

Transcatheter Arterial Embolization for Postpartum Hemorrhage: Indications, Technique, Results, and Complications

Philippe Soyer · Anthony Dohan · Raphael Dautry ·
Youcef Guerrache · Aude Ricbourg · Etienne Gayat ·
Mourad Boudiaf · Marc Sirol · Olivier Ledref

Received: 2 October 2014 / Accepted: 7 January 2015

© Springer Science+Business Media New York and the Cardiovascular and Interventional Radiological Society of Europe (CIRSE) 2015

Abstract Postpartum hemorrhage (PPH) is a potentially life-threatening condition, which needs multidisciplinary management. Uterine atony represents up to 80 % of all causes of PPH. Transcatheter arterial embolization (TAE) has now a well-established role in the management of severe PPH. TAE allows stopping the bleeding in 90 % of women with severe PPH, obviating surgery. Pledgets of gelatin sponge as torpedoes are commonly used for safe TAE, and coils, glue, and microspheres have been primarily used in specific situations such as arterial rupture, pseudoaneurysm, and arteriovenous fistula. TAE is a minimally invasive procedure with a low rate of complications, which preserves future fertility. Knowledge of causes of PPH, potential risks, and limitations of TAE is essential for a timely decision, optimizing TAE, preventing irreversible complications, avoiding hysterectomy, and ultimately preserving fertility.

Keywords Postpartum hemorrhage · Arterial embolization · Pelvic embolization · Emergency · Review

Introduction

Postpartum hemorrhage (PPH) is responsible for 25 % of maternal deaths worldwide and remains the major cause of emergency peripartum hysterectomy [1, 2]. In developed countries, the frequency of emergency peripartum hysterectomy was around 1/1,000 deliveries in 1990 and dropped to 1/2,000 deliveries in 2010 [3]. This substantial drop is presumably due to early identification of PPH with appropriate initial measures, marked improvements in medical resuscitation, and an increased use of conservative treatments including pelvic artery ligation, uterine compression sutures, balloon tamponade, and percutaneous transcatheter arterial embolization (TAE) [4–10].

P. Soyer (✉) · A. Dohan · R. Dautry · Y. Guerrache ·
M. Boudiaf · M. Sirol · O. Ledref
Department of Abdominal and Interventional Imaging, Hôpital
Lariboisière-AP-HP, 2 rue Ambroise Paré, 75475 Paris Cedex
10, France
e-mail: philippe.soyer@lrb.aphp.fr

A. Dohan
e-mail: anthony.dohan@lrb.aphp.fr

R. Dautry
e-mail: raphael_dautry@yahoo.fr

Y. Guerrache
e-mail: docyoucef05@yahoo.fr

M. Boudiaf
e-mail: mourad.boudiaf@lrb.aphp.fr

M. Sirol
e-mail: marc.siro@lrb.aphp.fr

O. Ledref
e-mail: olivier.ledref@lrb.aphp.fr

P. Soyer · A. Dohan · R. Dautry · E. Gayat
Diderot-Paris 7, Université-Sorbonne Paris-Cité, 10 Avenue de
Verdun, 75010 Paris, France
e-mail: etienne.gayat@lrb.aphp.fr

A. Ricbourg
Department of Obstetrics and Gynecology, Hôpital Lariboisière-
AP-HP, 2 rue Ambroise Paré, 75475 Paris Cedex 10, France
e-mail: aude.ricbourg@lrb.aphp.fr

E. Gayat
Department of Anesthesiology, Hôpital Lariboisière-AP-HP, 2
rue Ambroise Paré, 75475 Paris Cedex 10, France

Consequently, the remaining indications for emergency peripartum hysterectomy in women with PPH include abnormal placental adhesion and, at a lesser degree, failure of conservative treatments and complex uterine rupture [11].

TAE is now a well-accepted procedure for the management of PPH so that this technique should be considered as soon as blood transfusions are initiated. Early reports with limited number of women have shown the potential of this technique that was later confirmed by larger studies [12–19]. Uterine atony accounts for the majority of causes of PPH and is the most common indication for TAE [20]. However, as experience accumulates, other indications for TAE are now well-recognized, including uterine artery pseudoaneurysms [21, 22], invasive placentation [23–27], genital tears [28, 29], and other rarer causes [30–32].

This article presents a comprehensive overview of the current clinical applications of TAE in PPH, current limitations, and future prospects. Conclusions and practical approach to the use of TAE in PPH have been derived from an extensive analysis of the published literature.

General Considerations

Definition and Demographics

Primary or early PPH appears less than 24 h after delivery [14, 33]. Secondary or late PPH occurs more than 24 h, but less than 6–12 weeks after delivery [15]. PPH is defined as a blood loss >500 mL within 24 h after vaginal delivery and >1,000 mL in case of cesarean delivery [1–4, 33]. Severe PPH is defined as a life-threatening blood loss with hemodynamic consequences that need unusual therapy [1–4, 14]. PPH and severe PPH represent 6 and 1.86 % of all deliveries, respectively [3]. However, PPH is often underestimated because of difficulties in estimating blood loss and also because the bleeding can be missed in the absence of external bleeding in case of intra- or retroperitoneal bleeding or also in case of vaginal hematoma [29, 32, 34]. In developed countries, PPH represents 60 % of all maternal deaths [3] and is the main cause of peripartum hysterectomy [1, 35].

Causes and Risk Factors of PPH

The main causes of primary PPH are uterine atony (representing approximately 80 % of PPH) and trauma or laceration of the lower portion of the genital tract (representing approximately 5 % of PPH) [36]. Other causes of primary PPH include cesarean section, invasive placentation, congenital or acquired coagulation disorders, uterine

rupture or inversion, bladder flap hematoma, retention of blood clots or placental fragments, and fibroids [13, 24, 31, 36]. The main causes of secondary PPH are retained placenta, abnormal placentation, uterine subinvolution, coagulopathies, and ruptured pseudoaneurysm [21, 22, 34, 37]. Pseudoaneurysms are usually located on the uterine artery after cesarean section and on the lower genital arterial tree after vaginal delivery [21, 22].

Acknowledged risk factors for PPH include high parity, fetomaternal dystocia, multifetal pregnancy, hydramnios, uterine leiomyoma, abnormal placentation, and prior history of PPH [2, 4, 38–40]. However, most women with PPH have no risk factors so that the occurrence of PPH is hardly predictable [38].

Anatomy and Uteroplacental Vascularization

Vascular Anatomy of the Pelvis

Uterine and cervicovaginal vascularization is subjected to multiple variations that may have impact on the TAE procedure itself.

The internal iliac artery (IIA) divides into two main stems. The posterior division of the IIA gives rise to the iliolumbar, the lateral sacral, and the superior gluteal arteries, which are parietal arteries. The anterior division of the IIA gives rise to parietal and visceral branches. Visceral branches include uterine, vaginal, superior vesicle, and internal pudendal arteries. Parietal branches of the anterior division of the IIA include obturator and inferior gluteal arteries [41, 42]. A complex anastomotic network exists between IIA and mesenteric arteries, internal and external iliac arteries, and anterior and posterior trunks of the IIA [41–43].

Vaginal arteries arise from the anterior division of the IIA just below the origin of the uterine artery or from the uterine artery itself [41]. Small cervicovaginal branches originating from the uterine arteries give blood supply to the cervix. The vaginal artery distributes to the anterior and lateral surface of the vagina. Branches from the inferior bladder artery supply the middle portion of the vagina, and the internal pudendal artery supplies its lower portion, the posterior surface being vascularized by the middle rectal artery. A continuous arcade on the lateral borders of the vagina, uterus, and adnexa forms a complex arterial network [43].

Uterine Arterial Network

The uterus is predominantly vascularized by the uterine arteries, which penetrate the lateral aspect of the uterus at the level of the isthmo-corporeal junction [41]. Then, the uterine arteries divide into two main branches; one ascending branch giving blood supply to the myometrium and one descending branch giving blood supply to the



Fig. 1 A 37-year-old woman with postpartum hemorrhage due to uterine atony. Selective angiogram of anterior trunk of left internal iliac artery in left anterior oblique projection (25°) shows left uterine artery (arrows) with a typical tortuous shape, which divides into two branches. One branch extends cranially (arrowheads) and gives blood supply to the myometrium and the other runs caudally (curved arrow) and gives blood supply to the cervix and upper segment of vagina

cervix and portions of the vagina (Fig. 1) [36]. Arcuate arteries originate from uterine arteries and have a circumferential path around the myometrium and cross-sided anastomosis. Ovarian and round ligament arteries often participate to a complex anastomotic network that gives blood supply to the uterus [43]. The ovarian arteries usually arise from the abdominal aorta below the renal artery (Fig. 2) [36, 44]. The round ligament artery originates from the inferior epigastric artery or femoral artery so that it can be visible when a flush abdominal aortogram is performed [42, 43]. Most of the time, when selective angiography of the internal iliac artery is performed, round ligament artery is only visible on late pelvic angiographic images and often missed (Fig. 3). Collaterals to the uterus from ovarian artery, inferior mesenteric artery, round ligament artery, and internal pudendal artery are observed on angiography in 3.8, 1.3, 0.2, and 0.2 % of patients, respectively [44]. Depiction of these arteries is of importance because in some women, they may actively participate to the uterine arterial supply and may be a cause of failed TAE [44].

Anatomical Variations

The vascularization of the uterus is subjected to many variations [37, 41]. Of these, persistent sciatic artery, which



Fig. 2 A 31-year-old woman with postpartum hemorrhage due to uterine atony presenting with repeat bleeding after a first session of TAE. Control angiogram in frontal projection with the catheter (arrows) selectively placed in left uterine artery shows ovarian artery (arrowheads) with a typical tortuous appearance that has anastomotic connections (curved arrow) with left uterine artery and perfusion of the uterus, explaining repeat bleeding. Selective TAE of left ovarian artery was performed with gelatin sponge pledgets, thus stopping the bleeding

originates from the anterior division of the IIA and provides the major part of the arterial blood flow to the ipsilateral inferior limb, is of importance [45]. Timely identification of this rare variation is crucial to prevent irreversible ischemic damage of the lower limbs in case of TAE of uterine arteries [45]. Because of the potential risk, TAE should be restricted to the uterine arteries only should the anatomical distribution allows a safe TAE [45].

In case of a persistent sciatic artery, the external iliac artery has a small caliber, which should be an alerting finding, and the IIA has a markedly enlarged diameter (Fig. 4). At a lower level, angiography shows sciatic arteries coursing through the posterior aspect of the thighs and continuing as popliteal arteries [45]. The uterine arteries originate directly at the anterior aspect of the internal iliac artery, below the origin of the superior gluteal artery [45].

If the uterine arteries can be safely catheterized with substantial progression of the catheter, so that reflux of embolic material cannot occur, TAE may be considered with caution and performed by a well-trained interventional radiologist. Although there is no case of PPH with persisting sciatic artery treated by TAE, because of the

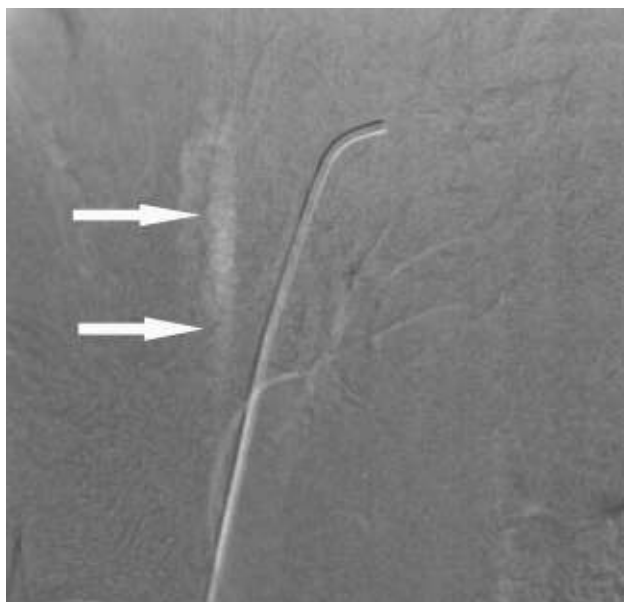


Fig. 3 A 35-year-old woman with postpartum hemorrhage due to uterine atony. Control pelvic angiogram of right external iliac artery in frontal projection during late phase shows right round ligament artery (*arrows*), which has a characteristic sinuous appearance. Of note, no contrast material is visible in the iliac arteries during this late phase

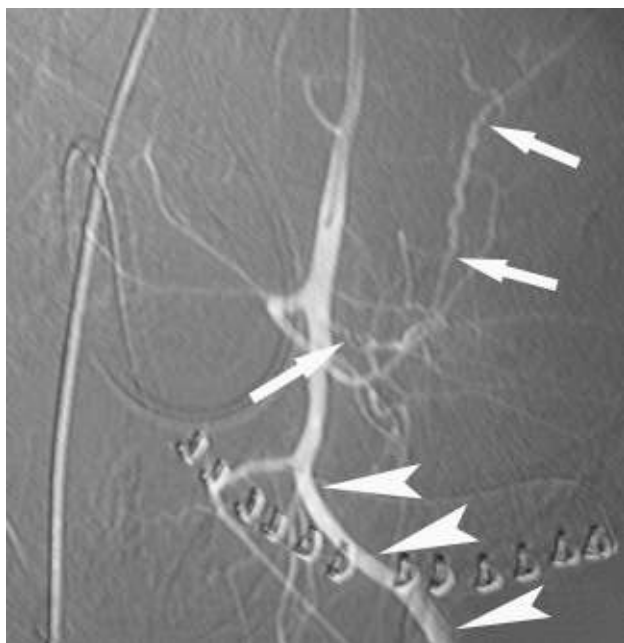


Fig. 4 A 19-year-old woman with primary postpartum hemorrhage after cesarean section. Angiogram obtained during selective catheterization of the left internal iliac artery shows tiny uterine artery (*arrows*) originating 1 cm below the origin of the superior gluteal artery. Persistent sciatic artery is clearly seen (*arrowheads*)

potential risk and catastrophic damage that untargeted embolic material may cause, hysterectomy should be the preferred option when TAE cannot be performed safely.

Multidisciplinary Management

Obstetrical and Medical Management

Obstetrical management includes vaginal inspection, manual uterine examination, external uterine massage, and suturing of soft-tissue injuries. Vaginal inspection must be performed after vaginal delivery and when a dilated cervix is present. These measures help exclude uterine rupture, which needs surgery, or other causes of PPH requiring specific medical or obstetrical treatment and also confirm uterine vacuity [3]. These measures have also to be performed when the woman is transferred to another institution for possible TAE. Bakri balloon can be used for uterine atony with well-established efficacy, although its other indications are not clearly defined yet [7].

Medical management of PPH includes resuscitation, blood transfusion, and administration of uterotonic drugs such as oxytocin and prostaglandin analogs used in conjunction with external massage of the uterus, bladder catheterization, and bimanual uterine compression [46].

Ultrasonography is performed to depict pelvic fluid that usually indicates hemoperitoneum, which mandates surgical evacuation [47]. In women with secondary PPH, computed tomography (CT) or magnetic resonance (MR) imaging helps depict the cause and location of the bleeding, which can be due to pseudoaneurysm or retained placental tissue, although ultrasonography fulfills that task in the majority of cases [36, 48]. CT and MR imaging may also depict extra-uterine sources of bleeding including rectus sheath hematoma or direct arterial injuries. In general, CT and MR imaging are used when PPH does not respond favorably to standard treatment and when there is no hemodynamic instability [36, 49, 50]. CT has an important role when intra-abdominal bleeding is suspected, in case of recurrent bleeding after TAE or in secondary PPH, as well as for the evaluation of postsurgical complications, although its use may be restricted to specific situations depending on local policies or hemodynamical status [48–50].

Surgical and Interventional Radiological Management

Invasive treatment is indicated when persisting hemorrhage is present in spite of appropriate medical and obstetrical treatment [46]. Surgical arterial ligation, uterine compression suture, TAE, or hemostatic hysterectomy can be used depending on the clinical presentation [5, 51–53]. Surgery is preferred when uterine rupture or bladder injuries are suspected [5, 52]. Arterial ligation is indicated after cesarean section as a first line option [53], but TAE is often preferred at many facilities where TAE is available 24 h a day and seven days a week [5, 54]. TAE should be

preferred to other techniques after vaginal birth, in secondary PPH or when coagulation disorders are present [5, 14, 15, 28].

Transcatheter Arterial Embolization

Practical Considerations

PPH is a severe condition, which needs rapid and effective control. Failure to rapidly control the bleeding results in severe coagulation abnormalities with intravascular disseminated coagulation and increased morbidity [1, 2]. Therefore, TAE must be performed rapidly and its use should be considered immediately when blood transfusion has started [14, 28, 33]. One study has reported the use of TAE in the surgical operating room to reduce delay in management [55], but in the majority of studies, TAE was performed in the interventional radiology room [14, 15, 17, 18, 21–24, 27, 29, 33, 40].

Ideally, TAE should be performed at least 30 min after prostaglandin analog administration has been stopped because these drugs induce a marked spasm of the uterine arteries, thus preventing satisfactory arterial occlusion [14, 21, 22, 28]. In this regard, arterial spasm during TAE constitutes a cause of repeat bleeding [56]. Because non-target embolization may have severe consequences, the embolization itself must be performed under fluoroscopic control [57].

A bleeding site with extravasation of iodinated contrast material on angiography is usually visible in approximately one third of the women [14, 18]. However, this frequency may reach up to 52 % [17]. In general, in women with PPH due to uterine atony, no bleeding site is visible, whereas a bleeding site is more frequently observed after traumatic vaginal delivery [21, 22, 28].

The mean overall procedure time for TAE in PPH greatly varies among operators ranging from 59 to 270 min, with a mean time extracted from several studies of 90 min [13, 14, 24].

Vascular Approach

A single puncture of the right common femoral artery allows catheterization of left and then right pelvic arteries using a Waltman loop [37, 58–60]. Ultrasonographic guidance may be needed for femoral artery puncture. A 5-F introducer with a 5-F cobra-shaped end-hole catheter is used in the majority of studies but there is no definite option regarding the diameter of the catheter. In this regard, a 4-F catheter can be used in case of major iliac spasm to avoid complete occlusion of right iliac artery and is the favored catheter for some operators. TAE for PPH with a single femoral approach and a 5-F cobra-shaped catheter is

successful in 97 %, whereas a coaxial 3-F microcatheter is needed in only 3 % of TAE procedures [58]. Microcatheters, which would preclude using pledgets of gelatin sponge, should not be used as a first line option, but only when hyperselective embolization is needed.

Many authors advocate selective embolization of uterine arteries only when possible, whereas others suggest that embolization of the anterior division of IIA can be performed safely with the advantage of being a faster procedure with no impact on the effectiveness of TAE [14, 15, 17, 37]. Finally, TAE must be bilateral because of cross-sided anastomosis between the two uterine arteries so that unilateral TAE may fail to stop the bleeding (Fig. 5) [14, 15, 37, 41].

It may be assumed that arterial closure devices may be useful in women with severe coagulation abnormalities, but their use has not been specifically reported in TAE for PPH [61].

Temporary and Permanent Occluding Agents

Conceptually, occluding (or embolic) agents can be classified as permanent or temporary. Temporary agents include gelatin sponge [60, 62] and homologous blood clots. Permanent embolic agents include metallic coils, poly-vinyl alcohol (PVA) particles, *n*-butyl-cyanoacrylate, and microspheres [63, 64].



Fig. 5 A 36-year-old woman with postpartum hemorrhage due to uterine atony. Selective angiogram of anterior trunk of right internal iliac artery obtained in frontal projection shows right uterine artery (arrow) and multiple cross-over anastomoses (arrowheads) with previously embolized left uterine artery. Because of cross-sided anastomosis, TAE must be bilateral

Home-made and hand-prepared pledgets of gelatin sponge as torpedoes are used by many authors [14, 15, 21, 22, 24, 28, 33, 37]. A fluid form of gelatin sponge (i.e., slurry) can be obtained by mixing tiny cubicles with iodinated contrast material [65]. This form should be used in women with retained placenta to induce thrombosis of intervillous spaces [24, 66]. The use of slurry of gelatin sponge should be avoided in other situations because of reported cases of uterine necrosis with this specific material [60, 67, 68], although some authors have reported safe and effective TAE with slurry [65].

Metallic coils are rarely needed. They can be used to occlude ruptured pseudoaneurysms, although their superiority over gelatin sponge pledgets or torpedoes is not clearly demonstrated [21, 22]. Coils can be used to perform sac embolization only, parent artery occlusion with inflow/outflow vessel occlusion or sac embolization along with parent artery occlusion with inflow/outflow vessel occlusion [69].

The use of PVA particles is not recommended as they may cause uterine necrosis [70]. The use of micro particles in case of PPH due to invasive placenta left in place after delivery is more debated because even the largest available microparticles may pass through arteriovenous shuntings and may escape into the systemic circulation [24, 66].

The use of cyanoacrylate glue (*n*-butyl-2-cyanoacrylate) should be restricted to pseudoaneurysm, arterial rupture, or arteriovenous fistulas, because this embolic agent has demonstrated effectiveness in such indications [15, 21, 71].

Radiation Exposure

In most women with PPH, the bleeding originates from the uterine arteries or one of their branches. A global pelvic angiogram is performed in specific cases when women have had prior pelvic arterial ligation, hemostatic hysterectomy, persistent bleeding after TAE, and secondary PPH, because in such situations, arterial anastomotic routes can be present. In case of uterine atony, an anterior division iliac angiogram is sufficient to determine the vascular anatomy of the pelvis.

TAE results in non-negligible mean estimated absorbed ovarian dose of up to 58.6 cGy (range 20–73 cGy) [72]. However, radiation during TAE greatly depends upon the procedure protocol [73]. In this regard, when using low-frequency pulsed fluoroscopy with limited use of magnified and oblique fluoroscopy, the mean estimated absorbed ovarian dose drops dramatically to 9.5 cGy (range 2.21–23.21 cGy), and the mean absorbed skin dose is 47.69 cGy (range 10.83–110.14 cGy) [73]. To decrease the radiation dose, the field of view, fluoroscopy time, and number of acquisitions should be minimized. A pulsed emission ≤ 7.5 frames/s for arterial catheterization, a

pulsed rate ≤ 4 frames/s to monitor TAE, and beam in anterior-posterior projection help minimize radiation exposure [74].

General Results and Causes of Failure

The overall success rate of TAE for stopping the bleeding in women with PPH is 86.5–96.3 % [14–16, 18, 67]. One study reported a success rate of 100 % in secondary PPH [15]. The primary success rate (i.e., when the bleeding stops after a single session of TAE) was 86.45 % (217/251 women) in the largest study published so far [18]. In the same study, the secondary success rate (i.e., when the bleeding stops after a single session of TAE) was 90.04 % (226/251 women) [18] and 96 % in other two studies [14, 17]. Of interest, repeat TAE after a failed first session of TAE has a success rate ranging from 67 to 80 % [14, 17, 60], with a success rate of 75 % in the study by Lee et al. [18]. A systematic review including 46 studies using a cumulative outcome method found an overall success rate of 90.7 % (95 % CI 85.7–94.0 %) for TAE [52].

Criteria of failed TAE include disseminated intravascular coagulation [18, 19, 75, 76], major transfusion (i.e., transfusion >5 or 10 red blood cell packs), blood loss $>1.5L$ [18, 19, 40], severe arterial vasoconstriction at the time of TAE [56], and invasive placenta, which has a failure rate of approximately 20 % [24, 25, 67]. It has been suggested that repeat TAE may be more frequently needed in women with active extravasation at initial angiography [60].

Sporadically, other specific causes of failed TAE include an ancillary uterine blood supply from an ovarian artery not seen at angiography or not embolizable [14, 17, 77] and performance of unilateral TAE [41].

Complications following TAE are observed in approximately 3 %, consisting predominantly in minor complications such as hematoma at the puncture site, dissection of the uterine artery, transient sciatic nerve paresis, and synchias [18, 40, 52]. Major complications such as uterine necrosis requiring hysterectomy [19, 68, 78, 79] or non-target embolization with retrograde migration of gelatin particles into the external iliac artery and acute occlusion of limb arteries requiring embolectomy [76] are extremely rare. Bladder necrosis has also been reported in case of TAE using gelatin sponge pledgets in a woman with placenta accreta [23]. Vesicovaginal fistula requiring surgical repair has also been described but the actual role of TAE as a causing factor is unclear [77]. Non-clinically significant intramyometrial hematoma has been described after TAE using gelatin sponge slurry [65].

Fever can be observed after TAE, but usually indicates a complication not related to TAE. Fever suggests endometritis that usually resolves with antibiotic therapy alone [13]

or infected pelvic hematoma requiring vaginal or percutaneous drainage [13]. In women with unexplained fever after TAE, CT should be performed to exclude an abscess or an infected hematoma.

Effects on Fertility and Menses

Although researchers have described transient ovarian failure after TAE [73, 80], many reports showed that women who undergo TAE can expect to have a return of normal menses with no adverse effect on fertility in 91–100 % of the cases [17, 39, 54, 72, 81–84]. In one study involving 20 women, 4 had a total of five full-term and two preterm pregnancies, and all delivered healthy infants by cesarean section with no recurrence of PPH [72]. Salomon et al. [39] have reported pregnancies in 5 out of 17 women who were followed up after TAE. Sentilhes et al. [82] have reported normal gestation in 19 women after TAE. Fiori et al. [83] have reported pregnancies in 38.3 % of women after TAE for PPH.

More recently, two studies have reported regular menstrual cycles in 110/113 women (97.3 %) and 176/176 women (100 %) who had TAE for PPH, and normal pregnancies in approximately 10 % of them [18, 85].

Clinical Setting and Technical Approach

Uterine Atony

Uterine atony results from uterine overdistension due to multiple pregnancies, polyhydramnios, or fetal macrosomia. Other causes include myometrial laxity secondary to multiparity, prolonged labor, and general anesthesia. Uterine atony is found in 70–80 % of PPH [17, 39, 86] and is the indication for which TAE has best results.

Angiography shows dilated uterine and arcuate arteries. On frontal projection, these arteries extend cephalad to the aortic bifurcation. There is no visible extravasation of contrast material agent because the bleeding is diffuse [33, 37, 57].

Torpedoes of gelatin sponge are the preferred occluding agents. When the common trunk of the IIA is occluded, caution must be given to not occlude the contralateral common trunk and the sacral lateral arteries to avoid sciatic nerve ischemia.

A global conclusion angiogram with delayed imaging helps assure that no distal recanalization of uterine arteries through patent collaterals has occurred after TAE. Should it be the case, further and more radical TAE must be performed to avoid repeat bleeding [14]. Peripheral arterial blood pressure rises soon or even immediately after TAE, and the rise or return to normal values of peripheral arterial

blood pressure is a strong indicator of success. Inspection and manual maneuvers confirm that external bleeding stops and the uterus usually becomes well retracted soon after TAE. Hemostatic function and platelet count return to normal values within the 24 h following successful TAE [14].

Genital Tract Laceration

Genital tract lacerations include perineal tears and cervical or vaginal lacerations. Lacerations are found in 0.2 % all vaginal deliveries [87] and may be the cause of bleeding in up to 11 % of PPH, alone or in association with atony [39]. Bleeding from vaginal or cervical laceration is generally controlled with surgical repair and vaginal packing, which should be performed first. Most severe lesions are due to instrumental extraction, mostly when spatulas are used. Failed suturing may result in severe bleeding, thus increasing the impaired coagulation status. In such case, TAE stops the bleeding and helps perform subsequent suturing in up to 100 % of women [28, 29].

Post-traumatic bleeding following vaginal delivery is related to arterial rupture, which can occasionally lead to formation of a pseudoaneurysm. The most frequent injured arteries are internal pudendal and vaginal arteries [17]. In some cases, rupture of pseudoaneurysm may occur late after delivery, thus causing secondary PPH [21]. Angiography frequently shows direct extravasation of contrast material, mostly from vaginal arteries [17, 21, 22, 28, 29].

The main goal of TAE in PPH due to genital tears is to stop the bleeding and perform subsequent suturing. Occlusion of the bleeding vessel is usually obtained with gelatin sponge pledgets [28, 29]. When no extravasation is visible or if the bleeding is located too distally precluding selective catheterization of the injured artery, gelatin sponge pledgets are used to selectively and bilaterally occlude vaginal arteries and uterine arteries and/or the anterior division of the IIAs.

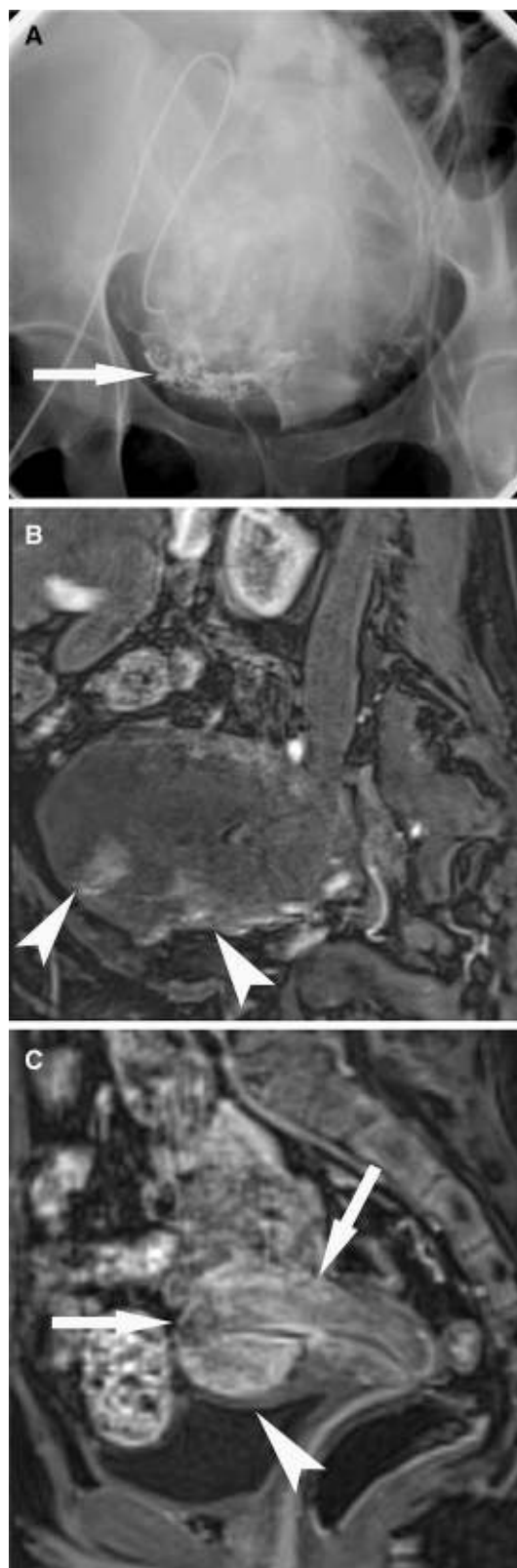
Metallic coils are efficient to treat direct arterial rupture when extravasation from a pseudoaneurysm is visible. However, their use should be restricted to failed TAE with gelatin sponge pledgets [21].

Retained and Invasive Placenta

Abnormal placental adhesions lead to primary PPH in 50 % of the cases [88]. Remaining placental tissue prevents adequate contraction and predisposes to excessive bleeding. Secondary PPH associated with abnormal placentation can be explained by a secondary spontaneous placental detachment, which causes bleeding.

In the setting of PPH, distinguishing retained placental tissue from clots related to normal lochia can be clinically

Fig. 6 A 37-year-old woman with postpartum hemorrhage due to placenta percreta with bladder involvement that was diagnosed during pregnancy before delivery on MR imaging. **A** Selective angiogram of right uterine artery shows multiple, tortuous, irregular, and enlarged branches (*arrow*) of right vesical artery that gives blood supply to the placenta. This appearance is typical of placenta percreta with bladder involvement. **B** Magnetic resonance imaging in sagittal plane obtained with a dynamic T1-weighted sequence after intravenous administration of a gadolinium chelate shows poor and peripheral placental enhancement (*arrowheads*) 1 day after bilateral TAE using pledgets of gelatin sponge. **C** 6 months after TAE, magnetic resonance imaging obtained in the sagittal plane after intravenous administration of a gadolinium chelate shows return to normal uterus (*arrows*) with complete resorption of the placenta. The interface between uterus and bladder shows normal enhancement with no visible enlarged vessels



challenging so that imaging is often required. Ultrasonography can show a thickened endometrium or a discrete intrauterine mass with increased vascularity at color or power Doppler ultrasonography. Assessment of residual vascularization after TAE and remaining placental volume is made with MR imaging (Fig. 6) [66].

In women with invasive placenta, TAE has a success rate of approximately 80 % [23–27], with 20 % of women requiring further surgery and hysterectomy [23, 27, 88] and a complication rate of 11 %, including uterine necrosis, endometritis, and synechiae [88, 89].

Some authors advocate placement of arterial catheters before delivery because of the possibility of cataclysmic hemorrhage [90, 91]. However, because of the risk of catheter displacement and because less than 50 % of women with invasive placenta actually require TAE, this approach is not recommended [90]. Recent national guidelines do not recommend prophylactic balloon catheter placement [92] because this approach conveys a risk of permanent sciatic nerve ischemia, arterial dissection, arterial rupture, and inferior leg ischemia [93–95]. Should prophylactic balloon catheters be used, they should be placed in the common iliac arteries instead of IIAs [96].

Angiographic features depend on the amount of retained placenta the degree of invasiveness and the number of recruited arteries, which may be uterine, vesical, pudendal, or obturator arteries. On angiographic examination, blood flow into the intervillous spaces presents as a continuous stream of contrast material [24] and multiple, tortuous and enlarged arteries are visible (Fig. 6).

TAE is aimed at stopping the bleeding and inducing blood vessel thrombosis and ischemia of retained placenta. Arteries to the placenta can be first occluded using slurry. Uterine arteries and anterior and posterior divisions of the IIA have to be occluded with gelatin sponge pledgets. TAE

helps reduce the time for complete placental resorption when conservative management is preferred [66]. Microspheres must be used with caution and those of largest available diameter should be preferred [24].

PPH and Uterine Inversion

Uterine inversion is an unpredictable emergency, occurring in 0.0002 % of all deliveries, which may lead to PPH [31]. If missed, it may lead to maternal death [30]. Uterine inversion is due to a premature traction of the umbilical cord before placental separation during the third stage of labor. Conservative treatment consists in uterine repositioning and TAE in case of bleeding [60].

Angiography shows abnormal position and path of the uterine arteries. In general, no active bleeding is visible, a finding similar to that observed in uterine atony (Fig. 7) [31].

TAE should be performed with gelatin sponge pledgets as torpedoes after uterine repositioning. In some instances, uterine inversion may be missed and the diagnosis is made at angiography during TAE (Fig. 7).

PPH and Pseudoaneurysm

The incidence of pseudoaneurysm in women with PPH is 3.06 % [21], but they can be present in the absence of PPH, underestimating their actual incidence [97]. Pseudoaneurysms are predominantly found after cesarean section (Fig. 8) [98] and can result in primary or secondary PPH [97]. Three-dimensional CT angiography helps demonstrate precise location and feeding vessels of pseudoaneurysm and plane TAE [36].

Angiography reveals extravasation in 50 % of cases [21, 22]. Pseudoaneurysms most frequently arise from internal pudendal arteries, cervical branches of the uterine artery or on vaginal arteries after vaginal birth, and on the uterine arteries after cesarean section [21, 99].

TAE is usually performed using gelatin sponge pledgets alone [21, 22]. A complete occlusion can also be obtained with metallic coils or *n*-butyl-2-cyanoacrylate in specific cases [21, 72]. One advantage of metallic coils is that, in theory, they can be used to fill the pseudoaneurysm only, thus preserving the parent artery [67]. However, in clinical practice, such selective occlusion is rarely feasible.

PPH after Cesarean Section

Cesarean section represents 25 % of all deliveries [85, 100]. Intraoperative damages to pelvic organs and severe bleeding are the most serious complications of cesarean delivery. Ligation is the recommended first line option for

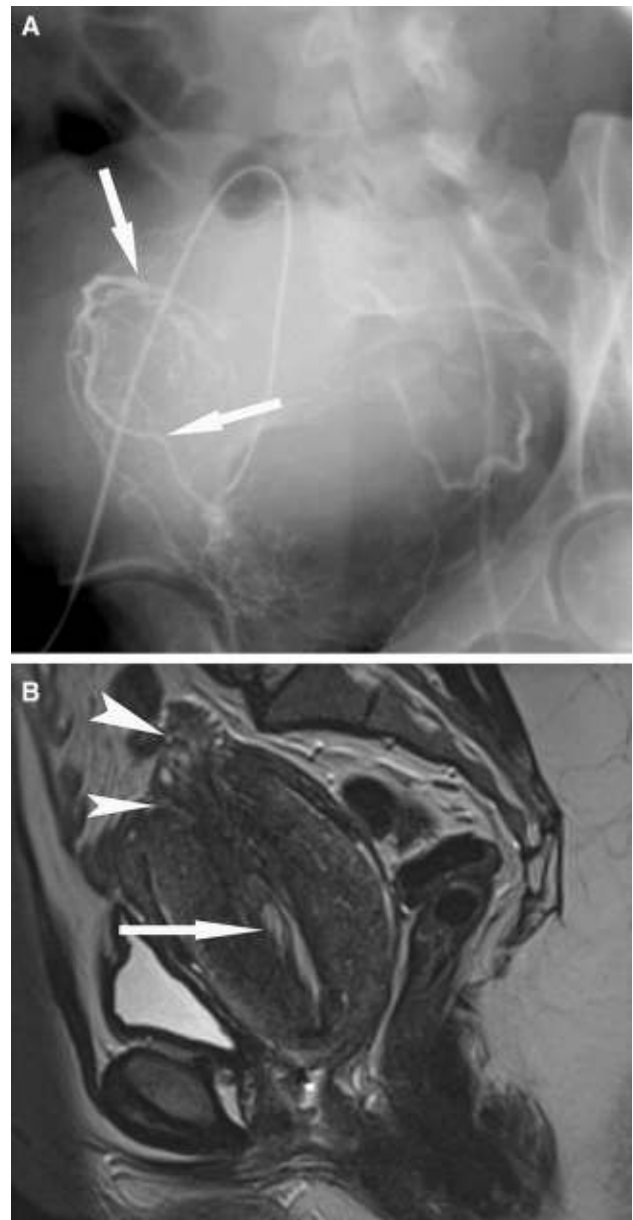


Fig. 7 A 30-year-old woman with primary postpartum hemorrhage due to uterine inversion. **A** Selective angiogram of right uterine artery in right anterior oblique projection shows right uterine artery (arrows). The uterine artery is abnormally displaced compared to the usual appearance. **B** Magnetic resonance imaging obtained in the sagittal plane shows uterine pedicle in central location (arrow), and the cervix is at the superior aspect of the uterus (arrowheads), thus confirming uterine inversion

primary PPH after cesarean section [5] but TAE should be strongly considered when PPH occurs while the woman is in the recovery room or in the postnatal ward [59, 92]. This is why women with PPH following cesarean section represent 26–43.8 % of women treated with TAE [14, 18].

After cesarean section, primary PPH is due to uterine atony, direct arterial injury, or abnormal placental

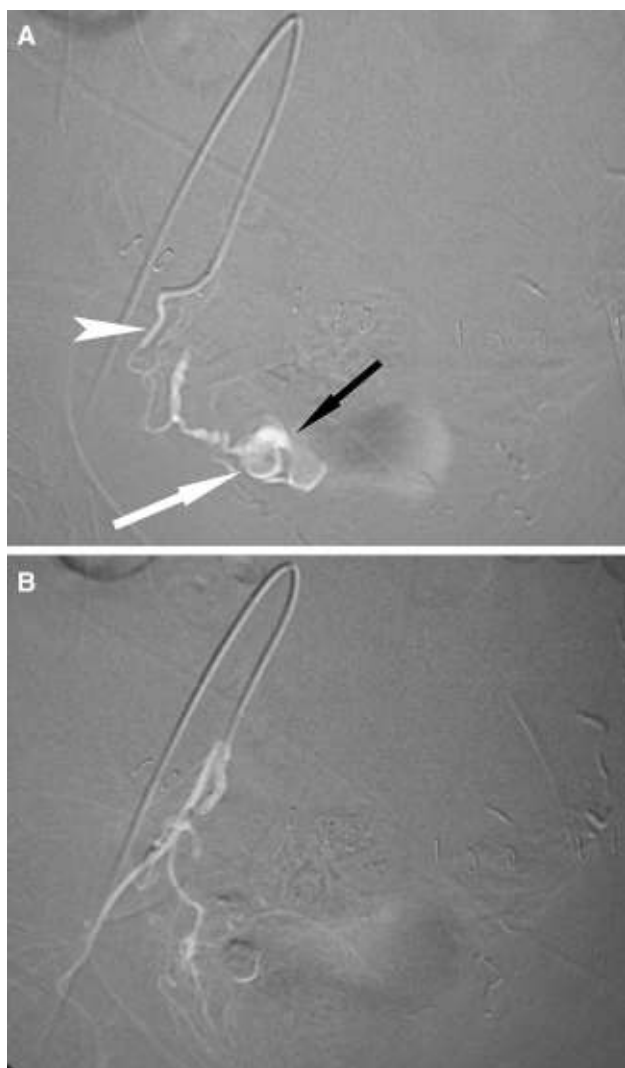


Fig. 8 A 35-year-old woman with primary postpartum hemorrhage after cesarean section. **A** Selective angiogram of right uterine artery (arrowhead) in frontal projection (0°) shows pseudoaneurysm (white arrow). Direct extravasation of iodinated contrast material is present (black arrow), indicating arterial injury. **B** Control angiogram in frontal projection (0°) after embolization of right uterine artery with gelatin sponge pledgets shows no persisting opacification of the pseudoaneurysm. No recurrent bleeding was observed during the follow-up

adhesion, whereas secondary PPH is frequently due to spontaneous pseudoaneurysm rupture [21, 22]. When present, a pseudoaneurysm may show variable size and is connected to the uterine artery or to one of its branches (Fig. 8) [21]. Pseudoaneurysm can be treated surgically but the endovascular approach is the favored one because of a high success rate [21, 22].

Persistent PPH after Surgical Arterial Ligation

Surgical arterial ligation has a success rate of 40–100 % [5]. Ligation involves internal iliac, uterine, ovarian, and

round ligament arteries. When arterial ligation fails to stop the bleeding, TAE or hysterectomy should be promptly performed. Ligation often results in multiple and complex anastomotic arterial routes [99].

Angiography can reveal incomplete and ineffective ligation, thus explaining persisting bleeding [14, 99]. Angiography can also show multiple collateral vessels including middle rectal, iliolumbar, or lumbar arteries that participate to uterine vascularization [99].

After ligation of the IIAs, there is a short artery stump that renders embolic agent deposit risky with a theoretical risk of reflux (Fig. 9) [99]. The anastomotic vessels are usually occluded with gelatin sponge pledgets but metallic coils may be needed in selected cases and especially when the use of a microcatheter is required [99]. In case of incomplete ligation with residual arterial lumen, collateral routes are often absent and TAE can be safely performed through incompletely ligated arteries using gelatin sponge torpedoes [99].

Repeat Bleeding after TAE

Repeat bleeding after TAE is observed in 5–10 % [14, 15, 17, 18]. Identified causes of repeat bleeding after TAE include arterial spasm [56], collateral vessels, and invasive placenta [23–25]. In case of repeat bleeding after a first



Fig. 9 A 35-year-old woman with primary postpartum hemorrhage after vaginal delivery. The woman had first ligation of right and left internal iliac arteries at the referring institution but was referred to us because of persisting bleeding. Selective angiogram of right internal iliac artery in frontal projection shows complete ligation (arrow) of right internal iliac artery. However, a newly developed anastomotic network involving the right iliolumbar artery (curved arrow) gives blood supply to the right uterine artery (arrowheads) through the anterior trunk of the right iliac artery. Of note, the posterior trunk of right internal iliac artery is not visible, suggesting inadvertent ligation

TAE, angiography often shows reopening of uterine arteries.

If uterine arteries are patent, repeat TAE is needed with gelatin sponge as pledgets or torpedoes. If uterine arteries are totally occluded, a flush abdominal aortogram should be made to determine the presence of spontaneous arterial anastomoses. It is important to perform a global aortogram with the distal tip of a pigtail catheter above the origin of the renal arteries to depict, if any, enlarged and opacified ovarian arteries. The angiogram can show multiple collateral vessels including middle rectal, iliolumbar, lumbar, or other arteries, such as inferior mesenteric artery, that participate in the vascularization of the uterus. More rarely, a round ligament artery or ovarian artery can be seen and thus explain repeat bleeding (Fig. 2) [101, 102]. Occlusion of the round ligament artery or ovarian artery often requires the use of a microcatheter so that metallic coils are needed [102].

Conclusion

PPH is a life-threatening condition, which needs a multidisciplinary management including gynecologists, anesthesiologists, and interventional radiologists. Knowledge of obstetrical and surgical history of the patient with PPH is mandatory to anticipate the most probable cause of PPH. TAE must not be delayed to avoid or limit complications due to severe coagulation disorders. Emergency TAE allows stopping the bleeding in the majority of women, even after failed surgery. Pledgets of gelatin sponge as torpedoes are effective in the majority of cases and should be the favored option for a safe TAE [65]. Metallic coils and microspheres should be restricted to specific cases.

Conflict of interest Philippe Soyer, MD, PhD, Anthony Dohan, MD, BSc, Raphael Dautr MD, Youcef Guerrache, MD, Aude Ricbourg, MD, Etienne Gayat, MD, PhD, Mourad Boudiaf, MD, Marc Sirol, MD, PhD, and Olivier Ledref, MD have no conflicts of interest.

Statement of Human Rights All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Statement of Informed Consent Informed consent was obtained from all individual participants included in the study.

References

- Khan KS, Wojdyla D, Say L, Gulmezoglu AM, Van Look PF (2006) WHO analysis of causes of maternal death: a systematic review. *Lancet* 367:1066–1074
- Al-Zigri I, Vangen S, Forsen L, Stray-Pedersen B (2008) Prevalence and risk factors of severe postpartum obstetric hemorrhage. *BJOG* 115:1265–1272
- Sheldon W, Blum J, Vogel J, Souza J, Gülmezoglu A, Winikoff B, WHO Multicountry Survey on Maternal and Newborn Health Research Network (2014) Postpartum haemorrhage management, risks, and maternal outcomes: findings from the World Health Organization Multicountry Survey on Maternal and Newborn Health. *BJOG* 121(Suppl 1):5–13
- Briley A, Seed P, Tydeman G, Ballard H, Waterstone M, Sandall J, Poston L, Tribe R, Bewley S (2014) Reporting errors, incidence and risk factors for postpartum haemorrhage and progression to severe PPH: a prospective observational study. *BJOG*. doi:10.1111/1471-0528.12588
- Morel O, Malartic C, Muhlstein J, Gayat E, Judlin P, Soyer P, Barranger E (2011) Pelvic arterial ligations for severe postpartum hemorrhage: indications and techniques. *J Visc Surg* 148:e95–e102
- Kaya B, Tuten A, Daglar K, Misirlioglu M, Polat M, Yildirim Y, Unal O, Kilic GS, Guralp O (2014) Balloon tamponade for the management of postpartum uterine hemorrhage. *J Perinat Med*. doi:10.1515/jpm-2013-0336
- Wright CE, Chauhan SP, Abuhamad AZ (2014) Bakri balloon in the management of postpartum hemorrhage: a review. *Am J Perinatol*. doi:10.1055/s-0034-1372422
- Liu S, Mathur M, Tagore S (2014) Complications and pregnancy outcome following uterine compression suture for postpartum haemorrhage: a single centre experience. *J Obstet Gynaecol*. doi:10.3109/01443615.2014.895309
- Joshi VM, Otiv SR, Majumber R, Nikam YA, Shrivastava M (2007) Internal iliac artery ligation for arresting postpartum haemorrhage. *BJOG* 114:356–361
- Keriakos R, Mukhopadhyay A (2006) The use of the Rusch balloon for management of severe postpartum haemorrhage. *J Obstet Gynaecol* 26:335–338
- Rossi AC, Lee RH, Chmait RH (2010) Emergency postpartum hysterectomy for uncontrolled postpartum bleeding: a systematic review. *Obstet Gynecol* 115:637–644
- Brown BJ, Heaston DK, Poulson AM, Gabert HA, Mineau DE, Miller FJ (1979) Uncontrollable postpartum bleeding: a new approach to hemostasis through angiographic arterial embolization. *Obstet Gynecol* 54:361–365
- Moore TR, Resnik R, Doemeny J, Chin H, Bookstein JJ (1992) Angiographic embolization in the management of hemorrhagic complications of pregnancy. *Am J Obstet Gynecol* 166:493–497
- Pelage JP, Le Dref O, Mateo J, Soyer P, Jacob D, Kardache M, Dahan H, Repiquet D, Payen D, Truc JB, Merland JJ, Rymer R (1998) Life-threatening primary postpartum hemorrhage: treatment with emergency selective arterial embolization. *Radiology* 208:359–362
- Pelage JP, Soyer P, Repiquet D, Herbretreau D, Le Dref O, Houdart E, Jacob D, Kardache M, Schurando P, Truc JB, Rymer R (1999) Secondary postpartum haemorrhage: treatment with selective arterial embolization. *Radiology* 212:385–389
- Vegas G, Illescas T, Muñoz M, Pérez-Piñar A (2006) Selective pelvic arterial embolization in the management of obstetric hemorrhage. *Eur J Obstet Gynecol Reprod Biol* 127:68–72
- Deux JF, Bazot M, Le Blanche AF, Tassart M, Khalil A, Berkane N, Uzan S, Boudghène F (2001) Is selective embolization of uterine arteries a safe alternative to hysterectomy in patients with postpartum hemorrhage? *AJR Am J Roentgenol* 177:145–149
- Lee HY, Shin JH, Kim J, Yoon HK, Ko GY, Won HS, Gwon DI, Kim JH, Cho KS, Sung KB (2012) Primary postpartum hemorrhage: outcome of pelvic arterial embolization in 251 patients at a single institution. *Radiology* 264:903–909

19. Cheong JY, Kong TW, Son JH, Won JH, Yang JI, Kim HS (2014) Outcome of pelvic arterial embolization for postpartum hemorrhage: a retrospective review of 117 cases. *Obstet Gynecol Sci* 57:17–27
20. Combs CA, Murphy EL, Laros RK Jr (1991) Factors associated with postpartum hemorrhage with vaginal birth. *Obstet Gynecol* 77:69–76
21. Dohan A, Soyer P, Subhani A, Hequet D, Fargeaudou Y, Morel O, Boudiaf M, Gayat E, Barranger E, Le Dref O, Sirol M (2013) Postpartum hemorrhage resulting from pelvic pseudoaneurysm: a retrospective analysis of 588 consecutive cases treated by arterial embolization. *Cardiovasc Intervent Radiol* 36:1247–1255
22. Soyer P, Fargeaudou Y, Morel O, Boudiaf M, Le Dref O, Rymer R (2008) Severe postpartum haemorrhage from ruptured pseudoaneurysm: successful treatment with transcatheter arterial embolization. *Eur Radiol* 18:1181–1187
23. La Folie T, Vidal V, Mehanna M, Capelle M, Jaquier A, Moulin G, Bartoli JM (2007) Result of endovascular treatment in cases of abnormal placentation with postpartum hemorrhage. *J Obstet Gynaecol Res* 33:624–630
24. Soyer P, Morel O, Fargeaudou Y, Sirol M, Staub F, Boudiaf M, Dahan H, Mebazaa A, Barranger E, le Dref O (2011) Value of pelvic embolization in the management of severe postpartum hemorrhage due to placenta accreta, increta or percreta. *Eur J Radiol* 80:729–735
25. Jung HN, Shin SW, Choi SJ, Cho SK, Park KB, Park HS, Kang M, Choo SW, Do YS, Choo IW (2011) Uterine artery embolization for emergent management of postpartum hemorrhage associated with placenta accreta. *Acta Radiol* 52:638–642
26. Chabrot P, Diop AN, Boyer L, Gallot D (2010) Maternal outcome after conservative treatment of placenta accreta. *Obstet Gynecol* 116:1219–1220
27. Diop AN, Chabrot P, Bertrand A, Constantin JM, Cassagnes L, Storme B, Gallot D, Boyer L (2010) Placenta accreta: management with uterine artery embolization in 17 cases. *J Vasc Interv Radiol* 21:644–648
28. Fargeaudou Y, Soyer P, Morel O, Sirol M, le Dref O, Boudiaf M, Dahan H, Rymer R (2009) Severe primary postpartum hemorrhage due to genital tract laceration after operative vaginal delivery: successful treatment with transcatheter arterial embolization. *Eur Radiol* 19:2197–2203
29. Yamashita Y, Takahashi M, Ito M, Okamura H (1991) Transcatheter arterial embolization in the management of postpartum hemorrhage due to genital tract injury. *Obstet Gynecol* 77:160–163
30. Achanna S, Mohamed Z, Krishnan M (2006) Puerperal uterine inversion: a report of four cases. *J Obstet Gynaecol Re* 32:341–345
31. Carberry GA, Pun CD, Dalvie PS (2012) Acute uterine inversion: case report and angiographic features. *J Vasc Interv Radiol* 23:1249–1250
32. Zahn CM, Hankins GD, Yeomans ER (1996) Vulvovaginal hematomas complicating delivery. rationale for drainage of the hematoma cavity. *J Reprod Med* 41:569–574
33. Pelage JP, Le Dref O, Jacob D, Soyer P, Herbreteau D, Rymer R (1999) Selective arterial embolization of the uterine arteries in the management of intractable post-partum hemorrhage. *Acta Obstet Gynecol Scand* 78:698–703
34. Prasertcharoensuk W, Swadpanich U, Lumbiganon P (2000) Accuracy of the blood loss estimation in the third stage of labor. *Int J Gynaecol Obstet* 71:69–70
35. Potts M, Hemmerling A (2006) The worldwide burden of postpartum haemorrhage: policy development where inaction is lethal. *Int J Gynaecol Obstet* 94(Suppl 2):S116–S121
36. Sierra A, Burrel M, Sebastia C, Radosevic A, Barrufet M, Albelá S, Buñesch L, Domingo MA, Salvador R, Real I (2012) Utility of multidetector CT in severe postpartum hemorrhage. *Radiographics* 32:1463–1481
37. Pelage JP, Le Dref O, Jacob D, Soyer P, Rossignol M, Truc J, Payen D, Rymer R (2000) Uterine artery embolization: anatomical and technical considerations, indications, results, and complications. *J Radiol* 81:1863–1872
38. Oberg AS, Hernandez-Diaz S, Palmsten K, Almqvist C, Bateman BT (2014) Patterns of recurrence of postpartum hemorrhage in a large population-based cohort. *Am J Obstet Gynecol* 210:229.e1–229.e8
39. Salomon LJ, deTayrac R, Castaigne-Meary V, Audibert F, Musset D, Ciorascu R, Frydman R, Fernandez H (2003) Fertility and pregnancy outcome following pelvic arterial embolization for severe post-partum haemorrhage. A cohort study. *Hum Reprod* 18:849–852
40. Sentilhes L, Gromez A, Clavier E, Resch B, Verspyck E, Marpeau L (2009) Predictors of failed pelvic arterial embolization for severe postpartum hemorrhage. *Obstet Gynecol* 113:992–999
41. Pelage JP, Le Dref O, Soyer P, Jacob D, Kardache M, Dahan H, Lassau JP, Rymer R (1999) Arterial anatomy of the female genital tract: variations and relevance to transcatheter embolization of the uterus. *AJR Am J Roentgenol* 172:989–994
42. Merland JJ, Chiras J (1981) Normal angiography. In: *Arteriography of the pelvis: diagnosis and therapeutic procedures*, 1st ed. Springer, Berlin, pp 5–68
43. Palacios Jaraquemada JM, Garcia Monaco R, Barbosa NE, Ferle L, Iriarte H, Conesa HA (2007) Lower uterine blood supply: extrauterine anastomotic system and its application in surgical devascularisation techniques. *Acta Obstet Gynecol Scand* 86:228–234
44. Chang S, Lee MS, Kim MD, Yoon CJ, Jung DC, Lee M, Park SI, Won JY, Lee do Y (2013) Inferior mesenteric artery collaterals to the uterus during uterine artery embolization: prevalence, risk factors, and clinical outcomes. *J Vasc Interv Radiol* 24:1353–1360
45. Soyer P, Boudiaf M, Jacob D, Hamzi L, Pelage JP, Le Dref O, Rymer R (2005) Bilateral persistent sciatic artery: a potential risk in pelvic arterial embolization in primary postpartum hemorrhage. *Acta Obstet Gynecol Scand* 84:605–606
46. Gayat E, Resche-Rigon M, Morel O, Rossignol M, Mantz J, Nicolas-Robin A, Nathan-Denizot N, Lefrant JY, Mercier FJ, Samain E, Fargeaudou Y, Barranger E, Laisné MJ, Bréchat PH, Luton D, Ouanounou I, Plaza PA, Broche C, Payen D, Mebazaa A (2011) Predictive factors of advanced interventional procedures in a multicentre severe postpartum haemorrhage study. *Intensive Care Med* 37:1816–1825
47. Lousquy R, Morel O, Soyer P, Malartic C, Gayat E, Barranger E (2011) Routine use of abdominopelvic ultrasonography in severe postpartum hemorrhage: retrospective evaluation in 125 patients. *Am J Obstet Gynecol* 204:232.e1–232.e6
48. Sellmyer MA, Desser TS, Maturen KE, Jeffrey RB Jr, Kamaya A (2013) Physiologic, histologic, and imaging features of retained products of conception. *Radiographics* 33:781–796
49. Takeda A, Koike W, Imoto S, Nakamura H (2014) Three-dimensional computerized tomographic angiography for diagnosis and management of intractable postpartum hemorrhage. *Eur J Obstet Gynecol Reprod Biol* 176:104–111
50. Dohan A, Dautry R, Le Dref O, Soyer P (2014) Avoiding CT angiography radiation in management of post-partum hemorrhage. *Eur J Obstet Gynecol Reprod* 180:194–195
51. Tsitlakidis C, Alalade A, Danso D, B-Lynch C (2006) Ten year follow-up of the effect of the B-Lynch uterine compression suture for massive postpartum hemorrhage. *Int J Fertil Womens Med* 51:262–265
52. Doumouchtsis SK, Papageorgiou AT, Arulkumaran S (2007) Systematic review of conservative management of postpartum hemorrhage: what to do when medical treatment fails. *Obstet Gynecol Surv* 62:540–547

53. AbdRabbo SA (1994) Stepwise uterine devascularization: a novel technique for management of uncontrolled postpartum hemorrhage with preservation of the uterus. *Am J Obstet Gynecol* 171:694–700
54. Salazar GM, Petrozza JC, Walker TG (2009) Transcatheter endovascular techniques for management of obstetrical and gynecologic emergencies. *Tech Vasc Interv Radiol* 12:139–147
55. Robert P, Giudicelli DP, Ronze S, Gauthier G, Julien V, Rondelet O (2010) Uterine embolization in the operating theater in severe post-partum haemorrhage. *Ann Fr Anesth Reanim* 29:655–657
56. Park JK, Shin TB, Baek JC, Shin JK, Choi WJ, Lee SA, Lee JH, Paik WY (2011) Failure of uterine artery embolization for controlling postpartum hemorrhage. *J Obstet Gynaecol Res* 37:971–978
57. Pelage JP, Laissy JP (2006) Management of life-threatening postpartum hemorrhage: indications and technique of arterial embolization. *J Radiol* 87:533–540
58. Pelage JP, Soyer P, Le Dref O, Dahan H, Coumbaras J, Kardache M, Rymer R (1999) Uterine arteries: bilateral catheterization with a single femoral approach and a single 5-F catheter. *Radiology* 210:573–575
59. Gonsalves M, Belli A (2010) The role of interventional radiology in obstetric hemorrhage. *Cardiovasc Intervent Radiol* 33:887–895
60. Kirby JM, Kachura JR, Rajan DK, Sniderman KW, Simons ME, Windrim RC, Kingdom JC (2009) Arterial embolization for primary postpartum hemorrhage. *J Vasc Interv Radiol* 20:1036–1045
61. Ratnam LA, Raja J, Munneke GJ, Morgan RA, Belli AM (2007) Prospective nonrandomized trial of manual compression and Angio-Seal and Starclose arterial closure devices in common femoral punctures. *Cardiovasc Intervent Radiol* 30:182–188
62. Dohan A, Pelage JP, Soyer P (2013) How to avoid uterine necrosis after arterial embolization for post-partum hemorrhage: a proposal based on a single center experience of 600 cases. *Eur J Obstet Gynecol Reprod Biol* 171:392–393
63. Das R, Champaneria R, Daniels JP, Belli AM (2013) Comparison of embolic agents used in uterine artery embolisation: a systematic review and meta-analysis. *Cardiovasc Intervent Radiol* 37:1179–1190
64. Yoon W (2004) Embolic agents used for bronchial artery embolisation in massive haemoptysis. *Expert Opin Pharmacother* 5:361–367
65. Pellerin O, Bats AS, Di Primio M, Palomera-Ricco A, Pinot de Villechenon G, Fournier L, Pagny JY, Beyssen B, Louail B, Lécure F, Sapoval M (2013) Postpartum hemorrhage treated with gelfoam slurry embolization using the superselective technique: immediate results and 1-month MRI follow-up. *Cardiovasc Intervent Radiol* 36:98–104
66. Soyer P, Sirol M, Fargeaudou Y, Bour L, Morel O, Dohan A, Boudiaf M, Gayat E, Hequet D, Barranger E, le Dref O (2013) Placental vascularity and resorption delay after conservative management of invasive placenta: MR imaging evaluation. *Eur Radiol* 23:262–271
67. Poujade O, Zappa M, Letendre I, Ceccaldi PF, Vilgrain V, Luton D (2012) Predictive factors for failure of pelvic arterial embolization for postpartum hemorrhage. *Int J Gynaecol Obstet* 117:119–123
68. Poujade O, Ceccaldi PF, Davitian C, Amate P, Chatel P, Khater C, Aflak N, Vilgrain V, Luton D (2013) Uterine necrosis following pelvic arterial embolization for post-partum hemorrhage: review of the literature. *Eur J Obstet Gynecol Reprod Biol* 170:309–314
69. Loffroy R, Rao P, Ota S, De Lin M, Kwak BK, Krause D, Geschwind JF (2010) Packing technique for endovascular coil embolisation of peripheral arterial pseudo-aneurysms with preservation of the parent artery: safety, efficacy and outcomes. *Eur J Vasc Endovasc Surg* 40:209–215
70. Cottier JP, Fignon A, Tranquart F, Herbreteau D (2002) Uterine necrosis after arterial embolization for postpartum hemorrhage. *Obstet Gynecol* 100:1074–1077
71. Kanematsu M, Watanabe H, Kondo H, Goshima S, Kato H, Furui T, Toyoki H, Morishige K (2011) Postpartum hemorrhage in coagulopathic patients: preliminary experience with uterine arterial embolization with N-butyl cyanoacrylate. *J Vasc Interv Radiol* 22:1773–1776
72. Eriksson LG, Mulic-Lutvica A, Jangland L, Nyman R (2007) Massive postpartum hemorrhage treated with transcatheter arterial embolization: technical aspects and long-term effects on fertility and menstrual cycle. *Acta Radiol* 48:635–642
73. Nikolic B, Spies JB, Campbell L, Walsh SM, Abbara S, Lundsten MJ (2001) Uterine artery embolization: reduced radiation with refined technique. *J Vasc Interv Radiol* 12:39–44
74. Thabet A, Kalva SP, Liu B, Mueller PR, Lee SI (2012) Interventional radiology in pregnancy complications: indications, technique, and methods for minimizing radiation exposure. *Radiographics* 32:255–274
75. Kim YJ, Yoon CJ, Seong NJ, Kang SG, An SW, Kim YS, Woo YN (2013) Failed pelvic arterial embolization for postpartum hemorrhage: clinical outcomes and predictive factors. *J Vasc Interv Radiol* 24:703–709
76. Bros S, Chabrot P, Kastler A, Ouchchane L, Cassagnes L, Gallot D, Boyer L (2012) Recurrent bleeding within 24 hours after uterine artery embolization for severe postpartum hemorrhage: are there predictive factors? *Cardiovasc Intervent Radiol* 35:508–514
77. Maassen MS, Lambers MD, Tutein Nolthenius RP, van der Valk PH, Elgersma OE (2009) Complications and failure of uterine artery embolisation for intractable postpartum haemorrhage. *BJOG* 116:55–61
78. Chou YJ, Cheng YF, Shen CC, Hsu TY, Chang SY, Kung FT (2004) Failure of uterine arterial embolization: placenta accreta with profuse postpartum hemorrhage. *Acta Obstet Gynecol Scand* 83:688–690
79. Chassang M, Novellas S, Baudin G, Bouaziz J, Bongain A, Chevallier P (2011) Uterine necrosis complicating embolization with resorbable material for postpartum hemorrhage. *J Radiol* 92:725–728
80. Amato P, Roberts AC (2001) Transient ovarian failure: a complication of uterine artery embolization. *Fertil Steril* 75:438–439
81. Stancato-Pasik A, Mitty HA, Richard HM 3rd, Eshkar N (1997) Obstetric embolotherapy: effect on menses and pregnancy. *Radiology* 204:791–793
82. Sentilhes L, Gromez A, Clavier E, Resch B, Verspyck E, Marpeau L (2010) Fertility and pregnancy following pelvic arterial embolisation for postpartum haemorrhage. *BJOG* 117:84–93
83. Fiori O, Deux JF, Kambale JC, Uzan S, Bougdhene F, Berkane N (2009) Impact of pelvic arterial embolization for intractable postpartum hemorrhage on fertility. *Am J Obstet Gynecol* 200:384.e1–384.e4
84. Gaia G, Chabrot P, Cassagnes L, Calcagno A, Gallot D, Botchorishvili R, Canis M, Mage G, Boyer L (2009) Menses recovery and fertility after artery embolization for PPH: a single-center retrospective observational study. *Eur Radiol* 19:481–487
85. Lee HJ, Jeon GS, Kim MD, Kim SH, Lee JT, Choi MJ (2013) Usefulness of pelvic artery embolization in cesarean section compared with vaginal delivery in 176 patients. *J Vasc Interv Radiol* 24:103–109
86. Ripley DL (1999) Uterine emergencies. Atony, inversion, and rupture. *Obstet Gynecol Clin North Am* 26:419–434
87. Parikh R, Brotzman S, Anasti JN (2007) Cervical lacerations: some surprising facts. *Am J Obstet Gynecol* 196:17–18
88. Alanis M, Hurst BS, Marshburn PB, Matthews ML (2006) Conservative management of placenta increta with selective

- arterial embolization preserves future fertility and results in a favorable outcome in subsequent pregnancies. *Fertil Steril* 86: 1514.e3–1514.e7
89. Hequet D, Morel O, Soyer P, Gayat E, Malartic C, Barranger E (2013) Delayed hysteroscopic resection of retained tissues and uterine conservation after conservative treatment for placenta accreta. *Aust N Z J Obstet Gynaecol* 53:580–583
 90. Mitty HA, Sterling KM, Alvarez M, Gendler R (1993) Obstetric hemorrhage: prophylactic and emergency arterial catheterization and embolotherapy. *Radiology* 188:183–187
 91. Ojala K, Perälä J, Kariniemi J, Ranta P, Raudaskoski T, Tekay A (2005) Arterial embolization and prophylactic catheterization for the treatment for severe obstetric hemorrhage. *Acta Obstet Gynecol Scand* 84:1075–1080
 92. Royal College of Obstetricians and Gynaecologists (2009) Green-top Guideline No. 52: Prevention and management of postpartum haemorrhage. RCOG, London
 93. Thon S, McLintic A, Wagner Y (2011) Prophylactic endovascular placement of internal iliac occlusion balloon catheters in parturients with placenta accreta: a retrospective case series. *Int J Obstet Anesth* 20:64–70
 94. Bishop S, Butler K, Monaghan S, Chan K, Murphy G, Edozien L (2011) Multiple complications following the use of prophylactic internal iliac artery balloon catheterisation in a patient with placenta percreta. *Int J Obstet Anesth* 20:70–73
 95. Teare J, Evans E, Belli A, Wendler R (2014) Sciatic nerve ischaemia after iliac artery occlusion balloon catheter placement for placenta percreta. *Int J Obstet Anesth* 23:178–181
 96. Clausen C, Stensballe J, Albrechtsen CK, Hansen MA, Lönn L, Langhoff-Roos J (2013) Balloon occlusion of the internal iliac arteries in the multidisciplinary management of placenta percreta. *Acta Obstet Gynecol Scand* 92:386–391
 97. Baba Y, Matsubara S, Kuwata T, Ohkuchi A, Usui R, Saruyama M, Nakata M, Suzuki M (2014) Uterine artery pseudoaneurysm: not a rare condition occurring after non-traumatic delivery or non-traumatic abortion. *Arch Gynecol Obstet* 290:435–440
 98. Mammen T, Shanthakumari H, Gopi K, Lionel J, Ayyappan AP, Kekre A (2006) Iatrogenic secondary post-partum haemorrhage: apropos of two uncommon cases. *Australas Radiol* 50:392–394
 99. Fargeaudou Y, Morel O, Soyer P, Gayat E, Sirol M, Boudiaf M, Dahan H, Barranger E, Mebazaa A, le Dref O (2010) Persistent postpartum haemorrhage after failed arterial ligation: value of pelvic embolisation. *Eur Radiol* 20:1777–1785
 100. Abbara S, Nikolic B, Pelage JP, Banovac F, Spies JB (2007) Frequency and extent of uterine perfusion via ovarian arteries observed during uterine artery embolization for leiomyomas. *AJR Am J Roentgenol* 188:1558–1563
 101. Betrán AP, Merialdi M, Lauer JA, Bing-Shun W, Thomas J, Van Look P, Wagner M (2007) Rates of caesarean section: analysis of global, regional and national estimates. *Paediatr Perinat Epidemiol* 21:98–113
 102. Wi JY, Kim HC, Chung JW, Jun JK, Jae HJ, Park JH (2009) Importance of angiographic visualization of round ligament arteries in women evaluated for intractable vaginal bleeding after uterine artery embolization. *J Vasc Interv Radiol* 20:1031–1035