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MATERNAL-FETAL MEDICINE

Efficacy of pelvic artery embolisation for severe postpartum hemorrhage

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Abstract
Purpose The purpose of our study was to evaluate the outcome of selective pelvic arterial embolisation (PAE) in women with severe postpartum hemorrhage (PPH).
Methods We performed a retrospective, controlled, single-center cohort study. A total of 16 consecutive women with PPH who underwent therapeutic PAE were included. As historical control group, we included 22 women with similar severity of PPH who were managed without PAE. Outcome measures included necessity of surgical interventions such as postpartum hysterectomy and laparotomy after vaginal delivery, the amount of red blood cell transfusions, and hematologic findings after the procedure.
Results PAE was successful in stopping PPH and preserving the uterus in all 16 women in the study group. No woman in the PAE group required a postpartum hysterectomy, whereas postpartum hysterectomy was unavoidable in two women in the control group. Laparotomy after vaginal delivery was necessary in two women of the group without embolisation. Hematologic parameters after the treatment were better in the PAE group than in the control group, although these differences were only in part statistically significant. There were no unwarranted effects of PAE identifiable in the study group.
Conclusion This is the first controlled study assessing the efficacy of PAE for the treatment of PPH. Our data suggest that PAE is effective for the treatment of severe PPH. In view of the lack of complications and unwarranted effects, clinical use of PAE in severe PPH seems justified, particularly in view of the life-threatening condition and the potential to preserve fertility in affected patients. Further evidence from well-designed prospective randomized-controlled trials would be nevertheless desirable in the future.

Keywords Postpartum hemorrhage · Selective pelvic arterial embolisation · Postpartum hysterectomy · Laparotomy after vaginal delivery

Abbreviations

DIC	Disseminated intravascular coagulation
FFP	Fresh frozen plasma
PAE	Selective pelvic arterial embolisation
PLT	Platelets
PPH	Severe postpartum hemorrhage
RBCU	Red blood cell unit
RCOG	Royal College of Obstetricians and Gynaecologists
rhFVIIa	Recombinant human factor VIIa
SEM	Standard error of the mean
UBT	Uterine balloon tamponade
WHO	World Health Organization

Introduction

Severe postpartum hemorrhage is the most common cause of maternal death according to the World Health Organization (WHO): 10.5% of all live births are associated with severe bleeding and 25% of the maternal death cases are caused by postpartum hemorrhage [1].

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The definition of postpartum hemorrhage (PPH) varies in the literature. Nevertheless, a blood loss of > 500 ml for vaginal delivery and > 1000 ml for a cesarean section are most widely accepted definitions [2].

The amount of blood loss in PPH based on visual examination is often underestimated by up to 50%. Therefore, PPH can also be defined as a 10% change in hematocrit between admission and the postpartum period, or if a transfusion was considered clinically necessary [3–5]. PPH is divided into primary hemorrhage, which indicates excessive bleeding from the genital tract of 500 ml or more in the first 24 h after a vaginal delivery, and secondary hemorrhage, which occurs after the first 24 h and until 6 weeks after the birth [2, 6].

Causes of primary postpartum blood loss include uterine atony, isolated or combined with traumatic or placental causes of blood loss; placental causes such as retained placental tissue, incomplete placental abruption, or placental implantation abnormalities; genital tract injuries such as perineal or vaginal laceration, cervical tear, uterine rupture or paravaginal and pararectal hematomas; and coagulation disorders such as primary coagulation disorders, such as hereditary hemophilia or plasmatic coagulation disorders based on blood loss [7]. Uterine atony and lower genital tract laceration are the most common causes of PPH [8–10].

Recently, Ekin et al. described previous cesarean delivery, prolonged labor, oxytocin augmentation and emergency cesarean delivery as strongest predictors of severe blood loss in women with PPH. In addition, uterine atony and abnormal placentation are the causes of bleeding significantly associated with severe PPH [11]. Secondary PPH is mainly related to retained products of gestation or infection [12].

Selective pelvic arterial embolisation (PAE)

In 1979, Brown et al. described, for the first time, the use of angiographic arterial embolisation as a new approach to hemostasis in cases of uncontrollable PPH. The authors considered angiographic embolisation to be an effective alternative approach to the control of pelvic hemorrhage and recommend that the technique be considered prior to surgical intervention [13]. The first embolisation was performed in a woman with PPH after hysterectomy and vascular ligation.

Since their successful case report was published, the method of PAE as treatment for cases of severe PPH has gained increasing worldwide acceptance as a treatment option in the management of PPH in which conservative measures fail. The most common cause of PPH requiring embolisation is uterine atony [12].

However, PAE is also indicated in hemorrhage from genital tract laceration, due to abnormal placentation, pseudoaneurysms, arteriovenous malformations, and postsurgical complications, surgical complications, or uterine tears at the time of cesarean section. Furthermore, the RCOG has recommended interventional radiology also be considered for bleeding while in the recovery unit or in the postnatal ward after a normal delivery or a cesarean section and for bleeding after hysterectomy [14]. Previous uncontrolled studies proposed a success rate of PAE of round 90% [8, 15].

PAE does not require general anesthesia, is not disturbed by coagulation disorders, and is reproducible [16]. Thus, if all medical measures are unsuccessful, uterine embolisation could be directly initiated if the woman is hemodynamically stable enough to be moved and the possibility of embolisation with interventional radiology is available nearby [17].

When performed by experienced interventional radiologists, complication rates after arterial embolisation are low, with the overall complication rate ranging between six percent and nine percent [8, 12, 16, 18–22]. Most complications are minor and associated with the arterial puncture and angiography in general. Postembolisation syndrome, with transient abdominal pain, fever, nausea, and mild leukocytosis, should be expected. Ischemic und neurological complications are rare [12].

After embolisation, most women do not report a change in the quality of menses or in the clinical symptoms of early menopause. Moreover, PAE does not appear to affect menstruation or fertility [23].

There is wide variety of publications that have investigated the effectiveness of PAE in retrospective case studies. However, none of these studies included a control group and, therefore, the efficacy of PAE might be overestimated.

The purpose of our study was, therefore, to evaluate the outcome of PAE in women with PPH by comparing the study group to a control group with similar severity of PPH, managed similarly but without PAE.

Methods

We performed a retrospective, controlled cohort study between 2004 and 2012. A total of 16 consecutive women with severe PPH underwent PAE as a rescue therapy after unsuccessful medical or surgical uterine-preserving management. As historical control group, we included 22 women with severe PPH whose inclusion characteristics were comparable to those of the PAE group, but who were managed without embolisation before this treatment was established in our hospital. Importantly, the clinical management of both groups did not differ between the groups, except in the use of PAE. All patients were treated according to the same clinical algorithm before PAE, which included fluid management, administration of tranexamic acid, uterotonic drugs, such as intravenous oxytocin, rectal misoprostol, and intravenous sulprostone, red blood cell units (RBCUs), fibrinogen, fresh frozen plasma (FFP),

platelets (PLT), and activated recombinant human Factor seven (rhFVIIa), as required. Surgical interventions included the repair of lower genital tract lesions, manual and instrumental uterine revision and curettage, uterine tamponade by a balloon catheter or by surgical towels, uterine compression sutures (mainly B-Lynch and Hayman sutures), and arterial vasoligation. PAE was performed as a last step before proceeding to a hysterectomy if the above-named treatments were ineffective. Prerequisite for PAE was hemodynamic stability.

After the decision to perform PAE was made, the woman was transported from the maternity unit to the radiology department, which was in immediate vicinity, accompanied by an anesthetist. Using a right-sided unifemoral approach, digital subtraction angiography was performed to determine anatomy and to locate possible extravasation of contrast agent. A contralateral internal iliac angiography was performed initially, and then a selective examination of the uterine artery was subsequently attempted in all cases. The ipsilateral internal iliac artery and uterine artery were also catheterized with the same catheter and via the same puncture site. Other anastomotic vessels, such as the vaginal branches, were assessed when necessary. The embolisation material consisted of mainly pledgets of absorbable gelatin sponge and embolisation microspheres; coils and *n*-butyl-2-cyanoacrylate were also used in a few cases.

Hematologic parameters were monitored repetitively before, during, and after PPH treatment. The hemostatic efficiency of the procedure was evaluated clinically and was considered good when visible hemostasis was noticed. Post-therapeutic transfusion rates of blood products were indirect parameters for significant bleeding and treatment failures. The success of the PAE was defined as a stopping of the hemorrhage, whatever the number of PAE procedures, with no subsequent surgical procedure due to persistent bleeding.

Outcome parameters included the success of PAE in stopping the bleeding, whether postpartum hysterectomy or laparotomy after vaginal birth was required, the amount of blood transfusions, and hematologic findings before and after the procedure.

Data were presented as mean ± standard error of the mean (SEM). All statistical calculations were performed with GraphPad Prism version 5.0 for Windows (GraphPad Software, San Diego CA, USA). Comparisons of the study groups were statistically analyzed by Student's *T* test or Mann–Whitney test if the data were not normally distributed assessed by Shapiro–Wilk normality test. Fisher's exact test was performed for categorical variables. A probability value (p) < 0.05 was considered to reach the level of significance.

The study was approved by the ethical committee of the canton of Bern, Switzerland. Date of ethics approval: 13.06.2016. Reference number to the ethics approval statement: 3037.

Results

Both groups were comparable in age, body weight, gestational age at delivery, parity, and in the delivery mode. All patient characteristics and delivery modes are shown in Table 1, and were not significantly different.

Uterine atony was the most common cause of bleeding in both groups. In the control group, uterine atony was significantly more often present than in the embolisation group (90.91 vs. 62.5%; $p = 0.0498$; Table 2). Injuries of the lower genital tract, abnormal placentation, and residual placental tissue were encountered more frequently in the embolisation group than in the control group, but these differences were not significant (Table 2). Disseminated intravascular coagulation (DIC), as an acquired hemorrhagic diathesis characterized by systemic activation of coagulation, did not occur significantly more frequently in the embolisation group (12.5 vs. 0%; $p = 0.171$; Table 2).

PAE was successful in stopping the PPH and preserving the uterus in all patients. In one patient, a second embolisation was necessary within the 9 h subsequent to the procedure due to recurrent bleeding after an interval of several hours of bleeding cessation. This second procedure, however, did stop the bleeding definitively. The duration of the entire procedure of an embolisation was approximately 102 min (range 60–160 min).

Angiography showed an extravasation of contrast agent in five of the 16 women. A PAE of both uterine arteries was performed in the majority of the cases. Other embolized arteries were the vaginal artery, the pudenda artery, and the internal iliac artery. In one woman, a pseudoaneurysm of the right uterine artery was embolized successfully. There were no major complications due to the pelvic artery embolisation. One woman complained of pain in the groin, the leg, and the pelvis after the procedure. In the follow-up, Asherman syndrome developed in two women, but, it was not clear

Table 1 Patient characteristics

Characteristics	PPH with embolisation, $n = 16$	PPH without embolisation, $n = 22$	Significance
Age (years, mean/SEM)	30.94 ± 1.49	31.82 ± 1.06	0.6237 (ns)
Weight (kg, mean/SEM)	67.24 ± 3.11	66.89 ± 2.15	0.9241 (ns)
Gestational age (weeks p.m., median/SEM)	38.8 ± 0.7	38.4 ± 0.8	0.6805 (ns)
Primiparity (%, *n*)	68.75 (11)	50 (11)	0.3262 (ns)
Cesarean section (%, *n*)	56.25 (9)	59.09 (13)	1.0 (ns)

SEM standard error of the mean, *NS* not significant

Table 2 PPH characteristics

PPH characteristics	PPH with embolisation, $n = 16$	PPH without embolisation, $n = 22$	Significance
Uterine atony (%, *n*)	62.50 (10)	90.91 (20)	0.0498
Injuries of the lower genital tract (%, *n*)	43.75 (7)	31.82 (7)	0.5105 (ns)
Abnormal placentation (%, *n*)	25 (4)	18.18 (4)	0.6984 (ns)
Residual placental tissue (%, *n*)	12.5 (2)	9.09 (2)	1.0 (ns)
DIC (%, *n*)	12.5 (2)	0 (0)	0.1707 (ns)
Blood loss, totally, estimated (ml, mean/SEM)	4920 ± 889.2	4767 ± 749.2	0.8842 (ns)

SEM standard error of the mean, *NS* not significant

whether this was due to the PAE or due to other treatments (curettage). The first woman had two curettages after delivery and one uterine tamponade before a PAE was performed. Subsequently she suffered from persisting amenorrhea. The second woman had a cesarean section with uterine compressive sutures before a PAE was performed. Subsequently she had a hysteroscopic resection of uterine synechiae and 2 years later a normal pregnancy.

The mean total estimated blood loss of the embolisation group was 4920 ± 889 ml. This was slightly higher than in the control group (4767 ± 749 ml) (p = n. s.) (Table 2). In the embolisation group, there was almost no further blood loss after the therapy, whereas, in the control group, in half the women, the bleeding did not stop completely after therapy.

The treatment cascade followed the algorithm and was comparable in both groups: Conservative surgical treatment in both groups comprised PPH with PAE and without PAE with no significant differences between the groups: curettage in 50 vs. 73%; sutures for injuries of the lower genital tract in 38 vs. 32%; uterine tamponade with balloon catheters or surgical towels in 63 vs. 50%; and uterine compression sutures in 38 vs. 41%. There were no differences between the groups regarding the use of intravenous prostaglandins: All women received sulprostone i.v. No woman in the embolisation group required a postpartum hysterectomy, whereas, all therapies were unsuccessful in stopping the bleeding in two women of the control group, so that a postpartum hysterectomy became inevitable. There was no difference in chorioamnionitis between groups, and there was no postpartum endomyometritis present (all women studied received antibiotic prophylaxis during or after PPH).

A laparotomy after vaginal delivery for either uterine compression sutures or a postpartum hysterectomy was necessary in two women in the group without embolisation. In contrast, no woman in the embolisation group required a laparotomy after vaginal delivery. These differences, however, were not significant (Table 3).

All women in the control group received recombinant human factor VIIa, whereas, in the embolisation group, the application of factor VIIa occurred only in six of 16 women (31%). This difference was significant, with $p < 0.0001$ (Table 3).

More RBC transfusions in total and before treatment were required in the embolisation group, but the difference was not significant (Table 3). This correlates well to the slightly higher blood loss before treatment in the PAE group (see above). On the contrary, significantly less units of FFP after treatment were administered in the embolisation group compared to the control group, and significantly less women in the embolisation group required FFP substitutions after treatment (0 vs. 31.8%; $p = 0.0144$; Table 3). The PLT transfusions before treatment were less in the control group, but PLT transfusions after treatment were less in the embolisation group. Overall, although not significant, there were less women who required PLT in the embolisation group (6.3%) than in the control group (13.6%) (Table 3).

The mean hemoglobin prior to the therapy was lower in the embolisation group and the mean hematocrit was lower in the group without embolisation. The mean platelet level before treatment was lower in the control group, whereas, the mean fibrinogen was lower in the embolisation group (Table 4). But, all these findings were not statistically significant. Nevertheless they emphasize the previous findings that the extent of bleeding was comparable between the two groups. After the treatment, the hematologic parameters were higher in the embolisation group than in the control group, especially platelets were significantly higher. (Table 4). During the follow-up after PPH treatment until discharge or until 6 weeks postpartum, no patient developed a severe complication of PAE such as thromboembolic events or necrotic degeneration of bladder or uterus. One patient experienced pain in the groin, leg and pelvis after the PAE procedure, and two patients were diagnosed with intrauterine synechiae at a later stage.

Discussion

Our study suggests that PAE is an effective treatment modality for severe PPH. It represents the first study on this issue published to date comparing PAE with a control group,

Table 3 Treatment

Treatment	PPH with embolisation, $n = 16$	PPH without embolisation, $n = 22$	Significance
Curettage (%, *n*)	50 (8)	72.73 (16)	0.3387 (ns)
Suture of injuries of lower genital tract (%, *n*)	37.5 (6)	31.82 (7)	0.7421 (ns)
Uterine tamponade/balloon tamponade (%, *n*)	62.5 (10)	50 (11)	0.5205 (ns)
Uterine compression sutures (%, *n*)	37.5 (6)	40.91 (9)	1.0 (ns)
Hysterectomy pp (%, *n*)	0	9.09 (2)	0.4993 (ns)
Laparotomy after vaginal delivery (%, *n*)	0 (0/7)	22.22 (2/9)	0.4967 (ns)
Recombinant human factor VIIa application (%, *n*)	31.25 (6)	100 (22)	< 0.0001
Transfusion RBC before treatment (mean/SEM)	10.94 ± 2.11	8.86 ± 1.05	0.6995 (ns)
Transfusion RBC after treatment (mean/SEM)	0.69 ± 0.35	0.86 ± 0.29	0.4403 (ns)
Transfusion RBC total (mean/SEM)	11.63 ± 2.11	9.95 ± 1.05	0.8587 (ns)
Transfusion FFP before treatment (mean/SEM)	6.00 ± 1.63	6.77 ± 0.83	0.2786 (ns)
Transfusion FFP after treatment (mean/SEM)	0.00 ± 0.00	0.86 ± 0.30	< 0.05
Transfusion FFP total (mean/SEM)	6.00 ± 1.63	7.64 ± 0.85	0.1415 (ns)
Transfusion PLT before treatment (mean/SEM)	1.56 ± 0.51	1.23 ± 0.24	0.8897 (ns)
Transfusion PLT after treatment (mean/SEM)	0.06 ± 0.06	0.18 ± 0.11	0.4703 (ns)
Transfusion PLT total (mean/SEM)	1.63 ± 0.52	1.48 ± 0.29	0.7012 (ns)
Patients with RBC after treatment (%, *n*)	25.00 (4)	40.90 (9)	0.4898 (ns)
Patients with FFP after treatment (%, *n*)	0.00 (0)	31.80 (7)	0.0144
Patients with PLT after treatment (%, *n*)	6.25 (1)	13.60 (3)	0.6245 (ns)

RBC red blood cell, *FFP* fresh frozen plasma, *PLT* platelet transfusion, *SEM* standard error of the mean, *NS* not significant, *pp* postpartum

Table 4 Hematologic findings before and after treatment

Hematologic characteristics (mean/SEM)	PPH with embolisation, $n = 16$	PPH without embolisation, $n = 22$	Significance
Hemoglobin before treatment (g/l)	64.63 ± 3.57	70.05 ± 3.281	0.3004 (ns)
Hemoglobin after treatment (g/l)	96.73 ± 2.78	90.77 ± 3.22	0.1967 (ns)
Hematocrit before treatment (%)	21.47 ± 1.03	20.77 ± 0.92	0.3549 (ns)
Hematocrit after treatment (%)	28.47 ± 0.93	26.23 ± 0.96	0.0848 (ns)
Platelets before treatment (× 109/l)	120.60 ± 24.75	106.91 ± 11.79	1.0000 (ns)
Platelets after treatment (× 109/l)	149.67 ± 17.23	109.00 ± 11.84	0.0127
Fibrinogen before treatment (g/l)	1.87 ± 0.25	3.82 ± 2.15	0.9052 (ns)
Fibrinogen after treatment (g/l)	4.67 ± 0.42	4.04 ± 0.40	0.3180 (ns)

SEM standard error of the mean, *NS* not significant

as previously published studies to date included only case series. Our data show that PAE was successful in all patients in stopping the bleeding, and no patient in the PAE group needed laparotomy after vaginal delivery or had postpartum hysterectomy, while there was no statistically significant difference evident in the PAE group compared to the control group with regard to blood loss and number of packed red blood cell transfusions.

In high-income countries, there appears to be an increasing rate of PPH, as reported in several studies [24–32]. The factors underlying the increase have been unknown, until now, but increasing cesarean section rates may contribute [31]. PPH can lead to death and serious complications that follow an extreme drop in blood volume and related organ failure, including acute renal failure, adult respiratory distress syndrome, coagulopathy, and shock [31, 33, 34]. As described extensively in the literature previously, and, as we can confirm, uterine atony is the most common cause for PPH, and thus for therapy [9, 10]. In our group with PAE, the distribution of causes for PPH was different from that in the control group. There were significantly less uterine atonies compared to the control group. This is a significant advantage of PAE, as has also been described by Tourné et al. [16].

The mean total estimated blood loss of the embolisation group was similar in both groups. However, as these

values were estimated and, therefore, imprecise, but this was one parameter that accentuated the comparability of both groups with regard to the magnitude of the PPH. After the therapy, there was almost no further blood loss in the embolisation group compared to the control group. In the control group, the blood loss did not stop in half the women, so more blood substitutes were required in the group without embolisation after treatment. Women in the embolisation group required significantly less applications of factor VIIa and less applications of FFP after therapy [35].

Recently, with advances in radiologic intervention, the chances of effective bleeding control have significantly increased due to PAE. In addition, uterine-sparing techniques do not carry significant advantageous over PAE, as described in a recent meta-analysis of conservative management for PPH [36, 37]. The success rate of the procedure performed by experienced interventional radiologists has been reported to be around 90% or more in the literature [8, 15]. In our study, the overall success rate of PAE for stopping the bleeding was 100%, while one patient had bleeding recurrence after several hours, which could be definitively stopped after a second PAE. There have been some studies describing predictive factors for the failure of PAE for intractable PPH. Sentilhes et al. reported that failure of PAE was associated with a higher rate of estimated blood loss (more than 1500 ml) and transfusion of more than 5 RBCUs [38]. In contrast to these studies, we reached our success rate in a group of women with severe PPH and with an estimated total blood loss of more than 4900 ml and a median transfusion of more than 11 RBCUs. Considering that visual assessment underestimates the amount of blood loss in approximately 45% of cases [16], we agree with the findings of Ji Yoon Cheong, that these two predictors could not be considered true or useful predictive factors [37]. Recently, Kim et al. found that DIC was the only independent predictor of PAE failure [39]. In our embolisation group, we found a very low DIC rate, but the DIC rate in the control group was lower (0 vs. 12.5%). These findings support the importance of triage in hemodynamically stable women to ensure the success of PAE.

During the past 20 years, the frequency of emergency peripartum hysterectomy has decreased from 1/1000 to 1/2000 deliveries in developed countries. This substantial drop might be due to marked improvements in medical resuscitation and an increased use of conservative treatments, including pelvic artery ligation, uterine compression techniques, uterine balloon tamponade (UBT), and, not least, of pelvic arterial embolisation [37, 40, 41].

Two postpartum hysterectomies were necessary in our control group, in contrast to the embolisation group, where no hysterectomy was required. These findings are also unanimously in agreement with other publications [12].

A laparotomy after vaginal delivery for either uterine compression sutures or a postpartum hysterectomy was necessary in two women of the group without embolisation. On the contrary, no women in the embolisation group had a laparotomy after vaginal delivery.

Both procedures are associated with an increased risk of complications when performed as an emergency, and the possibility to avoid them represents a significant advantage for PAE [42, 43]. Moreover, the necessity of a hysterectomy means the loss of fertility at the same time [23, 37].

There were no major complications due to PAE. Three women had minor problems, one with pain in the groin, leg, and pelvis after the procedure and two with intrauterine synechiae. The endometrial ischemia responsible for the appearance of diffuse synechiae can be related to the PAE, as in the literature ischemic complications are described after PAE, most likely in cases of PAE with small sized particles [44, 45]. However, before PAE both women received postpartum curettage and uterine compressive sutures, which might also be the cause of intrauterine synechiae in these women. In the literature the risk for Asherman syndrome after PAE is 12% [46], after uterine compressive sutures 18.5% [47] and after postpartum Curettage even 21.5% [48, 49]. It, therefore, remains open what the main cause for the intrauterine synechiae in the two women has been.

This study has limitations, which need to be acknowledged. First, the number of patients per group is limited. Second, and probably more important, the study design is retrospective and the control group is historical. Nevertheless, the groups are comparable regarding the most important characteristics of PPH severity, mode of delivery and cause of PPH, except somewhat more uterine atony in the control group, which should not have a general impact on our results due to the small difference. A further concern to be raised in our study design is potential differences in the PPH management cascade between the groups. While we cannot fully exclude this potential bias, we believe it to be unlikely because in both groups the same algorithm as defined by an in-house guideline according to the recommendations of the Swiss Society of Gynecology and Obstetrics was followed. Furthermore, the major interventions (prostaglandins, uterine tamponade, uterine compression sutures) were comparable between the groups. Third, we did not systematically assess unwarranted long-term effects of PAE on menstruation, fertility, or future pregnancy. We, therefore, cannot exclude long-term effects of PAE on these important outcomes.

Despite these limitations, which we fully acknowledge, our study adds important informations for the clinician on the effectiveness of PAE in PPH treatment as it represents the first controlled study of its kind. While appropriately powered, prospective randomized trials of PAE as a treatment for PPH are of utmost need, these studies are very

difficult to perform, in particular in patients with severe PPH like those included in our study. Until results of such studies are available, we consider PAE as an important and probably safe treatment modality in the armamentarium of PPH management.

Conclusion

In summary, our study suggests that PAE offers a minimally invasive, fertility-preserving alternative to conventional surgical treatment in the management of obstetric hemorrhage [12]. Our results support the view that PAE should have a prominent place in any management algorithm of severe PPH. PAE in PPH seems very effective and has low complication rates. We, therefore, recommend PAE for the treatment of severe postpartum bleeding, provided that the woman is hemodynamically stable and interventional radiology is readily available nearby [12].

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Compliance with ethical standards

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Conflict of interest All authors declare no conflict of interest. The authors alone are responsible for the content and writing of the paper.

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References

1. WHO (2005) The World health report: 2005 make every mother and child count. World Health Organization (WHO), Geneva
2. Devine PC (2009) Obstetric hemorrhage. Semin Perinatol 33:76–81
3. 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
4. Combs CA, Murphy EL, Laros RK (1991) Factors associated with hemorrhage in cesarean deliveries. Obstet Gynecol 77:77–82
5. Feinberg BB, Resnik E, Hurt WG et al (1987) Angiographic embolization in the management of late postpartum hemorrhage: a case report. J Reprod Med 32:929–931
6. 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
7. Bonnar J (2000) Massive obstetric haemorrhage. Baillieres Best Pract Res Clin Obstet Gynaecol 14:1–18
8. Deux JF, Bazot M, Le Blanche AF et al (2001) Is selective embolization of uterine arteries a safe alternative to hysterectomy in patients with postpartum hemorrhage? AJR Am J Roentgenol 177:145–149
9. Joupilla P (1995) Postpartum haemorrhage. Curr Opin Obstet Gynecol 7:446–450
10. Varner M (1991) Postpartum hemorrhage. Crit Care Clin 7:883–887
11. Ekin A, Gezer C, Solmaz U, Taner CE, Dogan A, Ozeren M (2015) Predictors of severity in primary postpartum hemorrhage. Arch Gynecol Obstet 292:1247–1254
12. Gonsalves M, Belli A (2010) The role of interventional radiology in obstetric hemorrhage. Cardiovasc Interv Radiol 33:887–895
13. 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
14. Royal College of Obstetricians and Gynaecologists (2007) The role of emergency and elective interventional radiology in postpartum hemorrhage. Royal College of Obstetricians and Gynaecologists Good Practice Guideline No. 6. Royal College of Obstetricians and Gynaecologists, London
15. Grönvall M, Tikkanen M, Metsätähti M, Loukovaara M, Paavonen J, Stefanovic V (2014) Pelvic arterial embolization in severe obstetric hemorrhage. Acta Obstet Gynecol Scand. doi:10.1111/aogs.12376
16. Tourné G, Collet F, Seffert P, Veyret C (2003) Place of embolization of the uterine arteries in the management of post-partum haemorrhage: a study of 12 cases. Eur J Obstet Gynecol Reprod Biol 110:29–34
17. Chauleur C, Fanget C, Tourne G, Levy R, Larchez C, Seffert P (2008) Serious primary post-partum hemorrhage, arterial embolization and future fertility: a retrospective study of 46 cases. Hum Reprod 23:1553–1559
18. Pelage JP, Le Dref O, Mateo J et al (1998) Life-threatening primary postpartum hemorrhage: treatment with emergency selective arterial embolization. Radiology 208:359–362
19. Ratnam LA, Gibson M, Sandhu C, Torrie P, Chandraharan E, Belli AM (2008) Transcatheter pelvic arterial embolisation for control of obstetric and gynaecological haemorrhage. J Obstet Gynaecol 28:573–579
20. Boulleret C, Chahid T, Gallot D et al (2004) Hypogastric arterial selective and superselective embolization for severe postpartum hemorrhage: a retrospective review of 36 cases. Cardiovasc Interv Radiol 27:344–348
21. 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
22. Chung JW, Jeong HJ, Joh JH et al (2003) Percutaneous transcatheter angiographic embolization in the management of obstetric hemorrhage. J Reprod Med 48:268–276
23. Salomon LJ, deTayrac R, Castaigne-Meary V et al (2003) Fertility and pregnancy outcome following pelvic arterial embolization for severe post-partum haemorrhage. A cohort study. Hum Reprod 18:849–852
24. Knight M, Callaghan WM, Berg C, Alexander S, Bouvier-Colle MH, Ford JB et al (2009) Trends in postpartum hemorrhage in high resource countries: a review and recommendations from the International Postpartum Hemorrhage Collaborative Group. BMC Pregnancy Childbirth 9:55

25. Joseph KS, Rouleau J, Kramer MS, Young DC, Liston RM, Baskett TF (2007) Investigation of an increase in postpartum haemorrhage in Canada. BJOG 114:751–759
26. Callaghan WM, Kuklina EV, Berg CJ (2010) Trends in postpartum hemorrhage: United States, 1994–2006. Am J Obstet Gynecol 202:353.e351–353.e356
27. Rossen J, Økland I, Bjarte Nilsen O, Eggebø TM (2011) Is there an increase of postpartum hemorrhage, and is severe hemorrhage associated with more frequent use of obstetric interventions? Obstet Gynecol Surv 66:18–20
28. Lutomski JE, Byrne BM, Devane D, Greene RA (2012) Increasing trends in atonic postpartum haemorrhage in Ireland: an 11-year population-based cohort study. BJOG 119:306–314
29. Ford JB, Roberts CL, Simpson JM, Vaughan J, Cameron CA (2007) Increased postpartum hemorrhage rates in Australia. Int J Gynecol Obstet 98:237–243
30. Blomberg M (2011) Maternal obesity and risk of postpartum hemorrhage. Obstet Gynecol 118:561–568
31. Mehrabadi A, Liu S, Bartholomew S, Hutcheon JA, Kramer MS, Liston RM, Joseph KS, For the Maternal Health Study Group of the Canadian Perinatal Surveillance System (Public Health Agency of Canada) (2014) Temporal trends in postpartum hemorrhage and severe postpartum hemorrhage in canada from 2003 to 2010. J Obstet Gynaecol Can 36(1):21–33
32. Henrich W, Surbek D, Kainer F et al (2008) Diagnosis and treatment of peripartum bleeding. J Perinat Med 36:467–478
33. American College of Obstetricians and Gynecologists (2006) ACOG practice bulletin: clinical management guidelines for obstetrician–gynecologists number 76, October 2006: postpartum hemorrhage. Obstet Gynecol 108:1039–1047
34. Bateman BT, Berman MF, Riley LE, Leffert LR (2010) The epidemiology of postpartum hemorrhage in a large, nationwide sample of deliveries. Anesth Analg 110:1368–1373
35. Huber AW, Raio L, Alberio L, Ghezzi F, Surbek DV (2012) Recombinant human factor VIIa prevents hysterectomy in severe postpartum hemorrhage. J Perinat Med 40:43–49
36. Doumouchtsis SK, Papageorghiou AT, Arulkumaran S (2007) Systematic review of conservative management of post-partum hemorrhage: what to do when medical treatment fails. Obstet Gynecol Surv 62:540–547
37. 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(1):17–27
38. 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
39. Kim YJ, Yoon CJ, Seong NJ, Kang SG, An SW, Kim YS et al (2013) Failed pelvic arterial embolization for postpartum hemorrhage: clinical outcomes and predictive factors. J Vasc Interv Radiol 24:703–709
40. Flood KM, Said S, Geary M, Robson M, Fitzpatrick C, Malone FD (2009) Changing trends in peripartum hysterectomy over the last 4 decades. Am J Obstet Gynecol 200:632.e1–632.e6
41. Ghezzi F, Cromi A, Uccella S, Raio L, Bolis P, Surbek D (2007) The Hayman's technique: a simple method to treat postpartum haemorrhage. BJOG 114:362–365
42. Zelop CM, Harlow BL, Frigoletto FD et al (1993) Emergency peripartum hysterectomy. Am J Obstet Gynecol 168:1443–1448
43. Vedantham S, Goodwin SC, Mc Lucas B, Mohr G (1997) Uterine artery embolization: an underused method of controlling pelvic hemorrhage. Am J Obstet Gynecol 176:938–948
44. Gaia G, Chabrot P, Cassagnes L et al (2009) Menses recovery and fertility after artery embolization for PPH: a single-center retrospective observational study. Eur Radiol 19:481–487
45. Cottier JP, Fignon A, Tranquart F, Herbreteau D (2002) Uterine necrosis after arterial embolization for postpartum hemorrhage. Obstet Gynecol 100(5 Pt 2):1074–1077
46. 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
47. Ibrahim MI, Raafat TA, Ellaithy MI, Aly RT (2013) Risk of postpartum uterine synechiae following uterine compression suturing during postpartum haemorrhage. Aust N Z J Obstet Gynaecol 53:37–45
48. Schenker JG, Margalioth EJ (1982) Intrauterine adhesions: an updated appraisal. Fertil Steril 37:593–610
49. Conforti A, Alviggi C, Mollo A, De Placido G, Magos A (2013) The management of Asherman syndrome: a review of literature. Reprod Biol Endocrinol 11:118