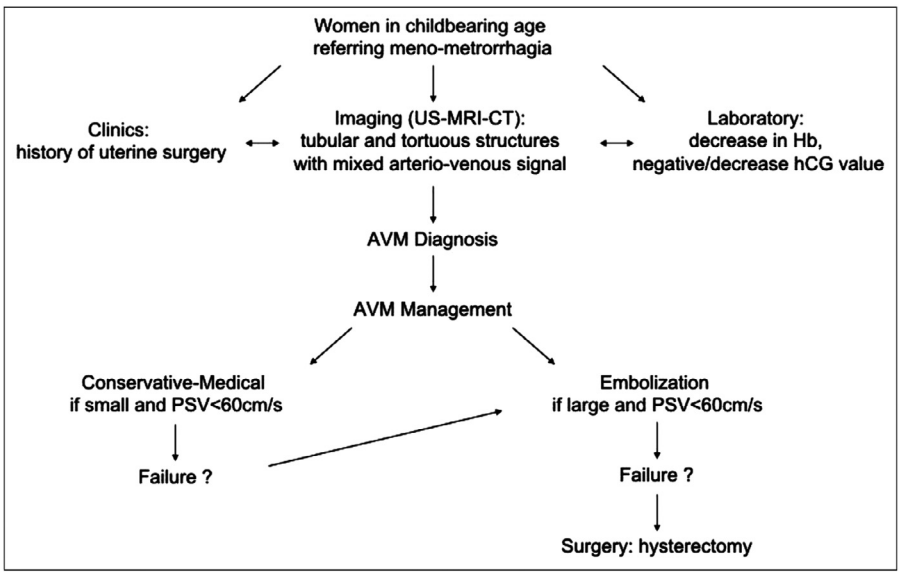
Ultrasound

The first level imaging examination is ultrasound (US); especially transvaginal color-Doppler US represents the initial approach in women suffering from vaginal bleeding because of its low invasiveness and costs as well as avoidance of contrast agents and X-rays. Typical finding is a tubular and tortuous hypoechoic structure with mixed signal at color-Doppler coming from arterial and venous flows into the myometrium (Fig. 1). Multiple studies agree in assessing that the course of symptomatic AVMs depends on the flow parameters measured on color-Doppler: expectant (when lesions are small with peak systolic value [PSV] <40 cm/s), medical (when lesions are relatively larger with PSV 40-60 cm/s), or definitive with embolization or surgery (when lesions are large and very vascular with PSV >60-70 cm/s).[9,16-18]

Magnetic resonance imaging

Contrast enhanced magnetic resonance imaging (MR) is the second step. The imaging findings are similar to US, and after contrast injection there is a clear enhancement of the lesion in the precocious data acquisitions. The panoramic view of

Table 1 Diagnostic and Management Flow-Chart of a Typical Patient With AVM



CT, computed tomography; Hb, hemoglobin; hCG, human chorionic gonadotropins; MR, magnetic resonance imaging; PSV, peak systolic value; US, ultrasound.

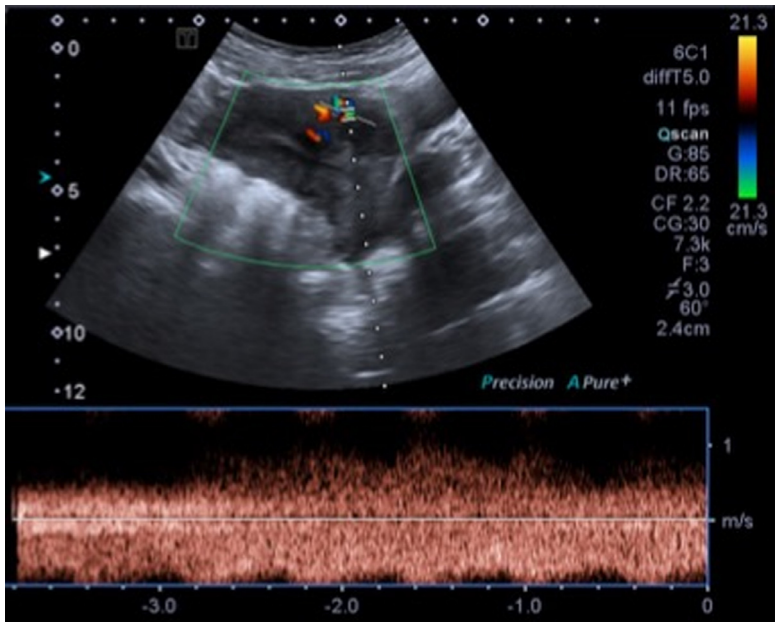


Figure 1 A 28-years-old female with menometrorrhagia a history of 1 previous cesarean section and intracavitary ablation for myoma. Transabdominal Color-Doppler US showing a focal vascular lesion into the uterine wall with mixed arterio-venous Doppler pattern.

MR allows one to identify properly the involved portion of the uterus and the vascular supply of the AVM (Figs. 2 and 3). It is of paramount importance to recognize 2 aspects at contrast-enhanced MR: an extrauterine extension of the lesion (Fig. 4) and the involvement of the endometrium. The first element suggests a congenital AVM and implies a more complex treatment strategy; the second may indicate a diagnosis of a retained product of conception rather than an AVM.[19]

Computed tomography

Contrast-enhanced Computed Tomography (CT) is usually avoided for the risks related to X-rays exposure of women in childbearing age. Imaging findings are similar to MR but there is a lower sensitivity in distinguishing the soft tissue involvement (Fig. 5).

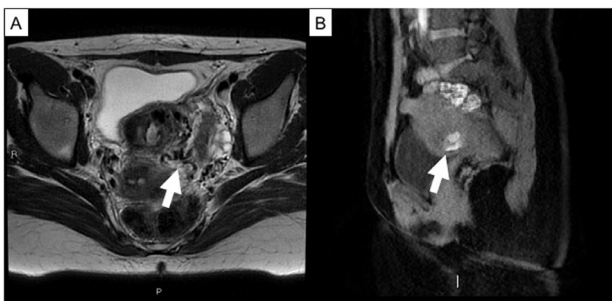


Figure 2 Same patient of Fig. 1. T2-weighted MR (A) in axial plane, white arrow showing a serpiginous vascular structure in the uterine wall; (B) Contrast-enhanced T1-weighted MR in sagittal plane acquired in precocious arterial phase showing a focal area of tubular enhancement (white arrow) corresponding to the AVM.

Angiography

The diagnostic role of angiography is nowadays related to the treatment session and demonstrates an arteriovenous fistula in the case of acquired AVM or a more complex vascular structure with arterial inflows, nidus and venous outflows in the case of congenital AVM.

Laboratory data

Relevant laboratory data, apart from blood count that may demonstrate a decrease in hemoglobin values, is the maternal serum Chorionic Gonadotropins (hCG): in case of AVM they are negative, while in case of retained products of conception they are positive and show a progressive decline.

The principal differential diagnosis should be made with retained products of conception but also with hypervascularized tumors (sarcoma) and pseudoaneurysms.

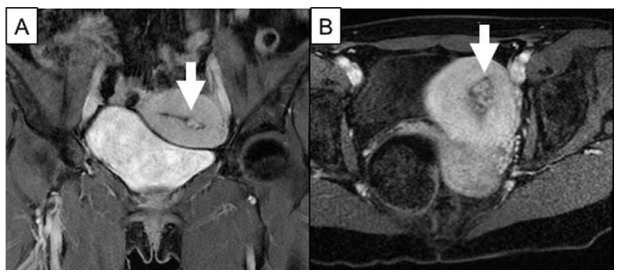


Figure 3 A 38-years-old female with menometrorrhagia and history of 2 abortions with dilation and curettage. T2-weighted MR (A) in coronal plane, white arrow showing a serpiginous vascular structure in the uterine wall and cavity (white arrow); contrast-enhanced T1-weighted MR (B) in axial plane acquired in precocious arterial phase showing a focal area of tubular enhancement (white arrow) corresponding to the acquired AVM.

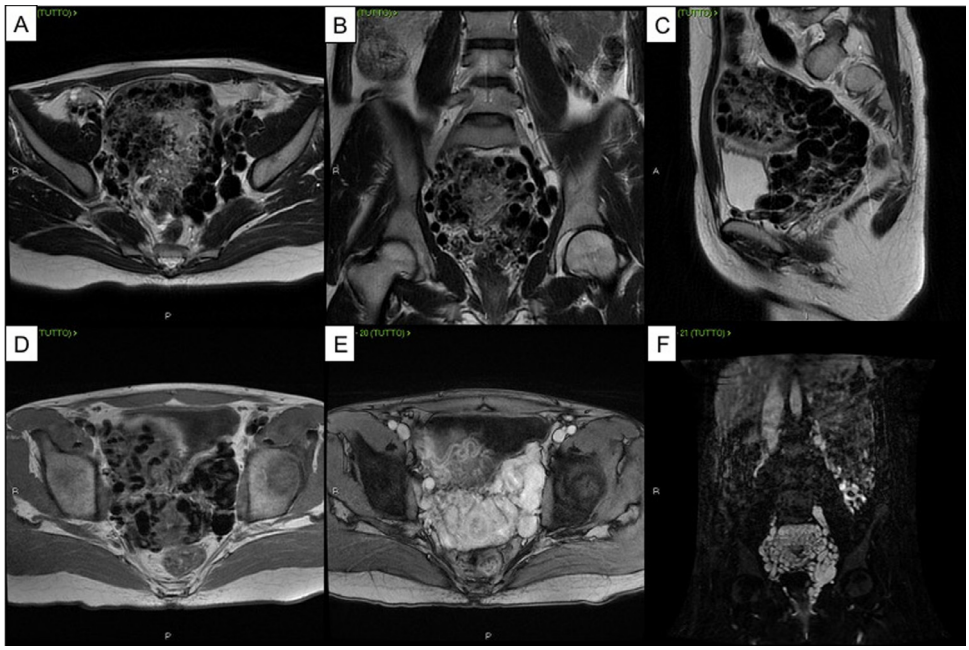


Figure 4 A 36-years-old female with menometrorrhagia and history of multiple spontaneous miscarriages. Preprocedural MRI showing a complex congenital AVM with uterine and extrauterine feeders. The vessel proliferation has completely subverted the uterine structure. T2-weighted sequence in axial (A), coronal (B) and sagittal (C) planes; T1-weighted sequence in axial plane (D); contrast-enhanced T1-weighted sequence in axial plane(E); TRICKS sub sequence (F).

The main issue in case of AVM misdiagnosis with retained product of conceptions is that dilation and curettage (D&C) may be performed. This actually would complicate the AVM angioarchitecture by creating new fistulous connections without any clinical improvements. Indeed, a history of D&C is a common finding in these patients.[7,20]

The correct instrumental diagnosis of uterine AVM is based on negative hCG test associated with imaging of abnormal uterine vascular network (high flow tortuous and serpiginous vessels)[11] in women in childbearing age suffering from meno-metrorrhagia.

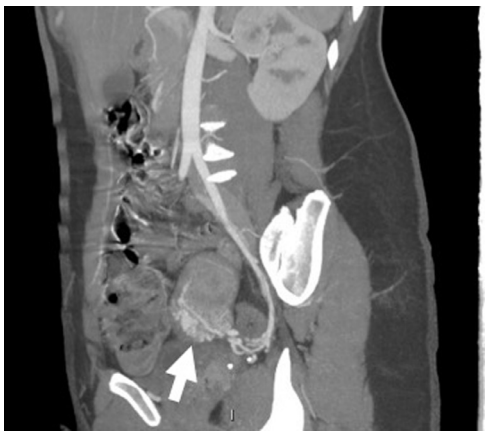


Figure 5 Same patient of Figs. 1 and 2. Contrast-enhanced CT scan in arterial phase with maximum intensity projection and oblique recontruction. The acquired AVM is indicated by the white arrow into the uterine wall supplied by uterine arteries.

Treatment

Different treatment approaches are applied in the management of uterine AVMs, including conservative-medical, endovascular embolization and surgery (Table 1).

Conservative-medical treatment

The asymptomatic lesions may regress spontaneously[10,21] and medical treatment can be adopted when lesions are small with peak systolic value (PSV) <60 cm/s.

Various medications have been tried, including methotrexate, and misoprostol, methylergometrine maleate and GnRH agonists.[16,22-24]

Recently oral norethisterone (a progesterone derivative) has demonstrated to be effective in bleeding resolution in around 90% cases of postabortal patients[18]; in this study performed on 30 patients with mean age 31 years, 17 (56.7%) had complete resolution of symptoms after a single 3-week course of progesterone therapy while 13 (43.3%) remained symptomatic and required another 3-week course of norethisterone. However, 3 out of 13 women remained symptomatic after 2 months and underwent to embolization; interestingly, all these 3 patients had a PSV measured at transvaginal US color-Doppler >83 cm/s, confirming that for large lesions with high-flow operative management is required.

Endovascular treatment

Since the first reported case of transcatheter uterine artery embolization for a uterine AV fistula in 1982,[25] embolotherapy has been considered during the years an alternative to

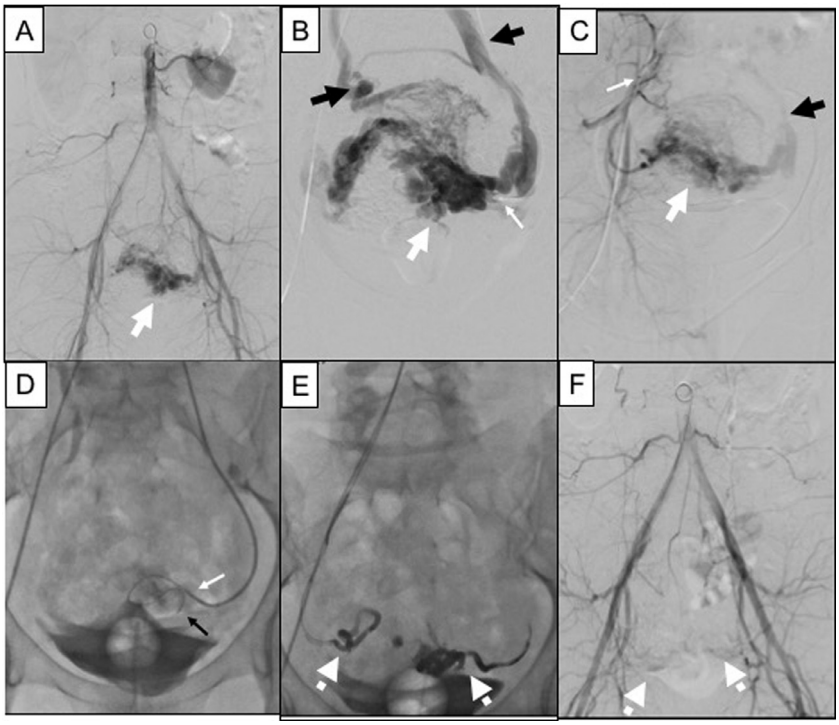


Figure 6 Same patient of Figs. 1, 2, and 5. Transarterial AVM Embolization. (A) Pelvic arteriography showing a precocious irregular vascularization of the uterus (white arrow) supplied by both uterine arteries. (B-C) Delayed arterial phases acquired through injection from the left (B) and right (C) uterine arteries (white thin arrow indicating the diagnostic catheter tip) showing the AVM (white arrows) and the venous drainage from both hypogastric veins (black arrows). (D) Fluoroscopy showing the embolization phase of the left uterine artery: the diagnostic 5 French catheter is positioned into the horizontal portion of the uterine artery (white thin arrow) and a 2.7 French microcatheter is advanced distally into the AVM nidus (black thin arrow). (E) Fluoroscopy showing the cast of the liquid embolic agent (Onyx 18, Medtronic-USA) (white dotted arrows). (F) Pelvic arteriography showing complete exclusion of the AVM from the vascular flow (white dotted arrows).

surgical intervention for uterine AVMs, with the major advantage of maintaining childbearing capacity. Nowadays transarterial embolization is the most applied approach to treat uterine AVMs[10] when lesions are large and very vascular with PSV > 60-70 cm/s.

As for all AVMs, the goal of the embolization is to occlude the point of fistula or the nidus. In the case of partial clinical improvement, the procedure can be repeated. The overall clinical and technical success rates are elevated, between 71% and 93%[7,26-28] with some patients requiring multiple embolization sessions to achieve complete success. In case of proximal embolization, the treatment is ineffective and recurrence should be expected despite an initial clinical improvement.

Preprocedural planning with contrast-enhanced MR or CT is mandatory in order to distinguish the AVM arterial feeders and venous drainages: maximum intensity projection and multiplanar acquisitions/reconstructions are useful tools. Arterial and precox venous phases should be carefully analyzed.

In case of an acquired AVM (Figs. 6-8), the lesion is usually supplied by one or both uterine arteries. During the initial diagnostic arteriography, a panoramic aorto-iliac arteriogram can confirm MR/CT findings and depict any additional small feeders. Both anterior hypogastric trunks and uterine arteries have to be investigated selectively. Patients are often young women and arterial spasms may happen that obstruct distal catheterization Therefore, gentle manouvres and microcatheters should be employed to navigate in uterine arteries up to the AVM. Venous drainage is usually in the hypogastric veins.

In case of congenital AVM (Figs. 9-11), the lesion presents a much more complex angioarchitecture, technically demanding; multiple arterial feeders are expected, coming from pelvic and extrapelvic districts (ovarian, lumbar, mesenteric,

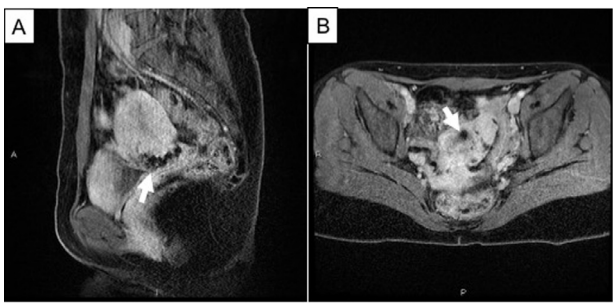


Figure 7 Same patient of Figs. 1, 2, 5, and 6. Postembolization MR acquired after 3 months. Contrast-enhanced T1-weighted MR in sagittal (A) and axial (B) planes showing enhancement of the uterus without opacification of the AVM represented by a dark serpiginous cast (white arrows).

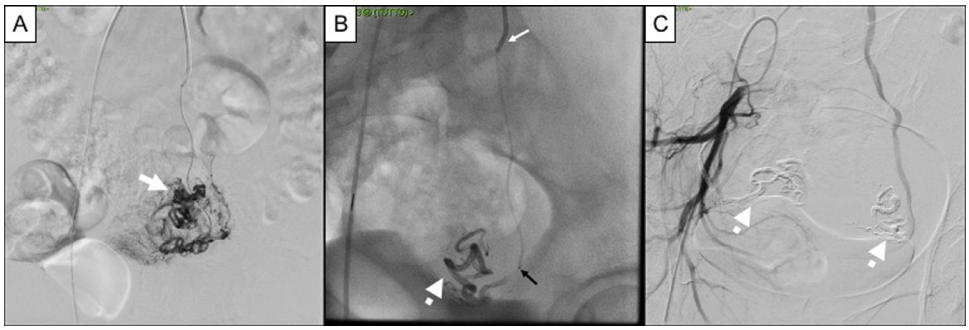


Figure 8 Same patient of Fig. 3. Transarterial AVM Embolization. (A) Superselective arteriography showing the irregular tubular structure of the AVM (white arrow). (B) Fluoroscopy showing the embolization phase of the left uterine artery: the diagnostic 5 French catheter is positioned into the proximal vertical segment of the uterine artery (white thin arrow) and a 2.7 French microcatheter is advanced distally into the AVM nidus (black thin arrow); the cast of the liquid embolic agent (Onyx 18, Medtronic-USA) (white dotted arrows) is evident. (C) Right hypogastric arteriography showing exclusion of the AVM from the vascular flow; the Onyx casts are appreciable (white arrows).

epigastric etc.); apart from the aorto-iliac examination, here an abdominal aortogram should be acquired additionally. Also multiple venous drainages have to be considered. More treatment sessions are expected because the possibility of recurrence/residual disease is high. In complex scenarios, the goal of the treatment may be only clinical improvement rather than complete vascular exclusion of the lesion.

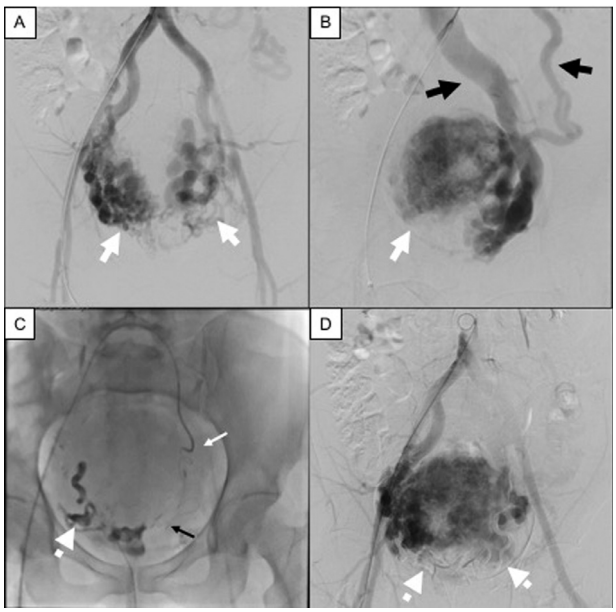


Figure 9 Same patient of Fig. 4. Transarterial AVM Embolization. (A) Precocious phase of the pelvic arteriography showing the irregular tubular structure of the AVM (white arrow) supplied by both hypogastric arteries. (B) Delayed phase of the pelvic arteriography showing the AVM (white arrow) and the venous drainage from the hypogastric and ovarian veins (black arrows). (C) Fluoroscopy showing the embolization phase of the left uterine artery: the diagnostic 5 French catheter is positioned into the middle vertical segment of the uterine artery (white thin arrow) and a 1.5 French microcatheter is advanced distally into the AVM nidus (black thin arrow); the cast of the liquid embolic agents (Onyx 18 and 34, Medtronic-USA) (white dotted arrow) is evident. (D) Pelvic arteriography showing partial exclusion of the AVM from the vascular flow in the inferior portion, the Onyx casts are appreciable (white arrows).

Different classification systems[29-31] have been proposed in the literature focusing on morphology[29,32] and clinical impact[30]; however, confidence of operators in each unique angiographic AVM architecture is essential in planning the proper endovascular strategy, considering also that the angioarchitecture can evolve while staging sessions of embolization.[33]

Multiple embolizing agents have been described, used alone or in association. There is no consensus as to the type of embolization technique and the type of embolic agent. In the first reports, coils, microspheres and gelatin sponge were the agents adopted.[7] However liquids should be preferred over metallic and particulated agents. Metallic agents produce a too proximal occlusion with the inability to completely embolize the nidus.

The particulated agents carry an elevated risk of migration into the venous district with consequent pulmonary and paradoxical embolizations, apart from the possibility of recanalization in case of reabsorbable elements. Several types of liquids agents are available and there is no demonstrated superiority of one over the others: since their recent introduction, the dymethil-sulfoxide family agents are generally preferred.[11,26,33,34] These derive from neurointerventions and, compared to glues, present some relevant advantages. The operators can handle the injection phase for longer times without the risk of microcatheter entrapment and the embolic liquid advances into the vessel by the manual injection and is not carried by the vascular flow. Compared to sclerosants and alcohol, full visibility of embolics penetration into the nidus is an important factor. Therefore nontarget embolizations are reduced.

Some authors[35] have proposed also a retrograde transvenous approach to the AVM because of the extremely tortuous arterial feeders: in this case balloon catheters were inserted into the draining vein to stop the outflow and then a sclerosant was retrogradely injected through the catheter into the nidus with successful obliteration.

As for all embolization procedures, postprocedural pain, fatigue, nausea and fever (postembolization syndrome) may occur in the first 48-72 hours, manageable with dedicated medical therapy: this should be considered as a sequela rather than a complication.

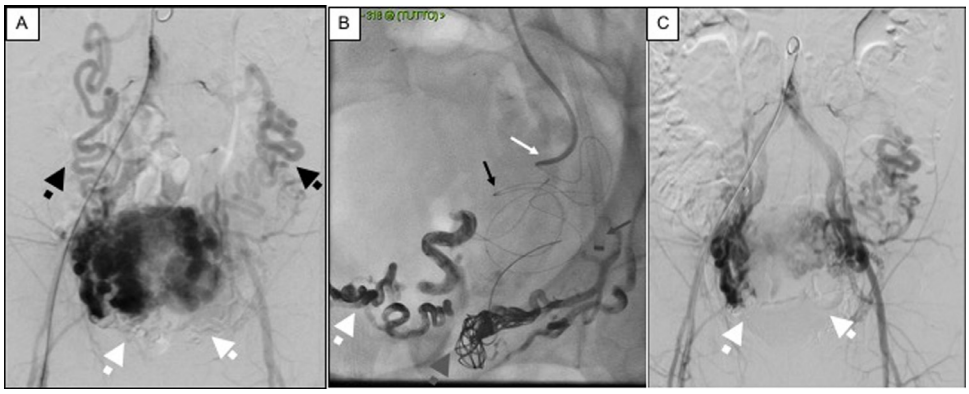


Figure 10 Same patient of Figs. 4 and 9. Second embolization session after 3 months. Transarterial AVM Embolization. (A) Precocious phase of the abdomino-pelvic arteriography showing the irregular tubular structure of the AVM supplied by both hypogastric arteries and ovarian arteries also (black dotted arrows); the Onyx cast of the previous embolization session is evident (white dotted arrows). (B) Fluoroscopy showing the embolization phase of left pudendal feeders: the diagnostic 5 French catheter is positioned into the anterior trunk of the left hypogastric artery and a 1.5 French microcatheter is advanced distally into the AVM nidus (black thin arrow); the cast of the liquid embolic agents (Onyx 18 and 34, Medtronic-USA) (white dotted arrow) is evident; a transvenous embolization approach was performed as well with a long introducer into the left hypogastric vein (grey thin arrow) and release of coils (grey dotted arrow). (C) Pelvic arteriography showing partial exclusion of the AVM from the vascular flow in the middle and inferior portion; the Onyx casts are appreciable (white arrows).

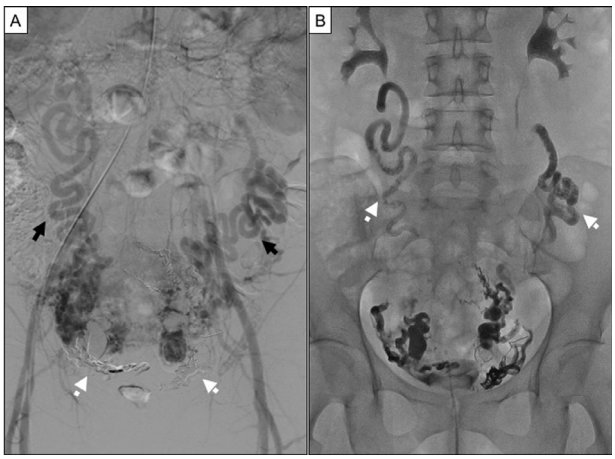


Figure 11 Same patient of Figs. 4, 9, and 10. Third session of embolization after 9 months from the previous. (A) The Onyx casts of the previous embolization sessions are evident (white dotted arrows). AVM is mainly supplied by the ovarian arteries (black arrows). (B) Fluoroscopy showing embolization of the both ovarian arteries with Phil 25 Microvention-USA (white arrows). The ovarian arteries have been embolized as last option to reduce the risk of hormonal impairment. After this procedure the patient referred significant clinical improvement and so it was decided an observational approach thereafter until eventual worsening.

The overall rate of procedural complications reported in literature after embolization is negligible.[10] Concerning obstetrical outcomes after uterine artery embolization, it is unclear which is the real effect of the procedure. Multiple papers have described successful pregnancies[10] concluding that uncomplicated pregnancies are achievable after AVM embolization.[36] Even the risk of ovarian failure because of not target embolization is low thanks to the abovementioned embolics managed by trained operators. The risk of uterine necrosis should be considered after definitive uterine embolization, as for myomas embolization; however, the vascularization is usually supplied by the wide uterine collaterals network, as ovarian, spinal, epigastric, and hypogastric arterial branches[37,38] and no cases of necrosis after embolization for uterine AVM are reported.

Clinical and radiological postprocedural follow-up is recommended, almost up to 3 months especially with congenital AVM where the risk of relapse is elevated.[11] Radiological follow-up must be performed with radiations free techniques.

Finally, special care needs to be paid to radioprotection; the guidelines for angiography in women in childbearing age should be applied.[39]

Surgical treatment

Prior to embolization, hysterectomy or uni/bilateral uterine artery ligation were the therapies of choice.

Today surgery is considered in case the endovascular approach fail such as when the previously embolized vessels recanalize or when collateral vessels develop despite multiple embolization sessions.

In these situations, hysterectomy remains the common recommendation.[40]

Some surgical fertility preserving approaches have been also reported in literature, in form of case reports:[28,39,41] they consisted mainly in laparoscopic ligation of the uterine arteries, in some cases associated with electrosurgical resection of the AVM bed.

Summary

Uterine AVMs are responsible of severe vaginal bleeding, occurring in women in childbearing age.

They are mainly acquired lesions after uterine surgery presenting as arterio-venous fistula limited to the myometrium.

Final diagnosis may be delayed, because vaginal bleedings in young women can have multiple differential etiologies: US, MR and CT imaging coupled with laboratory data are essential elements.

Today endovascular embolization represents the gold standard of treatment, able to obtain elevated success rates up to 90%; liquid embolics allow to reach the nidus distally, main goal of the therapy.

Surgery should be considered when embolization fails: in these cases, hysterectomy remains the common recommendation even if some surgical preserving approaches are developing.

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