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INVITED REVIEW

Multimodality imaging in secondary postpartum or postabortion hemorrhage: retained products of conception and related conditions

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Abstract Secondary postpartum hemorrhage (PPH) and postabortion hemorrhage are rare complications. Retained products of conception (RPOC) is among the most common causes of both secondary PPH and postabortion hemorrhage. Other less common causes of secondary PPH are uterine vascular abnormalities such as arteriovenous malformations and pseudoaneurysms. These are usually related to a history of a procedure such as dilation and curettage or cesarean delivery. Subinvolution of the placental site is an idiopathic cause of secondary PPH; this condition may be underrecognized and therefore could have a higher incidence than currently reported. Gestational trophoblastic disease is rare but commonly presents as secondary PPH and resembles RPOC in radiologic appearance. The first-line imaging modality for secondary PPH is ultrasound, but computed tomography and magnetic resonance imaging may be used if the ultrasound findings are indeterminate. Angiography is an important tool for the definitive diagnosis of uterine vascular abnormalities. Appropriate management requires radiologists to be familiar with the multimodality imaging features of secondary PPH or postabortion hemorrhage.

Keywords Postpartum hemorrhage · Postabortion hemorrhage · Retained products of conception · Uterine vascular malformation · Subinvolution of the placental site

Introduction

Postpartum hemorrhage (PPH) is divided into primary and secondary types: primary PPH is defined as blood loss greater than 500 mL in the first 24 h after delivery, and secondary PPH refers to excessive uterine bleeding occurring between 24 h and 6 weeks postpartum [1]. Secondary PPH is less common than primary PPH, and its bleeding is not usually catastrophic. A large cohort study reported that the incidence of severe secondary PPH is 23 per 10,000 deliveries (0.23%) [2]. Severe or continuing hemorrhage after abortion is also rare, occurring in less than 1% of abortions, with delayed bleeding being more common than immediate postabortion bleeding [3].

While primary PPH is usually the result of uterine atony, the major cause of secondary PPH or postabortion hemorrhage is retained products of conception (RPOC). Another important cause of secondary PPH is subinvolution of the placental site, which may be underrecognized. Other rare causes of secondary PPH are uterine arteriovenous malformations (AVMs), uterine pseudoaneurysms, and gestational trophoblastic disease (GTD).

The management of secondary PPH or postabortion hemorrhage depends on the degree and the cause of bleeding and on the patient's desire for future fertility. The presence of RPOC is usually treated with dilation and curettage (D&C) or hysteroscopic resection, while pseudoaneurysm and AVM

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are treated with uterine artery embolization (UAE). Accurate imaging diagnosis is therefore important for prompt and appropriate management of secondary PPH.

The first-line imaging modality for secondary PPH is ultrasound (US). Color Doppler US is very useful for evaluating lesion vascularity and plays an important role in the diagnosis of RPOC and other uterine vascular lesions. Magnetic resonance imaging (MRI) should be reserved for cases in which US findings are inconclusive. Contrast-enhanced computed tomography (CT) is available for emergency use, although this modality involves low-dose radiation exposure. Angiography is an important tool for arriving at a definitive diagnosis and subsequently treating vascular lesions.

We discuss herein RPOC and related conditions as the principal causes of secondary PPH and postabortion hemorrhage, focusing on their pathogenesis, radiologic characteristics, and best management.

Retained products of conception

The term RPOC refers to intrauterine tissue of trophoblastic origin that develops after conception and persists after delivery or termination of pregnancy [1]. The presence of chorionic villi on microscopy confirms the diagnosis of RPOC, which is among the most common causes of both delayed PPH and postabortion hemorrhage. The condition is seen more frequently after spontaneous abortion or termination of pregnancy than after vaginal or cesarean delivery [4]. In one prospective study, RPOC was more often diagnosed after first-trimester (17%) or second-trimester (40%) spontaneous or therapeutic abortion, occurring after a third-trimester delivery in only 2.7% of women [5].

The risk factors for RPOC are failure to progress during labor, placenta accreta, and instrumental delivery. Common symptoms include genital bleeding and pelvic pain. Serum human chorionic gonadotropin (hCG) levels may be mildly to moderately elevated.

On grayscale US, RPOC appears as an echogenic mass in the endometrial cavity, and various degrees of vascularity in the mass can be detected on color Doppler US. Detectable vascularity in an endometrial mass supports a high diagnostic confidence for RPOC; a lack of vascularity can be seen with either intrauterine clots or avascular RPOC.

Vascularity of an endometrial mass is graded according to degree [6]. Type 0 is defined as undetectable vascularity in the endometrium; type 1 (minimal vascularity) as some detectable color Doppler flow in the endometrium, but less than that seen in the myometrium in the same image section; type 2 (moderate vascularity) as vascularity equal to or nearly equal to that seen in the myometrium in the same image section; and type 3 (marked vascularity) as marked endometrial vascularity greater than that seen in the myometrium in the same image section (Fig. 1a).

Evaluation with MRI typically shows an endometrial polypoid mass with heterogeneous signals on T1- and T2-weighted images (Figs. 1b, 2a). Variable enhancement can be seen on postcontrast images based on the vascularity of the RPOC (Fig. 2b). Dynamic gadolinium-enhanced MRI may support the US findings regarding the depth of myometrial invasion and the vascularity of the mass [7]. Contrast-enhanced dynamic CT is also useful for a highly vascularized endometrial mass and for clarifying the vascular anatomy before UAE. In hypervascular RPOC, CT shows an intensely enhancing mass in the uterine cavity during the arterial phase (Figs. 1c, 3a); however, precise localization of the lesion is somewhat vague on CT compared with MRI.

The differential diagnosis of a vascular endometrial mass seen on imaging includes GTD and uterine AVM. These may present with similar postpartum or postabortion symptoms. GTD can be excluded if the serum level of the beta subunit of hCG (β-hCG) is markedly elevated. Hypervascular RPOC (type 3) and uterine AVM can have overlapping clinical appearances and can sometimes coexist, but strict differentiation may be difficult.

The management and treatment of RPOC depends on the degree of vaginal bleeding, the amount of vascularity, and the size of the endometrial component. If the patient complains of intermittent vaginal bleeding and the endometrial component appears hypovascular (type 0, 1), medical treatment and close follow-up may be adequate. If the endometrial component appears either hypovascular and large or contains moderate vascularity (type 2), D&C may be recommended. If the endometrial component appears hypervascular (type 3), either surgical removal of the residual tissue under US guidance or UAE may be recommended [8, 9] (Fig. 3b).

Uterine arteriovenous malformation/uterine vascular malformation

Uterine AVM is a rare but life-threatening condition that potentially causes massive genital bleeding. Recently, the term "uterine vascular malformation (UVM)" has been used to describe a vascular abnormality of the uterus. This entity is classified into "true AVMs" and "non-arteriovenous vascular malformations (non-AVMs)" according to the symptoms and angiographic findings [10]. True AVM is characterized by an arteriovenous shunting and is categorized as a high-flow UVM, whereas non-AVM lacks this shunting and is categorized as a low-flow UVM, which is thought to be the result of subinvolution of the placental site. Here we focus on true AVM in this section.

Uterine AVMs are classified into two categories: congenital and acquired. Secondary PPH is usually caused by an acquired AVM, which frequently results from trauma: either a D&C performed for spontaneous abortion, a

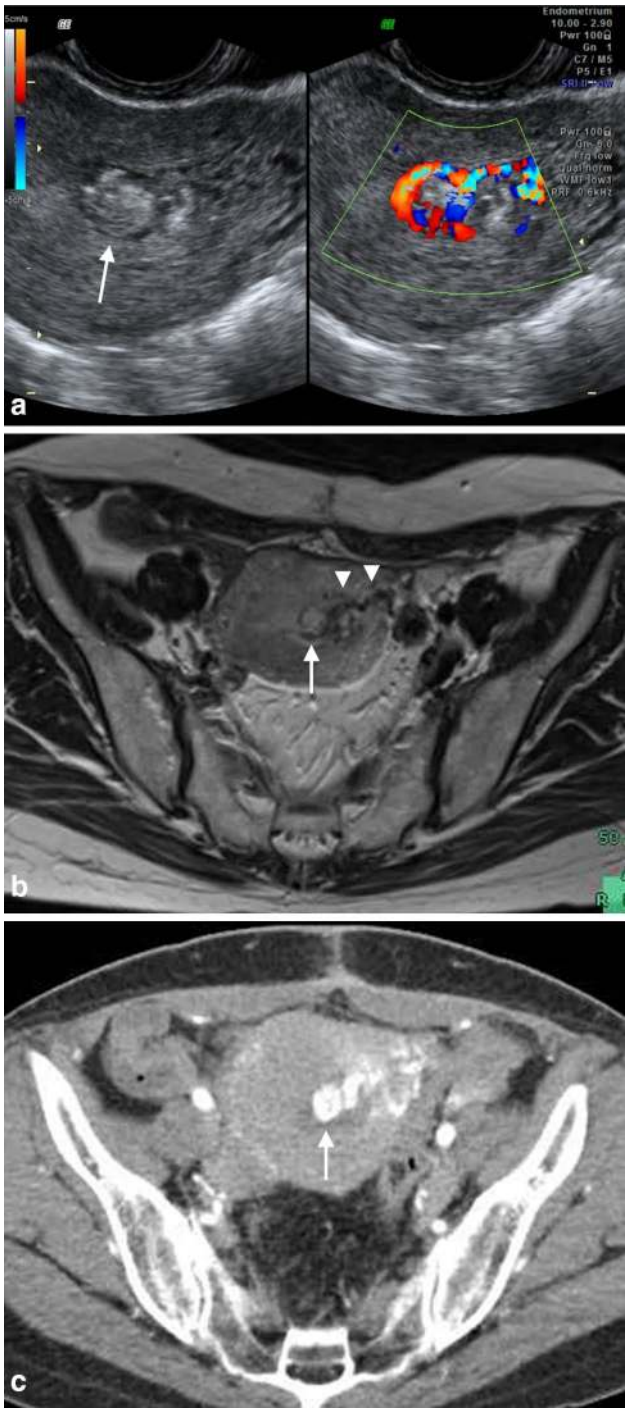

Fig. 1 42-year-old woman with massive genital bleeding 2 months after a cesarean delivery for cephalopelvic disproportion. **a** Doppler ultrasound shows an endometrial mass (arrow) with peripheral dominant vascular flow continuous with the left myometrium; the peak systolic velocity is 75 cm/s. **b** Magnetic resonance imaging (MRI) shows a hyperintense endometrial mass (arrow) with a peripheral flow void (arrowheads) continuous with the left myometrium on axial T2-weighted imaging. **c** Contrast-enhanced computed tomography (CT) during the arterial phase shows a hypervascular lesion (arrow) corresponding to the uterine cavity. Uterine artery embolization and subsequent hysteroscopic resection were performed, confirming the diagnosis of retained products of conception. Note that CT cannot delineate the peripheral vascular component from central hypervascular organization

therapeutic abortion, or cesarean delivery [11]. Uterine AVMs are thought to arise from the placental site, where venous sinuses become integrated into the myometrial scar after necrosis of the chorionic villi following iatrogenic uterine damage or pregnancy-related changes.

The incidence of uterine AVM is unknown, but one prospective study reports an incidence of true AVM of 0.1% in 959 consecutive patients undergoing either abortion or delivery [12], with a significantly higher rate after abortion. Another study reported the incidence of AVM as 4.5% in 464 patients with pelvic bleeding [13]. Uterine AVM is thought to be extremely rare condition, but the number of reports of uterine AVMs is increasing.

The most common symptoms of uterine AVM are menorrhagia or metrorrhagia, with either gradual or sudden onset. Bleeding is often intermittent and torrential.

The grayscale US findings of uterine AVM are nonspecific, but the addition of color Doppler allows for correct diagnosis [11]. The presence of multiple hypoechoic or anechoic serpentine spaces within the myometrium that exhibit vascular flow raises the suspicion of uterine AVM (Fig. 4). The identification of lesions with multidirectional, high-velocity, and low-resistance flow on spectral analysis, consistent with arteriovenous shunting, is highly suggestive for uterine AVM [14]. Timmerman et al. [10] reported that the peak systolic velocity (PSV) appears to be useful for predicting low- and high-risk patients with uterine AVM. The use of color Doppler US is now considered a diagnostic modality for uterine AVM.

The MRI findings of true AVM include a bulky uterus, a focal mass with disruption of the junctional zone, multiple serpiginous flow voids within the lesion, and prominent parametrial vessels [11]. Dynamic MRI and magnetic resonance angiography are useful for delineating vascular lesions. Arterial-phase CT may show the presence of feeding arteries, draining veins, and any extension of the AVM. Angiography is the gold standard for diagnosing AVM: findings include a dilated, tortuous uterine artery feeding a hypervascular uterine mass and early venous return to an enlarged vein. Diagnostic angiography is now rarely performed, as it is an invasive procedure reserved for patients who require UAE.

Management of uterine AVM depends on the hemodynamic stability of the patient, the degree of bleeding, the patient's age, and their desire for future fertility. The use of D&C is contraindicated because it can aggravate uterine bleeding. Conservative management may be appropriate when the patient is hemodynamically stable or the

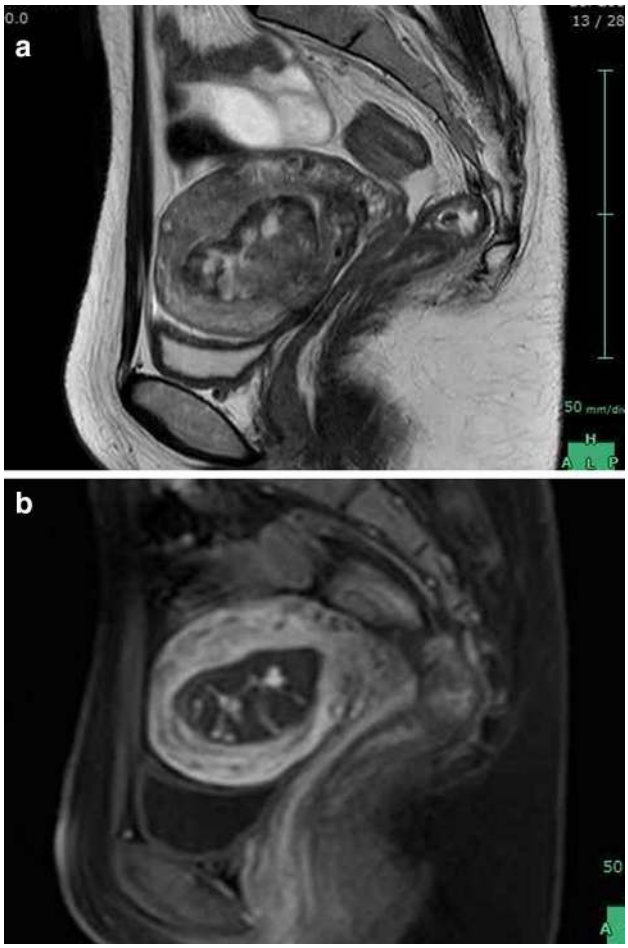

Fig. 2 35-year-old woman with massive genital bleeding 7 weeks after dilation and evacuation (D&E) for anencephaly at 13 weeks' gestation. **a** MRI shows a heterogeneous mass protruding into the endometrial cavity with both high and low signal intensity on T2-weighted imaging. **b** Gadolinium-enhanced (Gd+) fat-saturated T1-weighted imaging reveals slight mass enhancement. Repeat D&E was successfully performed, confirming the diagnosis of retained products of conception

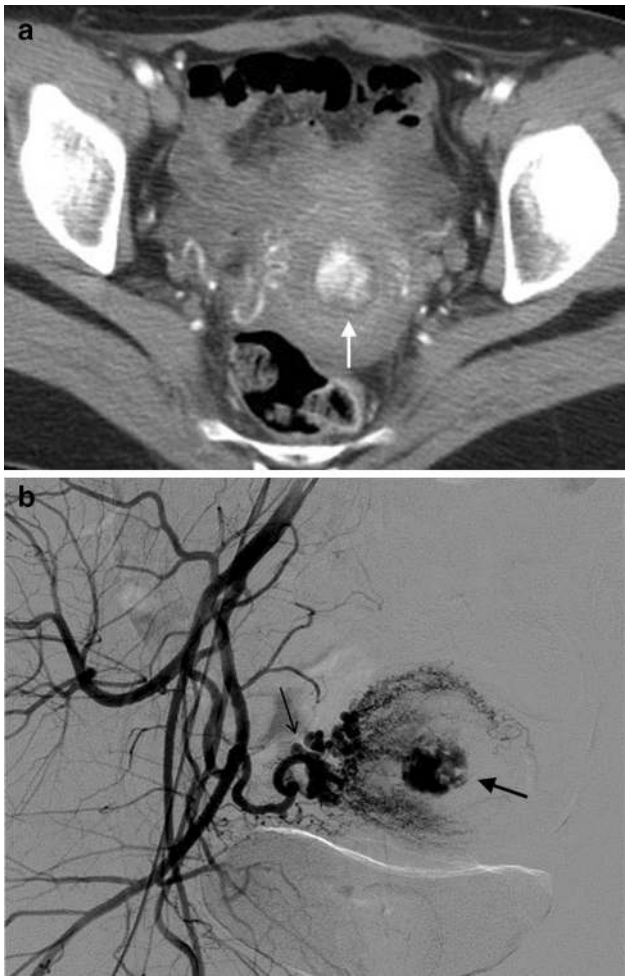

Fig. 3 25-year-old woman with genital bleeding 1 month after a normal vaginal delivery. Ultrasound showed an echogenic mass in the endometrial cavity at the lower uterine segment (not shown). **a** Contrast-enhanced CT during the arterial phase shows a markedly enhancing mass (arrow) in the uterine cavity. **b** Right internal arteriography shows a dilated right uterine artery (small arrow) and a hypervascular mass (arrow) corresponding to the uterine cavity. Uterine artery embolization and subsequent hysteroscopic resection were performed, confirming the diagnosis of retained products of conception

bleeding is persistent but mild [15–17]. Selective UAE is an effective treatment when there is persistent heavy bleeding or when conservative management fails. There are many reports on the safety and efficacy of UAE and the possibility of subsequent pregnancy after the procedure [18–22]. Balloon tamponade may be attempted before UAE to stop the bleeding and stabilize the patient [3]. Hysterectomy is not a preferred option but may be performed when UAE fails, the patient is hemodynamically unstable, or no future fertility is desired [14]. Timmerman et al. [10] suggest that women with a low measured PSV value (< 39 cm/s) may be treated conservatively, while those with a high PSV value (≧ 83 cm/s) require urgent treatment such as UAE.

Uterine artery pseudoaneurysm

Uterine artery pseudoaneurysm is a rare cause of PPH and a potentially life-threatening condition [23–26]. The incidence of uterine artery pseudoaneurysm is thought to be very low, reportedly causing around 3% of severe PPHs [2, 24]. A pseudoaneurysm usually lacks the typical vessel layers and is surrounded only by hematoma, the adventitial layer, or connective tissue, none of which can endure arterial pressure with resulting massive extravasation.

Pseudoaneurysms usually result from laceration or injury of the arterial wall and are often associated with surgical procedures such as operative delivery and D&C. Typically,

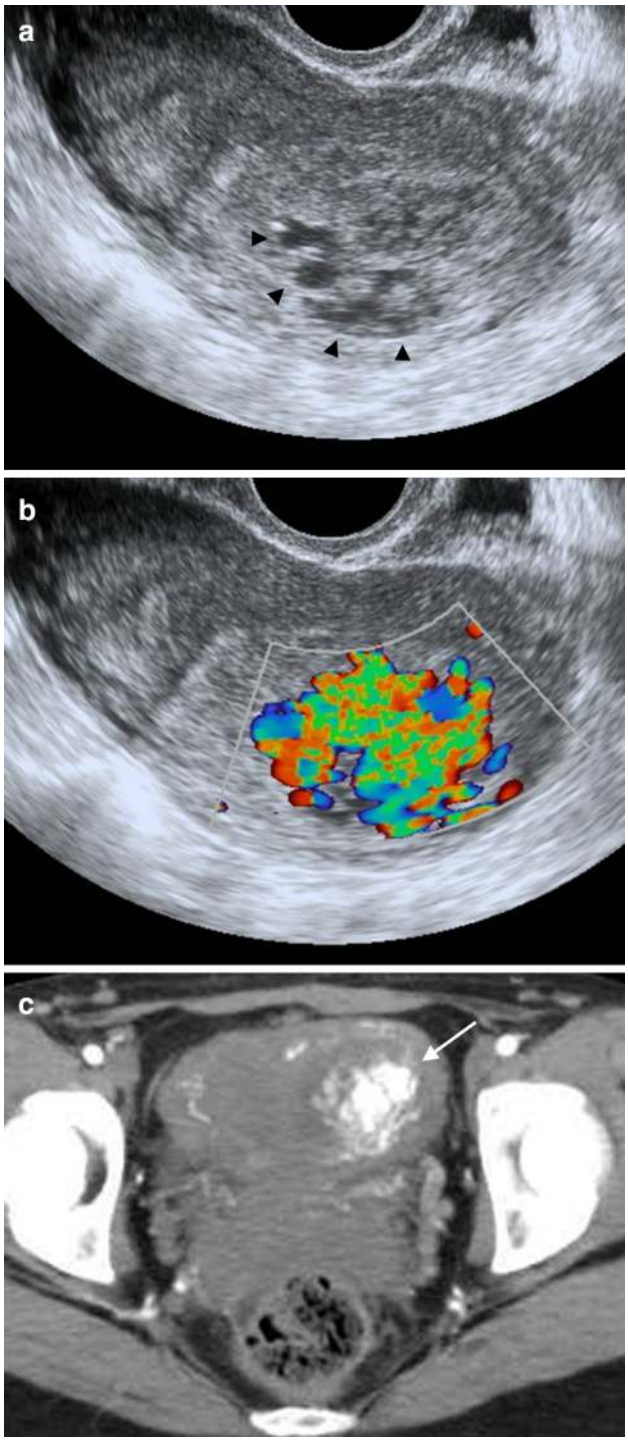

Fig. 4 24-year-old woman, 6 weeks after septic abortion at 16 weeks' gestation. **a**, **b** Doppler ultrasound shows myometrial anechoic spaces (arrowheads) with prominent vascular flow. **c** Contrast-enhanced CT shows a hypervascular lesion (arrow) corresponding to the myometrium. The genital bleeding was not severe, and the vascularity of the lesion gradually decreased and finally disappeared, suggesting spontaneous regression of a uterine vascular malformation

patients present with profuse, painless, late PPH, frequently after cesarean delivery. In contrast, the occurrence of uterine artery pseudoaneurysm after vaginal delivery without a traumatic procedure is extremely rare [27, 28]. The location of a pseudoaneusym after vaginal delivery tends to vary and may include the vaginal artery or obturator artery, while pseudoaneurysm after cesarean delivery usually involves the uterine artery [24].

The US findings of uterine artery pseudoaneurysm include an anechoic or hypoechoic intrauterine lesion (Fig. 5a), vascularity on color Doppler US, and turbulent bidirectional systolic and diastolic flow with aliasing on spectral Doppler. Contrast-enhanced CT is very useful for confirming the diagnosis in an emergency. Pseudoaneurysm may appear as a flow void on T2-weighted MRI with intense enhancement on gadolinium-enhanced MRI (Fig. 5b, c), and often surrounded by a hyperintense hematoma on T1-weighted imaging. However, MRI is not an ideal modality for emergency use. Angiography can clearly demonstrate and confirm the presence of a pseudoaneurysm, and facilicates subsequent embolization (Fig. 5d).

Transcatheter arterial embolization is a safe and effective treatment for uterine artery pseudoaneurysm, but D&C is contraindicated [23–26].

Subinvolution of the placental site

Subinvolution of the placental site (placental site vascular subinvolution or subinvolution of uteroplacental arteries) is an idiopathic cause of delayed PPH in which the large vessels of the placental bed fail to involute following delivery or termination of pregnancy. The cause of subinvolution is not known, but there is thought to be an abnormal immunologic relation between fetal-derived trophoblasts and maternal uterine tissue [29]. Hemorrhage resulting from subinvolution is most often seen in the second week postpartum, although it may occur up to several months later. It is more common in older multiparous women and may recur in subsequent pregnancies.

The macroscopic features of subinvolution are a soft uterus that is larger than expected for the timepoint after delivery [30]. Histologically, large superficial myometrial vessels are partially occluded by thrombi of various ages. The vessels are dilated, with hyaline material replacing the media. Extravillous trophoblasts persist in perivascular locations (Fig. 6a, Online Resources 1 and 2 of the ESM). These histologic findings are diagnostic of subinvolution in the appropriate clinical setting of secondary PPH. The diagnosis of subinvolution of the placental site is underrecognized, partly because pathologic specimens are needed to confirm the diagnosis. Some researchers find that the incidence of subinvolution of the placental bed is relatively high, occurring in 13–46% of women with

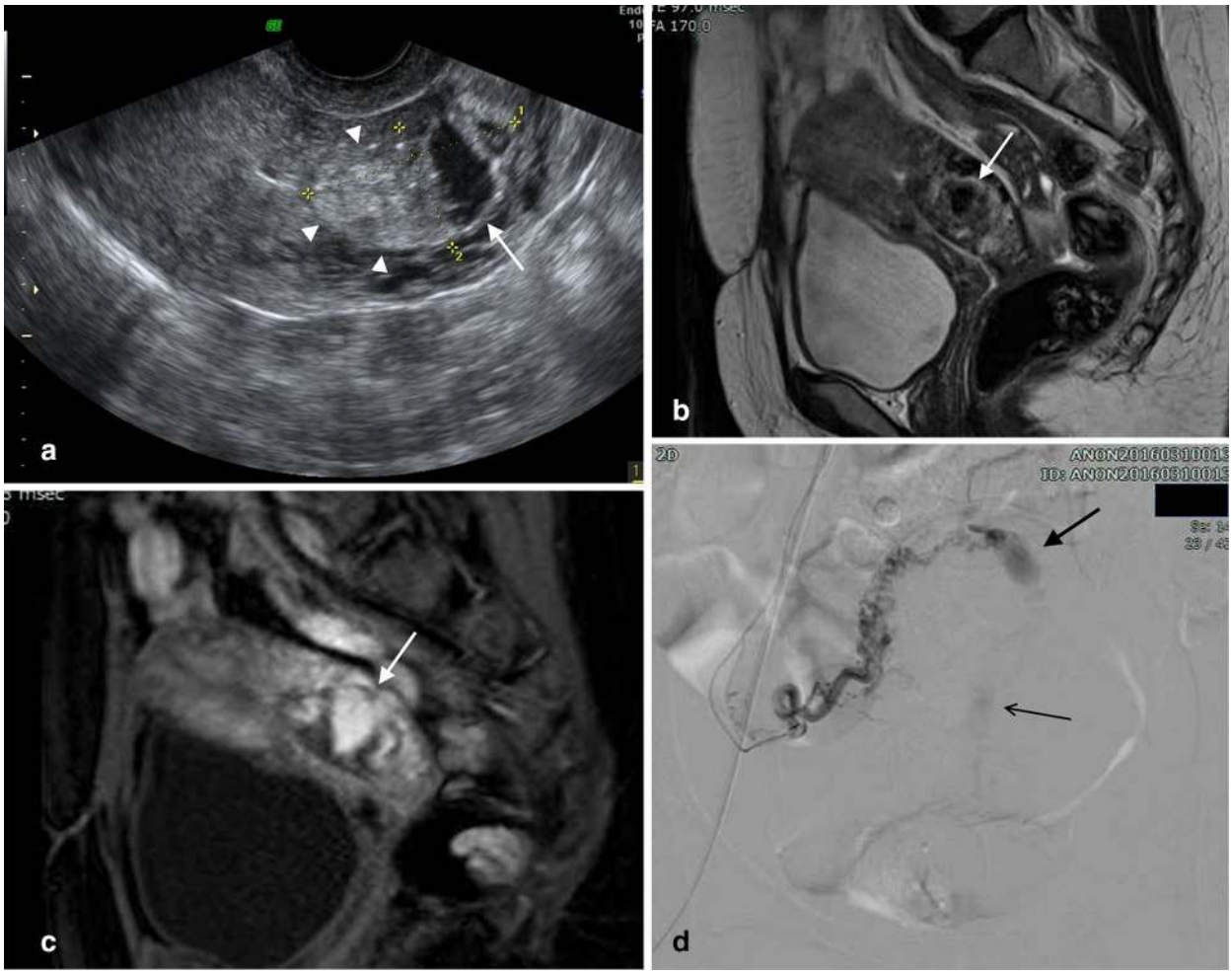

Fig. 5 44-year-old woman with massive genital bleeding 2 months after a cesarean delivery for placenta accreta. **a** Grayscale ultrasound reveals an echogenic mass (arrowheads) with an anechoic component (arrow), mainly in the lower segment of the posterior uterine wall, protruding into the endometrial cavity. Doppler ultrasound showed prominent vascular flow within the anechoic component of the mass; the peak systolic velocity was greater than 80 cm/s (not shown). **b** MRI shows a large flow void (arrow) within the mass on T2-weighted imaging. **c** Gd+ dynamic MRI in the arterial phase showed strong enhancement within the mass (arrow). **d** Angiography reveals an aneurysmal sac (arrow) corresponding to the mass, from which extravasation (small arrow) is observed. UAE and subsequent hysteroscopic resection were performed, confirming the diagnosis of retained products of conception. The clinical diagnosis was retained products of conception with concomitant pseudoaneurym secondary to placenta accreta and cesarean delivery

secondary PPH [2, 31]. Therefore, delayed postpartum or postabortion bleeding should always raise the possibility of subinvolution.

Grayscale US may show hypoechoic tortuous vessels within the myometrium at the location of the prior placental implantation site (Fig. 6b). Color Doppler US manifests prominent vascular flow with increased PSV and a low-resistance waveform [32]. There are no reports describing the CT and MRI features of subinvolution; however, we encountered a patient with an enlarged uterus containing vascular lakes at the posterior endometrial–myometrial interface on enhanced CT and MRI (Fig. 6c, d). Angiographically, subinvolution presents as hypertrophied uterine arteries. The uterine parenchyma rapidly opacifies and drains into large pelvic veins. No direct communication between the artery and vein is visualized [18].

The management of subinvolution of the placental site in bleeding symptomatic patients remains controversial. Conservative management with balloon tamponade and uterotonic drugs may be attempted; however, more aggressive treatment such as ligation of the uterine vessels, uterine compression sutures, and hysterectomy are considered standard therapies. Recently, some reports have recommended UAE to treat subinvolution [18, 32].

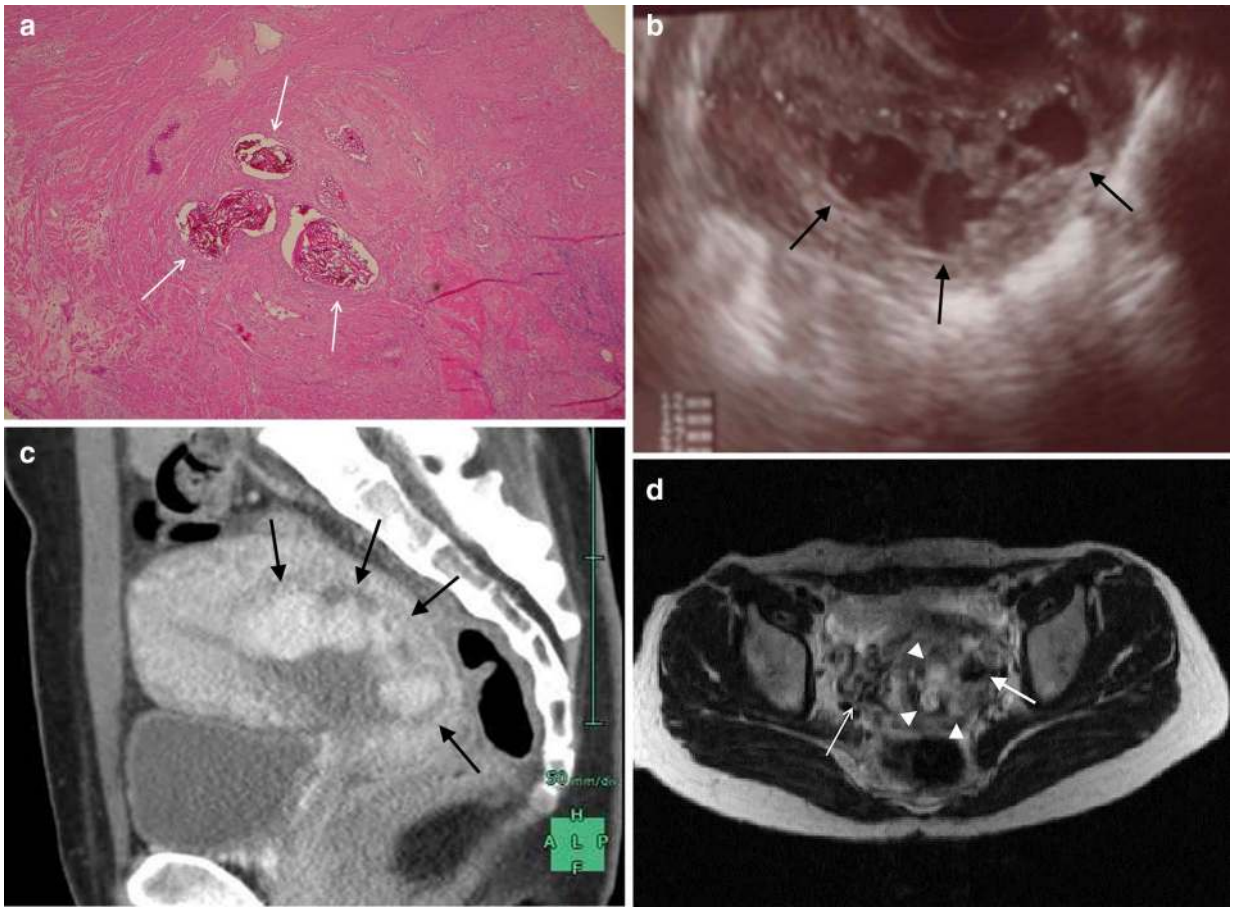

Fig. 6 32-year-old woman with massive genital bleeding 2 months after a spontaneous abortion at 8 weeks' gestation. **a** A low-power image shows large, patent, dilated superficial myometrial vessels (arrows) with pink hyaline material replacing the media and partial intravascular thrombosis (hematoxylin–eosin staining: HE). **b** Gray-scale ultrasound shows a large anechoic mass (arrows) in the posterior lower uterine segment. Doppler ultrasound showed prominent vascular flow within the mass (not shown). **c** Contrast-enhanced CT shows an enlarged uterus with vascular lakes (arrows) at the posterior endometrial–myometrial interface. **d** MRI shows high signal intensity (arrowheads) with a flow void (arrow) on T2-weighted imaging, corresponding to the vascular lakes on contrast-enhanced CT. There is a dilated and tortuous right uterine artery (small arrow). UAE and subsequent hysterectomy was performed. The initial pathologic diagnosis was uterine arteriovenous malformation, but detailed evaluation with immunostaining led to the final diagnosis: subinvolution of the placental site. These are the first reported CT and MRI findings of subinvolution of the placental site

Interpretation of uterine vascular lesions (highly vascularized retained products of conception, uterine arteriovenous malformation/uterine vascular malformation, and subinvolution of the placental site)

Uterine vascular lesions likely have some clinical and radiological overlap, and they seem to be closely related in their pathogenesis. Myometrial hypervascularity is often observed in normal pregnancy, particularly in the placental bed. The presence of hypervascular areas within the myometrium is a common finding in the postpartum and postabortion periods, and in most cases this appearance is associated with RPOC [33, 34]. Hypervascularity in the myometrium usually resolves spontaneously, but it tends to persist in the presence of RPOC. A possible explanation is that arteriovenous shunting within a uterine vascular lesion is thought to result from the development of arteriovenous fistulas within the placenta, usually caused by necrosis of the chorionic villi. Timmerman et al. [10] reported that 12 of 30 patients who were diagnosed with uterine AVMs had RPOC on pathologic examination. They mentioned that enhanced myometrial vascularity is a very common finding in the postpartum uterus, whereas UVM is less common and true AVM is exceedingly rare. An empty uterus has a high negative predictive value for ruling out RPOC, and vascular lesions within the myometrium are indicative of an AVM. The recent increasing number of reports of uterine AVMs may be associated with misleading imaging features of myometrially located RPOC; in other words, uterine AVMs may be overdiagnosed [9, 35].

Some physicians suggest that UVM should be considered subinvolution of the placental site [10, 18, 33]. They

Table 1 Causes of secondary postpartum or postabortion hemorrhage

	Frequency of occurrence	Site of lesion	β-hCG*	Imaging features	Pathologic features	Treatment	Differential diagnosis
RPOC							
Hypo- to moderate vascular RPOC	Most common	Endometrium	+	Endometrial echogenic mass with minimum-to-moderate vascularity	Chorionic villi	Conservative D&C	Blood clots Endometritis GTD
Hypervascular RPOC	Uncommon	Endometrium	+	Endometrial echogenic mass with marked vascularity		D&C** UAE hysteroscopic resection	GTD UVM Pseudoaneurysm
UVM							
Low-flow UVM (non-AVM***)	Uncommon	Myometrium	+	Uterine enlargement, large myometrial vessels without arteriovenous shunting	Subinvolution of uteroplacental artery	Conservative Balloon tamponade UAE Uterine suture Hysterectomy	Vascularized RPOC Pseudoaneurysm
High-flow UVM (true AVM)	Extremely rare	Myometrium	–	Tortuous, dilated, high-flow myometrial vessels with arteriovenous shunting	Proliferation of vascular channels with fistulae		Vascularized RPOC Pseudoaneurysm
Pseudoaneurysm of uterine artery	Rare	Endometrium-myometrium interface	–	Smooth-walled sac with high flow	Disruption of arterial wall	UAE	UVM Vascularized RPOC
GTD	Rare	Endometrium and myometrium	+++	Hypervascular uterine mass	Abnormal trophoblastic proliferation	Chemotherapy D&C Hysterectomy	RPOC UVM

AVM arteriovenous malformation, *D&C* dilation and curettage, *GTD* gestational trophoblastic disease, *β-HCG* human chorionic gonadotropin, beta subunit, *non-AVM* nonarteriovenous vascular malformation, *RPOC* retained products of conception, *UAE* uterine artery embolization, *UVM* uterine vascular malformation

* Possibly positive in postpartum or postabortion period, ** surgical removal under US guidance, *** probably represents subinvolution of the placental site

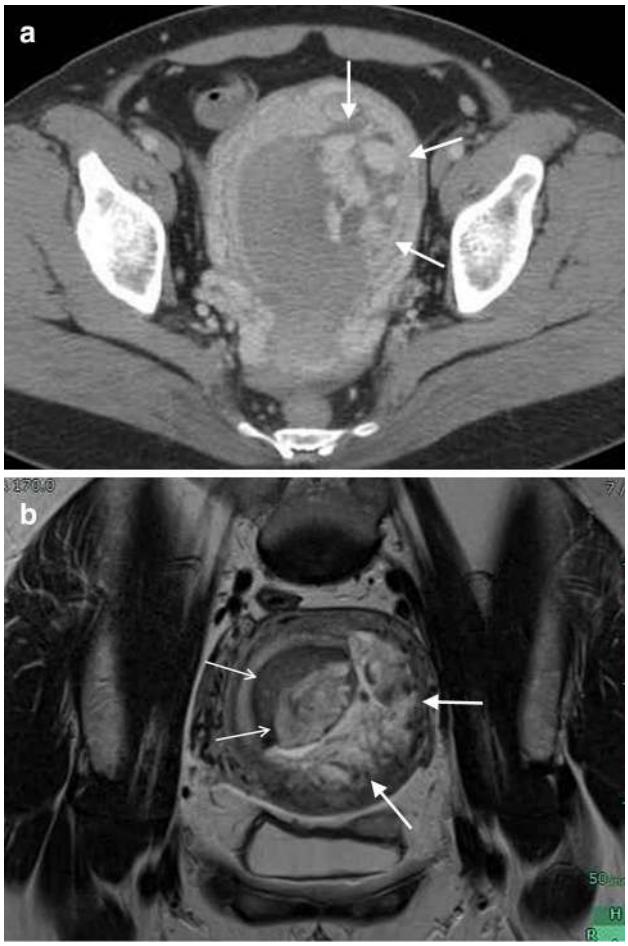

Fig. 7 40-year-old woman with persistent genital bleeding 3 weeks after dilation and curettage for molar pregnancy with markedly elevated serum human chorionic gonadotropin. **a** Contrast-enhanced CT shows marked enhancement of the mass (arrows), which protrudes into the endometrial cavity. **b** MRI shows a hyperintense mass in the left side of the uterine wall (arrows), with myometrial invasion on T2-weighted imaging. Blood clots are also seen in the uterine cavity as masses with low to high signal intensity (small arrows)

categorize true AVM as a high-flow UVM and non-AVM as a low-flow UVM (the latter probably the result of subinvolution of the placental site). This explains the natural course of myometrial hypervascularity and the spontaneous regression of UVM, where delayed involution of the placental bed occurs over time. Darlow found that the hCG levels of patients with uterine vascular lesions resolved slowly as the lesions regressed [36], which supports the aforementioned hypothesis that low-flow UVM represents subinvolution of the placental bed.

A clinical management dilemma arises from the overlap in these uterine vascular lesions. Timor-Tritsch recommends stratification of uterine vascular lesions by PSV regardless of the presence of RPOC, with UAE considered if the PSV of the lesion is high (≥ 60–70 cm/s), a recommendation based on Timmerman's report [10, 37]. Another study of uterine AVM, including patients with concomitant RPOC, suggests that PSV values greater than 76.2 cm/s may indicate a dangerous AVM, and that PSV values less than 35.8 cm/s appear safe [16]. The authors found that the PSV of an AVM is a strong differentiating variable on multivariable analysis, and concluded that US can be used to choose the most appropriate treatment.

The disorders causing secondary postpartum or postabortion hemorrhage are shown in Table 1.

Gestational trophoblastic disease

The entity of GTD encompasses a spectrum of trophoblastic cell proliferation disorders with excessive production of β-hCG, including hydatidiform mole, invasive mole, and choriocarcinoma [38, 39]. The latter two conditions may manifest as secondary PPH or postabortion hemorrhage. Invasive moles may occur as a complication of a hydatidiform mole invading the uterus; they occasionally spread to the lungs or vagina. Choriocarcinoma is a chorionic epithelial neoplasm that can arise after a molar or nonmolar pregnancy, with metastases usually seen in the lung and brain.

The initial diagnosis is based on a multimodal approach: the patient's history (e.g., D&C of a molar pregnancy, normal term or preterm pregnancy, abortion, ectopic pregnancy), serial quantitative β-hCG levels, and pelvic US. The US findings of invasive mole and choriocarcinoma are heterogeneous, echogenic, hypervascular uterine masses with possible myometrial invasion. Areas of necrosis and hemorrhage can be seen within choriocarcinoma. The hypervascular nature of these tumors can be helpful in detecting myometrial invasion, although invasion is not always detectable. The CT appearance of the primary tumor is nonspecific, but CT is particularly useful for detecting distant metastases. Pelvic MRI is sometimes used as a problem-solving tool when evaluating myometrial and parametrial tumor invasion (Fig. 7). Choriocarcinoma is usually seen as an intrauterine mass with heterogeneous high signal intensity on T2-weighted imaging and marked peripheral enhancement with central necrosis on postcontrast images. Enhancing parametrial soft tissue is characteristic of local spread. Metastatic disease is also detected by MRI, particularly within the pelvic organs and lymph nodes [40]. The imaging findings of GTD may overlap with those of RPOC, but clinical information of a prior molar pregnancy with a persistently elevated serum β-hCG level strongly suggests GTD. The treatment of GTD is D&C, with chemotherapy used for invasive disease [38, 39].

Conclusions

The presence of RPOC is among the most common causes of both delayed PPH and postabortion hemorrhage, and may occasionally accompany vascular lesions (e.g., AVM, UVM, pseudoaneurysm). Hypervascular RPOC and AVM and UVM should be treated carefully because they may cause life-threatening bleeding, but care should be taken to avoid overdiagnosis of AVM. Subinvolution of the placental site is not a rare cause of secondary PPH, but it is underrecognized by clinicians and categorized as a low-flow UVM. Color Doppler US plays an important role in diagnosing these uterine vascular lesions and may assist in determining the treatment strategy by measuring the PSV. The entity of GTD is in the differential diagnosis for RPOC by radiologic appearance, but is usually excluded by a high serum hCG level.

Compliance with ethical standards

Conflict of interest Sadayuki Murayama received a research grant from Toshiba Medical Systems. The remaining authors declare that they have no conflict of interest.

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