Uterine venous malformations in the puerperium: 2 Atypical cases and literature review

Gianluca Raffaello Damiani [a,*], Miriam Dellino [a], Eliano Cascardi [b,c,**], He Xuamin [a,d], Daniele Di Gennaro [a], Antonella Vimercati [a], Amerigo Vitagliano [a], Antonio Malvasi [a], Vera loizzi [g,h], Cristiana Paniga [e], Laura Lanteri [e], Raffaello Alfonso [a], Ettore Cicinelli [a], Antonio Pellegrino [f]

[a] Obstetrics and Gynecology Unit, Department of Biomedical Sciences and Human Oncology, University of "Aldo Moro", 70124 Bari, Italy
[b] Department of Medical Sciences, University of Turin, 10124 Turin, Italy
[c] Pathology Unit, FPO-IRCCS Candiolo Cancer Institute, 10060 Candiolo, Italy
[d] San Raffaele Hospital, Milan,Italy
[e] University of Milan-Biccocca,Department of Medicine and Surgery, Milan, Italy
[f] Department of Obstetrics and Gynecology, ASTT LECCO, Alessandro Manzoni Hospital, Dell'Eremo Street 11, Lecco, Italy
[g] Department of Interdisciplinary Medicine (DIM), University of Bari "Aldo Moro", 70124 Bari, Italy
[h] Oncology Unit IRCSS Istituto Tumori "Giovanni Paolo II", 70124 Bari, Italy

A B S T R A C T

Uterine arteriovenous malformations (AVMs) is a rare but high-risk cause of uterine bleeding. The clinical management of this condition is challenging, as the ultrasound picture can sometimes be unambiguously interpreted. Moreover, in the puerperium in which acquired AVMs are most frequently formed, it is necessary to discuss the correct management in a multidisciplinary and personalized manner. We present two cases of AVMs developing in the puerperium, both with a vaginal delivery and spontaneous and complete secondment. The symptom of onset was an episode of bright red blood loss in the puerperium, on the 14th and 21st postpartum days, respectively. Transvaginal ultrasound showed a hypervascularized lesion in the myometrium with turbulent vascular flow, confirmed by transabdominal ultrasound and angiography. To date, there are no guidelines on the management of MAVs. In our cases we opted for a conservative approach, in order to preserve the fertility of the patient. These experiences reported have the purpose of enriching a literature still sparse on the subject and in the future to be able to represent a fulcrum for official recommendations.

1. Introduction

AVMs (uterine venous malformations) are rare and sometimes-misunderstood causes of abnormal uterine bleeding that can potentially be life-threatening. This pathology can be congenital or acquired and consists of an abnormal passage of blood in which the arteries, often hypertrophied, converge in one or more discharge veins, skipping the capillary bed (which physiologically reduces pressure) and creating vascular fistulas within the myometrium. For this reason, venous secretions are subjected to an abnormal pressure regime with all the consequences of the case [1,2]. The word malformation suggests a lesion that has always been present since birth; however these lesions are more commonly acquired. At the etiological basis of congenital AVMs there may be an endothelial cell anomaly. Acquired AVMs, on the other hand, are usually associated with previous pregnancies especially with a caesarean section or intrauterine curettage and less frequently after pelvic surgery such as following myomectomy. Many assumptions have been made to explain this. It seems that some procedures, such as dilation and curettage, stimulate reactive angiogenesis [1–3]. In fact, high levels of human chorionic gonadotropin or a hyperestrogenic state also stimulate angiogenesis and vascularization, increasing the risk of this condition [2]. Other theories suggest the increased immune response, which occurs after surgery, plays a key role in abnormal angiogenesis [4]. An incidence of AVMs of 0.5% is estimated, however

this figure may be underestimated due to the difficulty in clearly identifying the disease. In fact, most AVMs do not cause any symptoms, and diagnosis often occurs after the appearance of abnormal uterine bleeding or incidental detection on an ultrasound diagnosis. However, AVMs have been linked to a number of clinical consequences, including iron-deficiency anemias, infertility, miscarriage and complications related to childbirth. Therefore, the MAV should be the subject of multidisciplinary investigations, in consideration of the difficulty of identification and the absence to date of a consensus on the management methods.

2. Materials and methods

We report two cases of AVMs, one of them treated with two repeated embolization procedures. The research was conducted for significant studies exploring the electronic databases PubMed, ScopuS, Medline, Cochrane library, clinicaltrial.gov embase, and was performed independently by two researchers (Fig. 1).

The inclusion criteria were as follows: studies referring to acquired arteriovenous malformations of the uterus in the puerperium. Studies with congenital iatrogenic AVMs were excluded from the review. Studies reporting the diagnosis of gestational trophoblastic disease have also been excluded. In addition, we excluded studies reporting physician-induced abortions and AVMs in anatomical regions other than

the uterus. The most important aspect of this entity is the correct diagnosis, due to the real possibility of making a wrong diagnosis, also in the puerperium, in which various entities such as placental polyps, placental residues, or atony of the uterus could lead to a misdiagnosis and lead to complicated consequences for patients and doctors. We focused our review on the ultrasound aspect, due to the confusion of terms and definitions in the literature. Titles and abstracts were reviewed to identify relevant articles. Two authors reviewed all abstracts independently for eligibility, assessed the risk of bias, and extracted the data. Reviewers discussed the inconsistencies and a consensus was reached among the authors. We used combinations of the medical subject header terms (MeSH), keywords, and relevant word variants such as uterine fistula.

For MAV acquired in the puerperium we found 21 articles of which about ten were included in our review. Only a systematic review was found as well as 2 overviews of the narrative. The remaining studies were 2 retrospective; one with small patient sample sizes and the other a case series. A total of 83 patients with puerperium-acquired AVMs were found in our review. We informed all patients about the use of their personal data for scientific purposes, under the protection of the privacy law, and they accepted and signed a related informed consent. Considering that data analyzed in this study were collected during routine clinical activity and fully anonymized, a formal Institutional Review Board approval was exempted. The study was not advertised, and no remuneration was offered to the patients to enter or continue the study.
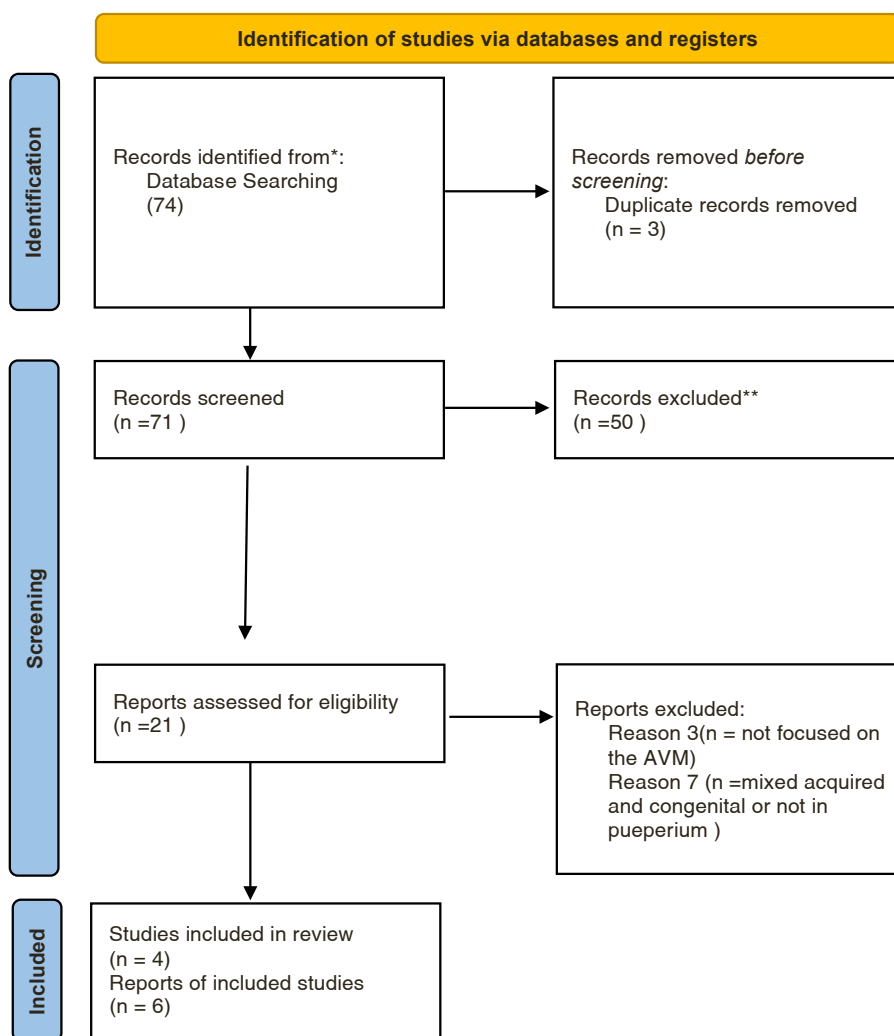

Fig. 1. a PRISMA flow diagram, which include searches of databases and registers only. Legend: *Consider, if feasible to do so, reporting the number of records identified from each database or register searched (rather than the total number across all databases/registers); * *If automation tools were used, indicate how many records were excluded by a human and how many were excluded by automation tools.

An independent data safety and monitoring committee evaluated the ad interim and final results of this study.

3. Case presentations

3.1. Case 1

Case 1. was a 28-year-old Caucasian woman, on her third pregnancy, whose current pregnancy had a regular course. In her medical history there was a family history of arteriovenous malformations and a personal history of recurrent episodes of headaches with a diagnosis of a suspected cerebral arteriovenous malformation. Her obstetric history showed a miscarriage followed by dilation and curettage 2 years earlier, and a full-term eutocic birth after the threat of a premature birth at 23 gestational weeks a year earlier. During pregnancy, first-trimester ultrasound was performed with redating of pregnancy. No prenatal diagnosis was performed. Serial ultrasound check-ups were performed, which were found to be regular, for the previous obstetric history. A vaginal rectal swab was negative. MRI angiography was performed and a regular cerebral vascular tree and internal carotid ectasia demonstrated no contraindications to vaginal delivery. At 39 weeks' gestation the patient went to the emergency room for contractile uterine activity; At evaluation, a complete dilation was found for which the patient was transferred to the delivery room. There was a spontaneous delivery and a spontaneous and complete placenta; Estimated blood loss was 300 ml; uterotonic prophylaxis was performed at the time of delivery. A male newborn weighing 3440 g was born with apgar I 10, apgar V 10 and arterial pH 7.39. Two hours after delivery, there was uterine atony treated with uterine massage and uterotonic therapy with Methlergometrin, Tranexamino acid and Syntocinon. Total blood loss was 1050 ml. The remaining puerperal course was regular; Exclusive breastfeeding was initiated and she was discharged on the 2nd postpartum day with hemoglobin of 9 g/dL and oral martial therapy. Day 13 of postpartum, the patient presented to our hospital for moderate blood loss at home. Blood tests showed Hb 10 g / Dl and beta-HCG 46 mUI /Ml. Transvaginal ultrasound showed a linear uterine cavity; at the middle third of the uterus, there was a doubtful rounded formation of $28\times15\times25$ mm, slightly vascularized by the color Doppler (Fig. 2), which seemed to deepen in the middle of the myometrium with the suspicion of AVMs.

An abdominal ultrasound was performed showing a focal alteration of about 3 cm characterized by serpiginous vascular structures with predominant venous flow. It was located on the posterior side of the uterine fundus (Fig. 2), in the context of the deepest portion of the myometrium, near the endometrial thickness without evident intraluminal involvement. This formation had afferent vascular origin from the left uterine vessels, compatible with AVMs. On the 15th day of postpartum an angiography was performed that highlighted the ectasia of the left uterine artery that also presented another irregularity. The presence of short stenosis at the origin was noted, after which a coarse branch originated with an extremely serpiginous course that mainly provided to an arteriovenous vascula or alteration of the uterus. Selective embolization of the left uterine artery with fragmented Spongostan and subsequent embolization of arteriovenous malformation with metal coils were performed (Fig. 3).

A post-embolization ultrasound check showed the absence of branches afferent to the vascular alteration that originated from the left uterine artery and the presence of a normal right uterine artery. The patient was discharged in good general condition on the 2nd day after embolization (Hb 10.4 g / dL). On the 25th day of postpartum, she went
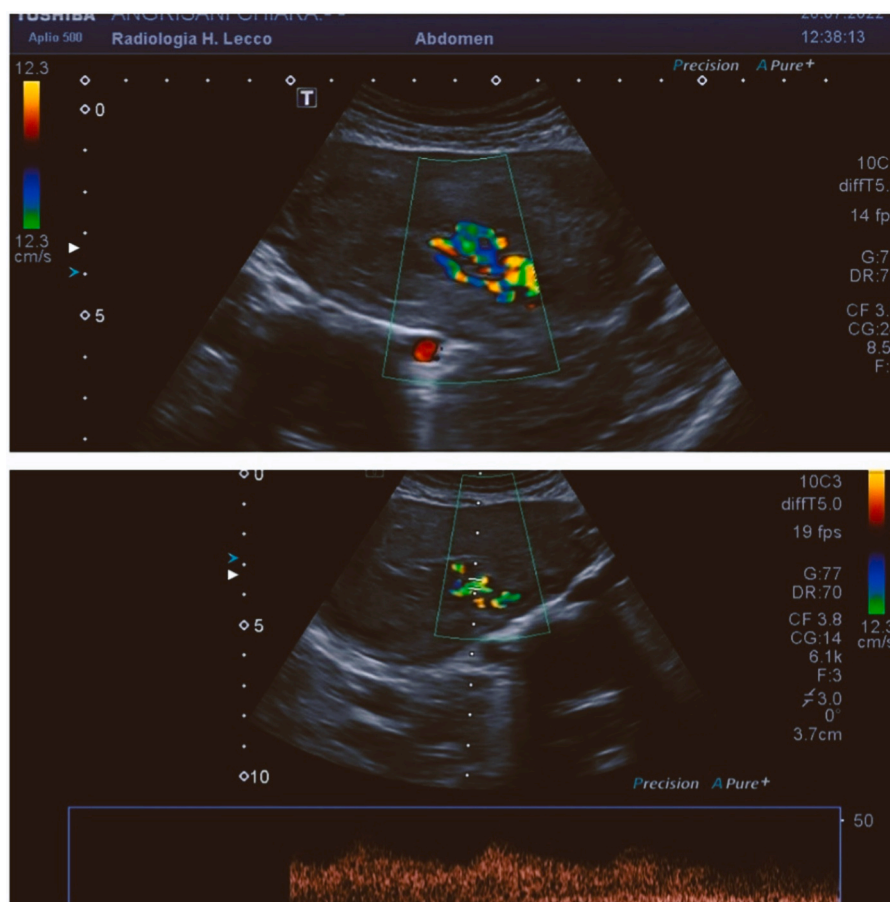

Fig. 2. Abdominal ultrasound: focal alteration about 3 cm characterized by serpiginous vascular structures with predominant venous flow.
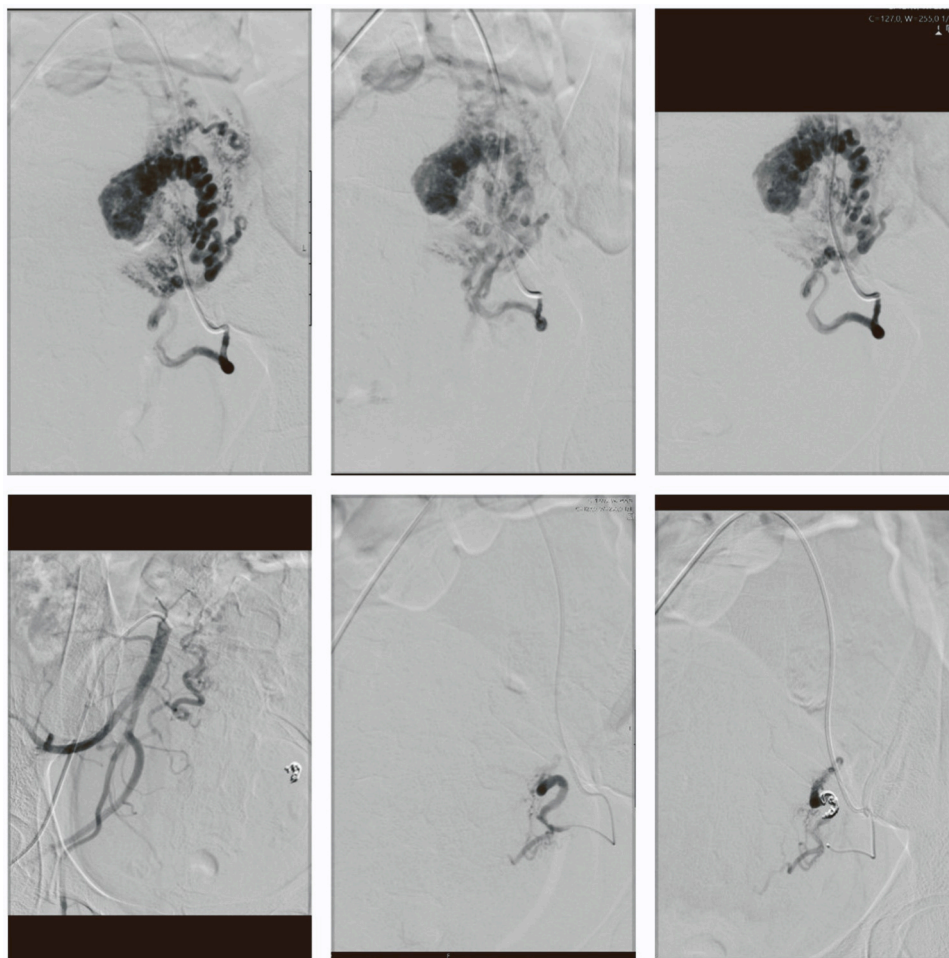
Fig. 3. Angiography: highlighted ectasia of the left uterine artery which also had another irregularity such as the presence of short stenosis at the origin, so selective embolization of the left uterine artery with fragmented Spongostan and subsequent embolization of arteriovenous malformation with metal coils were performed.

to the emergency room for a new episode of bleeding. Blood tests showed Hb 11.9 g/dL. At the level of the third center of the uterine cavity, transvaginal ultrasound showed an uneven endometrial thickening of 20 mm with vascularization originating from the left side of the uterus with a vessel crossing the myometrial thickness. Therefore, the patient was hospitalized and underwent angiography that showed reperfusion of the left uterine artery, through collateral vessels, immediately after the previously embolized spirals. The gross arteriovenous vascular alteration of the uterine body was still recognizable. Another embolization was performed with 700 μm embozene particles and subsequent embolization of collateral vessels coming from the left uterine artery with 355/500 μm.

After the procedure, ultrasound control of the abdomen showed the reduction of arterial perfusion in the portion of the uterine body on the same side. However, gross arteriovenous vascular alteration was still present with slightly reduced flows in relation to the development of collateral vessels from the gonadal arteries and the uterine artery of the other side. Brain MRI was performed to study cerebral circulation and proved regular. She was discharged on the 6th day after embolization in good general condition (Hb 11.5 g / dL, bHCG 11 U / ml). Oral progestogen therapy was initiated. Three weeks after the second embolization, she went to the emergency room for persistent spotting after an episode of menorrhagia; hemoglobin was stable on blood tests (Hb 12.2 g/dL). Transvaginal ultrasound showed a linear endometrial cavity at the fundic level with a thickness of 5 mm, while, at the isthmus level, there was a non-uniform hyperechoic area of 19 × 16 mm with doubtful vascularization that deepened in the posterior myometrium. The

abdominal transsound performed showed a coarse arteriovenous vascular alteration starting from the uterine fundus with endocavitary development, and the flows had not change significantly compared to those found in the previous control. The patient was discharged with careful follow-up every 3 months without sequelae.

3.2. Case 2

The second case was a 30-year-old Caucasian woman, primigravida, whose pregnancy, followed by a private doctor, had a regular course. Her personal and family history was without complications. During pregnancy, the first trimester ultrasound was performed showing an embryo CRL corresponding to amenorrhea. The combined test was at low risk for trisomies. The second trimester ultrasound was normal, and the III trimester ultrasound check up showed fetal growth at the lower limits, however, with regular maternal-fetal Doppler A 41 + 3 gestational weeks. She was hospitalized for labor. This was performed with a cervical balloon, misoprostol (2 administrations), amniorex and oxytocin. On 08/08/2022 there was an operative delivery assistance with a traction of the obstetric suction cup (Kiwi Omni Cup) due to the concerning cardiotocographic disposition and the extension of the II stage of labor. Detachment was spontaneous and complete. A right paramedian episiotomy was performed. Blood loss was 250 ml, so uterotonic prophylaxis was performed at the time of delivery (Syntocinon 10 IU IM). A female newborn weighing 2875 g was born with apgar I 7, apgar V 10 and venous pH 7.30. The puerperal course was regular, and the newborn breastfed. The patient was discharged on the 2nd day after

giving birth with Hb 11.9 g/dL. On the 21st day after birth, she was admitted to the emergency room for abundant loss of bright red blood at home. At transvaginal ultrasound, the uterine cavity at the fundic level appeared dilated by vacuolar material with a thickness of 20 × 33 mm, with color score 0 (Fig. 4).

There was a suspicion of retained placental material. 1 fl of Methergin IM was administered. She was discharged in good clinical condition (Hb 13.9 g/dL) with oral therapy with Methergin for five days. A week later, an ultrasound re-evaluation was performed which showed a persistent non-homogeneous thickening of 13 × 26 mm at the fundic level, slightly reduced compared to the previous control. Oral progestogen therapy and two-month reassessment were recommended. On the 34th day after giving birth, she went to the emergency room for an episode of metrorrhagia. On transvaginal ultrasound evaluation, the uterine cavity was enlarged with non-homogeneous material of 12 × 19 mm in which there was an area with burning vascularity suggestive of AVMs with an intramyometrial vascular peduncle in the right anterolateral site. Blood chemistry revealed moderate anemia (Hb 8.4 g / dL), so the patient was hospitalized and transfused with 2 bags of GRC. bHCG was analyzed for diagnostic supplementation and was 10 IU / ml. Ultrasound control of the abdomen showed a hyperechoic tissue with vascular structure that had markedly accelerated, and turbulent flows on color Doppler evaluation suggesting the presence of arteriovenous fistula in AVMs acquired during postpartum. On the 35th day after delivery, the patient underwent angiography and the diagnostic evaluation presented the well-known arteriovenous fistula, small in size, with a thin and tortuous afferent vessel that originated from the left uterine artery. Due to the impossibility of superselective microcatheterization and patient stability, the embolization procedure was suspended. The right uterine artery was also evaluated with no evidence of collateral supplies to the known fistula. A second angiography was performed at 48 h: an attempt to catheterize the thin vascular branch with a micro catheter was unsuccessful, so it was decided to embolize the left uterine artery with absorbable Spongostan material, immediately distal to the origin of the cervicovaginal artery. The vascularization of the uterus appeared regular, the right vascular dilatation with turbulent flow previously described was absent (Fig. 5). The patient was prescribed oral progestogen therapy.

4. Discussion

AVMs may be suspected in women of reproductive age with metrorrhagic menstrual cycles or who have had postpartum bleeding or uterine curettage. Uterine bleeding can often be intermittent, metrorrhagic, and diagnosed when it fails to respond to medical therapy. In some women, symptoms related to pelvic congestion (pelvic pain, dyspareunia), rarely heart failure, may be associated if the vascular lesion is very large [5,6]. At the etiological level, we report two clinical cases with different medical histories. In fact, in the first case, the patient had performed a scraping of the intrauterine cavity after an abortion, and this is in accordance with the data of the literature that report how intrauterine surgical manipulations can be associated with an increase in immune response and angiogenesis, disturbing the uterine physiology. In fact, Peitsidis et al. [7] report a systematic review of 91 studies, which highlighted the presence of AVMs acquired after curettage in 95 of the 103 patients with AVMs. But uterine surgery may not be the only cause of AVMs, as in the second case we reported, in which the patient had not undergone intrauterine surgery. In a retrospective study by Kim et al. [8] in which 19 patients who developed AVMs after delivery, about a quarter of patients had no history of curettage. In these cases the hypothesized etiopathogenetic mechanism is aberrant regression of the placental bed or abnormal vascular communication after chorionic villus necrosis. As far as the diagnostic procedure is concerned, clinically the diagnosis may not be immediate but sometimes the presence of a pulsatile mass can be detected on physical examination [9–11]. B-HCg assay may also guide diagnosis; in fact, in women of childbearing age who present with abnormal vaginal bleeding and a negative serum b-HCG value, AVMs should always be suspected [12–17]. Conversely, in patients with positive serum hCG levels even after 1 month after delivery, a placental polyp should be suspected [18]. Finally, for those patients with persistent high serum hCG levels, gestational trophoblastic disease and placental site trophoblastic tumor may be excluded [19–22]. In the past, the diagnosis of AVMs was often only histological after the discovery of arteriovenous fistula after hysterectomy [23–25], but today, thanks to the advent of imaging modalities such as color Doppler ultrasound, magnetic resonance imaging, computed tomography and pelvic angiography, diagnosis is easier and possible earlier. Pelvic angiography remains the gold standard for the diagnosis of AVMs [26–28], although it is not routinely used since ultrasound has a good detection [28–30]. In fact, ultrasound, in particular with the use of color Doppler, can allow the differential diagnosis between retained products of conception (RPCO), subinvolution of the placental bed, adenomyosis [31–35] and uterine AVMs that mainly involve only the myometrium showing serpiginous tubular vascular structures [36–38]. Even in the
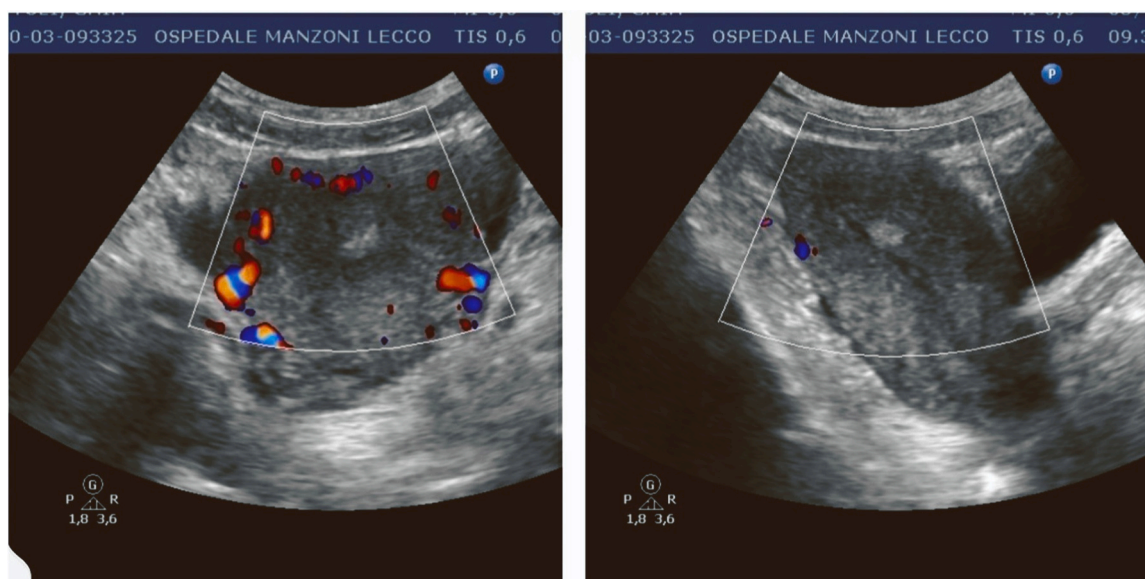

Fig. 4. Ultrasound exam: the uterine cavity at the fundic level appeared dilated by vacuolar material with a thickness of 20 × 33 mm.
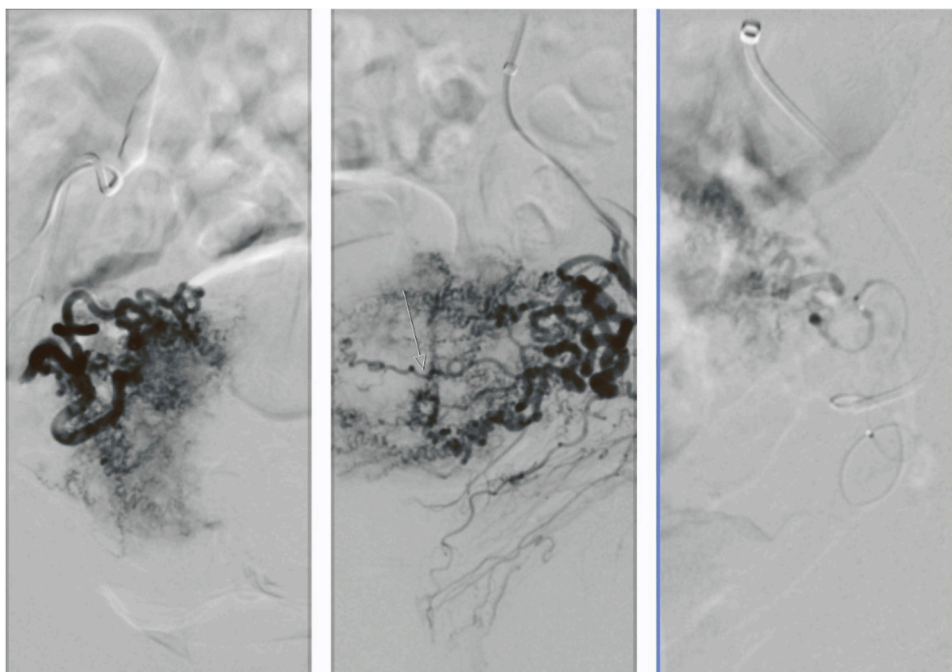
Fig. 5. Angiography: evaluation pre and post UAE. Pre UAE: it performed an arteriovenous fistula, small in size, with a thin and tortuous afferent vessel that originated from the left uterine artery. Post UAE: uterine vascularization appeared regular, right vascular dilatation with turbulent flow previously described was absent.

cases we reported, imaging allowed the detection of the presence of a hypervascularized area in a hypoechoic myometrial area, with high multidirectional flow velocity and low vascular resistance. In AVMs, a direct communication is created between an artery and a vein (arteriovenous fistula) [3,39–42] (Table 1), which some authors describe as a "color mosaic" model in which a low-resistance/high-velocity flow pattern is found in the abnormal vessels [43,44].

The final diagnosis of AVMs is confirmed by angiography, which is considered the "gold standard" technique by many authors. With this technique it is possible to highlight the early venous filling of a uterine vascular network that represents a pathognomonic finding [3]. If diagnostic doubts persist, follow-up with CT or MRI angiography may be necessary [2]. MRI and angiography can provide the exact anatomical location of the lesion also showing the relationship with the pelvic vessels [43]. Once the diagnosis of uterine AVMs is confirmed, treatment depends on the clinical condition of the patient. Both of our patients had conservative embolization treatment. But the management of AVMs varies depending on the patient's symptomatology, their hemodynamic status, the size and location of lesions, and patient age, as well as the desire for future fertility. Treatment options for post-pregnancy AVMs include medical treatment, uterine artery embolization (UAE), or hysterectomy. In 1982 Forssman et al. [46,47] first described the conservative approach of uterine arteriovenous aneurysm, introducing gelfoam into the uterine artery. Subsequently, in a review that included 100 women with iatrogenic AVMs after intrauterine curettage and with acute abnormal uterine bleeding, 59% of patients underwent UAE, 29% had a hysterectomy, 6% responded to methylergometrine, and 6% had spontaneous resolution. The goal is first and foremost to maintain hemodynamic stability and contain bleeding. In fact, a blood transfusion and a utero-cervical-vaginal tamponade following vaginal bleeding is sometimes necessary [48]. In addition, several treatment regimens have been described to reduce leeching in AVMs, including: progestogens, gonadotropin-releasing hormone (GnRHa) agonists, methotrexate, combined hormonal contraception, uterotonics or danazol [49]. Moreover, a systematic review recently reported an overall success rate of 88% for the medical treatment of uterine AVMs [50]. The advantage of medical treatment is that it is easy to obtain and cheaper than

procedural options. It also prevents risks and potential future fertility implications of embolization. The most frequent embolic agent used in patients who desire fertility is gelfoam due to its temporary nature [36, 51]. On the other hand, no difference in clinical outcome with other embolic agents is reported [40,45]. Although medical management is a fertility-sparing option, it is advisable in patients who are hemodynamically stable and/or have mild uterine bleeding. In patients with heavy uterine bleeding and unstable edynamic status, UAE or other procedural options remain the treatment of choice. Embolization is generally performed by the catheter femoral artery in a similar way to embolization of leiomyomas [46,52]. UAE is the treatment of choice, as it is a less invasive treatment option for patients who wish to preserve fertility. It is also fast, with minimal side effects and complications, shorter hospital stays, and faster recovery. A recent study, although of a retrospective nature and with small sample size, showed that UAE was successful in the symptomatic management of all fifteen patients presenting with post-pregnancy AVMs without significant complications. Half of the women who wished to become pregnant conceived, demonstrating how this procedure can be offered to young patients with post-pregnancy AVMs. In particular, in recent years the overall success rate after embolization treatment has been 88.4%, with 79.2% after first embolization and 66.7% after repeated embolization [9,53], thanks to improvements in this technique. Post-embolization complications are most frequently pelvic pain and fever. If vaginal blood loss remains uncontrollable even after UAE, hysterectomy should be performed promptly to avoid catastrophic bleeding [45]. In cases where medical treatment or UAE are contraindicated, other treatment options are hysterectomy, hysteroscopic resection, uterine artery ligation and AVMs resection [54]. Possible side effects of UAE are fever, infection, transient claudication of the buttocks and lower limbs, pain, perineal skin peeling, urinary bladder necrosis, uterovaginal and recto-vesicovaginal fistulas [39,43,48]. Although rare, pulmonary embolism is also another concern in the treatment of AVMs by UAE, as particles from catheter embolization can accidentally enter the pulmonary circulation, causing a pulmonary embolism [49]. Hysteroscopy is a feasible and safe alternative treatment modality for AVMs. However, hysteroscopic treatment should only be reserved for hemodynamically stable patients without heavy

Table 1

acquired UTERINE VENOUS MALFORMATIONS IN PUERPERIUM.

Paper ID	Study type	Sample size	Ultrasound findings
[2]	Case report	1 discovered postpartum (vaginal)	Echogenic, vascular structure posterior to the endometrial complex
[3]	Systematic review	47 patients of the UAVMs were discovered postpartum (vaginal or cesarean section).	Hypoechoic tortuous spaces involving the uterine wall. US Doppler: dilated and tortuous blood vessels with a multidirectional high velocity and low resistance flow in the myometrium.
[4]	Retrospective study	6 cases discovered postpartum (vaginal or cesarean section).	Vascular channels within the myometrium
[37]	Overview	6 studies	Hypoechoic or anechoic intrauterine lesion. Ill-defined mass that consists of multiple hypoechoic or anechoic serpentine or cystic structures in the myometrium, with a normal adjacent endometrium. US Doppler: bidirectional systolic and diastolic flow, aliasing, low-resistance and high-velocity turbulent flow. Lack the typical myometrium- to-endometrium vascularization pattern.
[9]	Overview	4 studies	Endometrial thickening and heterogeneity. US Doppler: nidus of turbulent serpentine vessels.
[45]	Case report	2 patients	US Doppler: various abnormalities that cannot be differentiated from the findings of placental polyps Presence of blood flow in placental polyp tissue as well as the size of the vascularized mass and arteries that supplies blood to the placental polyp.
[39]	Case report	1	US Doppler: bidirectional flow Velocity is high, with absolute values ranging from 175 cm/s to > 250 cm/s.
[43]	Case series	15	US Doppler: "Color mosaic" pattern. (turbulent high-velocity blood flow). Low-resistance flow and mixing of arterial and venous waveforms in the myometrium. Systolic and diastolic velocities being 4–6 times higher than observed in normal myometrial vessels
[6]	Review		Multiple hypoechoic or anechoic serpentine spaces within the myometrium. US Doppler: Spectral analysis demonstrates high- velocity and low resistive index.
[46]	Retrospective study	17	Ultrasonographic findings of a UVM are classically hypoechoic tortuous spaces in the myometriumUS Doppler: low vessels impedance and high velocity flow. These hypervascular areas can be located uni or bilaterally in the myometrium.

bleeding with mild to moderate AVMs [50]. In addition, the resection procedure can cause a defect of the normal myometrium during excision of AVMs tissue, therefore it can have an impact on future fertility. If the patient is hemodynamically stable or there is profuse vaginal bleeding in progress, Yan et al. reported the possibility of using USgHIFU to treat a patient with AVMs, although this study reported no follow-up results [2]. Other alternatives to hysterectomy are laparoscopic bipolar coagulation of the bilateral uterine arteries and suspensory ligament of the ovary and mesosalpinges near the uterine side. The literature reports that after this conservative laparoscopic procedure at 12-month follow-up there was a noticeable narrowing of lesion size and a noticeable decrease in all impedance measurements [PI, RI, VI (S/D)] during the 12-month follow-up. This method is linked to a number of complications such as uterine necrosis and pelvic pain. Furthermore, for those who wish to preserve fertility, simultaneously blocking bilateral uterine arteries and ovarian ligaments can significantly increase the risk of endometrial atrophy and may decrease ovarian function [54]. On the other hand there are a series of benefits, such as relief of symptoms, lack of radiation exposure and reduction of surgery time. In their last study, Chen et al. employed a combined laparoscopic method, in which the uterine arteries were occluded before performing myometrial resection at the level of AVMs. Subsequently, the uterus was reconstructed with an intact uterine cavity and abnormal vaginal bleeding was successfully stopped after the operation, but amenorrhea was reported following the formation of uterine adhesions [53]. A conservative approach was also proposed in a recent [54] article involving a combination of bilateral uterine artery ligation and AVMs suture. In this case bilateral ligation of the uterine artery was performed first. But because bilateral uterine artery ligation alone did not completely control AVMs, an AVMs suture was performed using an absorbable barbed wound closure device that blocked blood flow in AVMs. The benefits of this combination treatment modality include lack of radiation exposure, no risk of pulmonary embolism consistent with UEA, and rapid symptomatic relief. The disadvantages of this procedure were the risk associated with general anesthesia and the skills needed to perform laparoscopic surgery.

5. Conclusions

In view of the literature search, due to the scarcity of cases there are no universal treatment guidelines for uterine AVMs. The patient with abnormal postpartum uterine bleeding should be carefully evaluated and followed in the days following delivery. The diagnosis of AVMs is often not immediate through ultrasound and color-Doppler but must be considered, especially if the patient presents an endouterine scraping in their history. The surgical management of AVMs must be customized, and although in the literature there are new emerging techniques, UEA, to date, represents the elective treatment, especially for patients who wish to preserve fertility.

In addition, a more in-depth discussion on this topic by the various specialists (gynecologist, vascular surgeon, radiologist) would allow to devise guidelines for the management of AVMs starting from a multidisciplinary and personalized approach. Finally, we underline that the final therapeutic decision should always ultimately be approved by the patient, following detailed counseling and signing of specific and accurate informed consent.

Institutional review board statement

Considering that data analyzed in this study were collected during routine clinical activity and fully anonymized, and that investigators did not perform any interventional procedure, formal Institutional Review Board approval was exempted due to the observational nature of the study.

Informed consent statement

Informed consent was obtained from all subjects involved in the study.

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CRediT authorship contribution statement

Conceptualization, **G.R.D** and **M.D.**; methodology, **G.R.D.**; software, **H.X.**; validation, **D.D.G.**, **R.A.** and **A.V.** **(Amerigo Vitagliano)**; formal analysis, **G.R.D.**; investigation, **A.V. (Antonella Vimercati)** and **V.L.**; resources, **A.M.**; data curation, **E.C. (Eliano Cascardi)**; writing—original draft preparation, **G.R.D, E.C. (Eliano Cascardi)** and **M.D.**; writing—review and editing, **C.P.**, **L.L.**, **R.F.**, **G.R.D, E.C. (Eliano Cascardi)** and **M.D.**; supervision, **E.C. (Ettore Cicinelli)** and **A.P**. All authors have read and agreed to the published version of the manuscript.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this article.

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