Treatment of Primary Postpartum Hemorrhage in a Tertiary Perinatal Center

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SUMMARY

The aim of this study was to investigate the causes of primary postpartum hemorrhage (PPPH) and the success of managing it in a tertiary perinatal center.

A clinical retrospective study was conducted at a tertiary perinatal center over a ten-year period (2010–2019). Significant perinatal, maternal, and neonatal data were investigated: maternal and neonatal data, possible causes of hemorrhage and therapeutic approach.

Out of a total of 29 543 deliveries, PPPH was diagnosed in 215 (0.73%) parturients. Uterine atony was diagnosed in 72.09%, uterine rupture in 1.39%, colpaporrhexis in 0.47%, and cervicovaginal rupture in 20% of the parturients. A retained placenta was recorded in 6.19% of the parturients, placenta accreta spectrum disorders in 5.12%, disseminated intravascular coagulation in 3.26%, and obstetric hemorrhagic shock developed in 3.72% parturients. Pharmacotherapy was sufficient for PPPH management in 133 (61.86%) parturients. Abdominal hemostatic procedures (B-Lynch, Cho, O'Leary) were performed in 32 cases (14.88%), and combined hemostatic and ligating methods in 10 (4.65%) parturients. Transvaginal hemostatic procedures were performed in 18.10% of the parturients: hemostatic sutures (Hebisch Huch, Habek, Losickaja) in 14.88%, balloon/gauze tamponade in 1.86%, and combined hemostatic and tamponade procedures in 1.39%. Peripartum hysterectomies were performed in 11 (5.12%) parturients due to refractory bleeding and a morbidly adherent placenta. There were no maternal deaths due to PPPH.

An adequately recognized etiology of PPPH with adherence to therapeutic guidelines directly correlates with the perinatal outcome.

KEYWORDS

Primary postpartum hemorrhage; Etiology; Management; Uterine sparing surgery; Peripartum hysterectomy

Introduction

Early or primary postpartum hemorrhage (PPPH) is defined as blood loss exceeding 500 mL in a vaginal delivery or 1 000 mL during a cesarean section (CS) within the first 24 hours. Alongside complications of hypertensive disorders in pregnancy, sepsis, and embolisms, obstetric hemorrhage remains the leading cause of maternal mortality in both developed and developing countries, representing persistent variables in perinatal epidemiology throughout human history[1–5].

The mnemonic 4T etiopathogenetic classification of postpartum hemorrhage (PPH) causes is well-known: *Tone* disorder of the uterus (atony), *Trauma* (genital lacerations), *Tissue* (residual placental tissue), and *Thrombin* (coagulopathy). The pandemic rise in CS rates in recent decades has led to an increase in placenta previa and/or morbid placentation (placenta accreta spectrum disorders (PASD)), which, alongside uterine atony which still accounts for about 50% of PPH cases, are causes of PPPH and poor maternal outcomes today. The absence of two fundamental physiological peripartum mechanisms — myotamponade and thrombotamponade — are the main risk factors for PPPH development. Hence, the primary therapeutic approach focuses on uterotonic and antifibrinolytic therapy. The therapeutic approach to treating PPPH, alongside existing prophylactic and therapeutic uterotonic therapy, has changed over the past two decades with 1. the introduction of the antifibrinolytic, prophylactic and therapeutic use of tranexamic acid (TXA) and 2. expanding the spectrum of numerous hemostatic compression and ligation surgical procedures (uterine sparing surgery), all aimed at preventing the development of obstetric hemorrhagic shock, and reducing emergency peripartum hysterectomies (EPH) and poor maternal outcomes. These two fundamental procedures are now included in all global guidelines for the management of obstetric hemorrhage, including PPPH, as the most common acute obstetric condition[1–3,5,6].

The aim of this study was to investigate the causes of PPPH and the success of managing it in a tertiary perinatal center.

Materials and methods

This clinical retrospective study was conducted at a tertiary perinatal center, the University Department of Gynecology and Obstetrics at Clinical Hospital "Sveti Duh" in Zagreb, analyzing data spanning a ten-year period (2010–2019). The study was approved by the Ethics Committee of Clinical Hospital "Sveti Duh" (No. 01-1980/2) on July 20, 2020. Out of a total of 29 543 deliveries in the study period, there were 215 deliveries with defined PPPH (> 500 mL during and after vaginal delivery or > 1000 mL during and after CS within 24 hours, following the routine prophylactic procedure of active management of the third stage of labor with an intravenous injection of 5 IU of oxytocin during vaginal delivery and CS).

Upon establishing the diagnosis of PPPH, all patients were prescribed a pharmacologic uterotonic/uterostyptic (oxytocin/methylergometrine) and antifibrinolytic therapy (TXA) and volume replacement with crystalloids and colloids to prevent the development of obstetric hemorrhagic shock, and their vital signs were monitored. In cases where pharmacologic treatment was ineffective, further surgical procedures were employed. Significant perinatal, maternal, and neonatal data were investigated. The maternal data included: age, gestational age, parity, history of previous surgical procedures, gynecological diseases, comorbidities during pregnancy, and peripartum events — labor induction, vaginal birth after cesarean section (VBAC), the incidence of CS, type of anesthesia, premature rupture of membranes, vaginal assisted procedures (episiotomy, vacuum extraction, shoulder dystocia release), placenta previa/abruption, and the

Table 1. Maternal and neonatal data (n = 215)

Age (mean)		20-45 (31.60)
Gestational week (mean)		26-41 (38.7)
Parity (mean)		1-5 (1.61)
Primiparous		125 (58.14%)
Anamnestic data (%)	Previous PPPH	2 (0.93%)
	Previous cesarean section	19 (8.84%)
	Previous uterine operation (myomectomy, hysteroscopy)	4 (1.86%)
	Uterine anomaly	5 (2.33%)
	Medically assisted reproduction	16 (8.84%)
	Twin pregnancy	9 (4.19%)
Comorbidity (%)	Gestational anemia	19 (8.84%)
	Gestational diabetes	26 (12.09%)
	Hypertensive disorders in pregnancy	12 (5.58%)
	Myomatous uterus	6 (2.79%)
	Thrombophilia	5 (2.33%)
	Cholestasis	2 (0.93%)
	Thrombophlebitis	2 (0.93%)
	Oligohydramnios	21 (8.40%)
	Amnioinfection syndrome with panmetritis	1 (0.46%)
Peripartal data (%)	Induced delivery	48 (22.33%)
	Vaginal birth after cesarean	8 (3.72%)
	Cesarean delivery	53 (24.65%)
	urgent cesarean section	18 (8.37%)
	Epidural analgesia	81 (37.67%)
	Spinal anesthesia	53 (25.85%)
	Preterm rupture of membranes	43 (20.0%)
	Uterine inertia	12 (5.58%)
	Episiotomy	58 (29.98%)
	Vacuum extraction	16 (7.44%)
	Shoulder dystocia	8 (3.72%)
	Placental abruption	7 (3.26%)
	Placenta previa	11 (5.11%)
	Placental abnormality (succenturiata, bilobata)	4 (1.86%)
	Obstetric shock development	8 (3.72%)
Neonatal data (%)	Male	107 (47.7%)
	Female	117 (52.23%)
	Birth weight (mean)	750-5020 (3391.0)
	> 4000 grams	33 (14.73%)
	Apgar score 1 min/5 min (mean)	9.69/9.80
	Fetal malpresentation	15 (6.97%)
	Intrapartal hypoxia	14 (6.25%)
	Early neonatal death	1 (0.47%)

development of obstetric shock. Neonatal data included: sex, birth weight, Apgar score, the presence of fetal malpresentation, and intrapartum hypoxia (Table 1). The possible causes of PPPH (Table 2) were analyzed, as well as therapeutic approaches to its treatment (medicamentous, transabdominal, and transvaginal hemostatic compressive or ligating procedures, balloon/gauze tamponade, or combined procedures) and the need for EPH (Table 3). The results are presented as mean values and percentage representations.

Results

Out of a total of 29 543 deliveries, PPPH was diagnosed in 215 (0.73%) parturients. The average age of the parturients was 31.60 years, 58.14% of them were primiparous, and 8.84% conceived using medically assisted reproductive techniques. Previous CS was present in 8.84% of the parturients, gestational anemia in 8.84%, gestational diabetes in 12.09%, and hypertensive disorders in pregnancy in 5.58%. Labor was induced in 22.33% of the parturients,

Table 2. The etiology of primary postpartum hemorrhage (n = 215)

Uterine atony	155 (72.09%)
lower uterine segment atony	44 (29.93%)
Uterine rupture	3 (1.39%)
Colpaporrhexis	1 (0.47%)
Cervical and vaginal rupture	43 (20%)
Retained placenta	13 (6.19%)
Morbidly adherent placenta	11 (5.12%)
Coagulopathy	7 (3.26%)

Table 3. Therapy for primary postpartum hemorrhage (n = 215)

Medicamentous therapy (uterotonics, uterostyptics, antifibrinolitics)	133 (61.86%)
Transvaginal hemostatic procedures	39 (18.10%)
Cervical hemostatic sutures (Losickaja, Habek, Hebish-Huch)	32 (14.88%)
Balloon/gauze tamponade	4 (1.86%)
uterine	2 (0.93%)
cervicovaginal	2 (0.93%)
Combined procedures (hemostatic sutures + tamponade)	3 (1.39%)
Abdominal hemostatic procedures	32 (14.88%)
B-Lynch compressive suture	10 (4.65%)
Cho compressive suture	10 (4.65%)
O'Leary ligature	2 (0.93%)
Combined compressive and ligation procedures	10 (4.65%)
Peripartal hysterectomy	11 (5.11%)
Early relaparotomy (intraabdominal hemorrhage)	6 (2.79%)

vacuum extraction was performed in 7.44% of deliveries, and CS was performed in 24.65% of the parturients. Epidural analgesia was administered to 37.67% of the parturients, preterm rupture of membranes was present in 20%, placenta previa was diagnosed in 5.11%, and placental abruption in 3.26% of the parturients.

Macrosomia was observed in 14.73% of the newborns, intrapartum fetal hypoxia in 6.25%, and one neonate died in the early neonatal period. Uterine atony was diagnosed in 72.09% of the parturients, of which 29.93% had lower uterine segment atony (LUSA). Uterine rupture was found in 1.39%, colpaporrhexis in 0.47%, and cervicovaginal rupture in 20% of the parturients. Retained placenta was recorded in 6.19% of the parturients, PASD in 5.12%, disseminated intravascular coagulopathy (DIC) in 3.26%, and obstetric hemorrhagic shock in 3.72% of the parturients. Conservative medicamentous treatment was sufficient for managing PPPH in 133 (61.86%) parturients. Abdominal hemostatic procedures (B-Lynch, Cho, O'Leary) were performed in 32 cases (14.88%). A combination of hemostatic and ligating methods was successfully performed in 10 (4.65%) parturients. Transvaginal hemostatic procedures were performed in 18.10% of the parturients, including cervical hemostatic sutures (Hebisch Huch, Habek, Losickaja) in 14.88%, balloon/gauze tamponade in 1.86%, and combined hemostatic sutures and tamponade procedures in 1.39% of the parturients.

Peripartum hysterectomies were performed in 11 (5.12%) parturients due to refractory bleeding and PASD. There were no maternal deaths due to PPPH in the studied material. Early relaparotomies due to intra-abdominal postoperative hemorrhage were recorded in 6 (2.79%) cases.

Discussion

A prompt diagnosis of PPPH is the main factor in establishing timely and correct therapeutic interventions. However, despite the existence of guidelines for treating PPPH and the prophylactic use of uterotonic and antifibrinolytic agents, downplaying the condition of PPPH and inaccurate estimates of blood loss are the main factors leading from silent to overt obstetric hypovolemic shock and the development of coagulopathy, acidosis, and hypothermia as aggravating factors of poor maternal outcomes[7]. Therefore, the goal of treating PPPH is to reduce blood loss, prevent the development of obstetric hemorrhagic shock, secondary anemia, and multi-organ damage, prevent hysterectomy, and preserve menstrual cycles and fertility. This applies to prehospital and hospital procedures in acute obstetrics and the prevention of severe conditions requiring treatment in the ICU[3–8]. Pirzada et al.[9] recently showed in their study a 32% admission rate of obstetric patients to the ICU due to respiratory failure, 15% due to PPH complications, and 14% due to sepsis, demonstrating the present-day chronic, unchanged etiology of the admission of obstetric patients to the ICU, and increased maternal morbidity and mortality.

Maher et al.[8] highlighted risk factors significantly associated with PPPH, including increased maternal body mass index, multiparity, assisted reproduction, fetal macrosomia, mode of delivery, and previous PPH. Abecasis et al.[3] reported 1 811 (0.56%) cases of PPPH out of 322 497 deliveries, of which 28% were severe PPH. Independent risk factors for developing severe PPPH include IVF pregnancy, previous CS, preeclampsia, placental abruption, and uterine rupture. An IVF pregnancy, and fetuses large for gestational age are independent risk factors for PPPH following vaginal delivery, while placenta previa is independently associated with PPPH after CS only. In 2013, Kramer et al.[10] published a large study of 8 571 209 deliveries of which 25 906 (3.0/1000) had severe PPH. The rate

rose from 1.9 to 4.2 per 1 000 deliveries from 1999 to 2008, with increases in severe atonic and non-atonic PPH, due especially to PPH with transfusion, but also PPH with hysterectomy. Significant risk factors for the occurrence of PPH include maternal age ≥ 35 years, multiple pregnancies, fibroids, preeclampsia, amnionitis, placenta previa or placental abruption, cervical laceration, uterine rupture, instrumental vaginal delivery, and CS. Placenta accreta spectrum disorders were identified in 46 cases with an incidence of 2.49 per 1 000 births: placenta accreta (56%), increta (39%), or percreta (4%). The CS rate was 65%, and the hysterectomy rate was 39%. In 9% of the cases, the placenta primarily remained in situ. In total, 54% of the patients required blood transfusion. Almost identical risk factors were observed in this study, with 0.73% of PPPH in the examined sample.

The introduction of antifibrinolytic TXA for prophylaxis and the treatment of PPH significantly reduced maternal morbidity and mortality due to hemorrhagic obstetric shock, coagulopathy, and the need for massive transfusions. Treatment with uterotonic agents/uterostyptics and TXA should be initiated immediately upon the diagnosis of PPPH to attempt to establish the missing mechanism of thrombo- and myotamponade[1,2,11–13]. In Croatia, TXA was introduced in 2009 for routine clinical prophylactic and therapeutic use, and a significant reduction in the need for transfusion treatment was observed; however, systematic research on this use has not been conducted so far.

Uterine-sparing surgery is currently the state-of-the-art approach to treating PPPH, and operating on PASD in the “pale pathway” with preoperative use of arterial balloon tamponade or embolization, perioperative ligation of iliac or uterine blood vessels, as well as compressive procedures, significantly reduces blood loss and improves overall perinatal outcomes[12,14,15]. Kong and To compared the management of severe PPH using uterine balloon tamponade and B-Lynch compression sutures with menstrual and reproductive outcomes. They concluded that there was no significant difference in the success of management between these two methods, except that in the management of PPH in the case of placenta previa, uterine balloon tamponade provided better results in regular menstrual cycles and later overall clinical outcomes. Uterine balloon tamponade was more frequently performed after vaginal delivery, with subsequent procedures and EPH being more common in cases of PASD, DIC, and blood loss exceeding 1.5 L[16,17]. German authors established intrauterine tamponade with chitosan as early as 2012 and demonstrated this method's significant success in refractory PPH, reducing hysterectomies and massive transfusions, with recent research confirming these results[18,19]. In the data we analyzed, intrauterine balloon tamponade as a successful hemostatic method was used in 9 parturients, 8 of whom underwent vaginal delivery.

Compression sutures of the uterus have been in use since 1996, with significant success in treating PPPH and drastically reducing EPH while preserving reproduction. Recently, in a review article on the management of PPH with compression sutures, Moleiro et al. concluded that in most women, the menstrual cycle was regular with preserved reproduction and the possibility of conception and pregnancy compared to women in whom this hemostatic method was not applied[20]. We confirmed this in our studies, demonstrating the success of B-Lynch compression sutures and successful pregnancies[21,22]. Selective devascularization by the ligation of colpo-uterine or larger pelvic blood vessels in severe, refractory PPPH is also a highly successful method of treating PPPH and preserving the uterus[12,15,23]. In a study on the impact of bilateral uterine artery ligation, Verit et al.[24] demonstrated that this procedure did not have a negative effect on ovarian reserve markers 6 months postpartum, or on uterine vascularization.

Emergency peripartum hysterectomies are most commonly performed in refractory forms of PPPH or uterine wall destruction due to PASD

or morbidly adherent placenta previa, with major risk factors being PASD, previous CS, and current CS[12,15,25]. Vural et al.[26] identified abnormal placentation as the main indication for EPH, while CS and previous CS were significant risk factors for EPH. This is supported by our own study on EPH conducted at a tertiary center 15 years ago[27]. In a study by de Gregorio et al.[28], EPH was performed at a rate of 12.8 per 10 000 deliveries with a maternal mortality rate of 2.4%, with death occurring after CS in 80.5% of cases, due to abnormal placentation in 53.7%, uterine atony in 26.8%, uterine rupture in 14.6%, and uterine infection in 4.9% of cases. The median number of transfused products was 11 packed red blood cells, 10 fresh frozen plasma units and 2 platelet concentrates. Similar research[29] supports risk factors for EPH at a rate of 2.2/1 000 deliveries: abnormal placentation appeared in 49.0% of the cases, uterine atony in 41.2%, and uterine rupture in 9.8%. Maternal morbidity was 25.5%, and mortality was 5.9%. The authors of that study concluded that abnormal placentation and previous CS are risk factors for EPH with significant maternal morbidity and mortality. A study by Kallianidis and colleagues[30] on the epidemiology of EPH in 9 developed European countries showed that uterine atony and PASD were the main indications, each with an incidence of about 35%, along with CS delivery and delivery after CS. The prevalence ranged from 2.6/10 000 births in Denmark to 10.7/10 000 births in Italy, with a mean of 5.2/10 000 births. A recent study by de Vries et al.[31] presented the outcomes of severe PPH (blood loss > 2.5 L) comparing France and the Netherlands. The incidence of adverse maternal outcomes after vaginal birth was 9.1% in the Netherlands versus 41.12% (88/214) in France. Hemostatic agents, such as fibrinogen and second line uterotonics, and uterine-sparing interventions were less frequently applied in the Netherlands after vaginal birth (394/1 002, 39.3% vs 134/214, 62.6%) and CS (133/342, 38.9% vs 155/270, 57.4%). Relaparotomies due to intra-abdominal bleeding are a rare incidence following severe PPH, most commonly performed due to technical difficulties during surgery, DIC, preeclampsia, an urgent CS, and significant blood loss necessitating massive transfusions due to the development of obstetric hemorrhagic shock. In our study, 6 relaparotomies were performed for postoperative intra-abdominal hemorrhage after CS, and a group of researchers[32] found that the mentioned risk factors and a blood loss of > 500 mL during the initial CS were independently associated with an increased risk of relaparotomy for intra-abdominal hemorrhage.

Based on years of research on PPPH and published literature so far, and following global obstetric guidelines, the first B-Lynch compressive suture was introduced in Croatia. Our own technique of transvaginal cervical suture in cases of LUSA was demonstrated, and therapeutic options for treating LUSA were showcased, with TXA being introduced in 2009 for prophylaxis and the treatment of PPPH. It is worth mentioning that distinguishing between corporeal atony and LUSA is necessary, as their therapeutic principles differ. This has been illustrated in our work, aligning with research that supports the distinction between these two entities based on etiology and therapy[33-37]. Peri-partum curettage for treating PPPH was discontinued, as there were no indications for this procedure, nor was there a need to negate the effect of thrombotamponade with antifibrinolytics.

According to this study, our hospital achieved a therapeutic effect in 61.86% of cases with medical therapy, resolved PPPH in 17.20% of cases with a transvaginal surgical approach, and in 14.88% of cases with a transabdominal approach, adhering to the principles of uterine-sparing interventions (intrauterine tamponade, compression sutures, vascular ligation). An emergency peripartum hysterectomy was performed in 11 instances of PPH (5.11%)[38]. Pelvic (uterine) embolization was not conducted due to logistical reasons concerning the organization of interventional radiology.

Conclusion

Education on the medical and surgical treatment of PPPH was provided to midwives, residents, and specialists through continuous learning courses, and new recommended methods from the literature were introduced into professional guidelines. This is considered essential, because continuous work on knowledge and skills through refresher courses positively correlates with an early identification and diagnosis of PPPH, early implementation of procedures, and outcomes. However, despite recommendations and known risk factors for PPPH and EPH — such as CS and PASD — globally, the number of CS cases continues to rise unreasonably and antianthropologically. Therefore, it can be concluded that we will continue to induce increases in PPPH and maternal mortality, and consequently disrupt overall perinatal statistics through iatrogenically conditioned procedures. An adequately recognized etiology of PPPH with adherence to therapeutic guidelines directly correlates with the outcome in the mother.

DECLARATION OF COMPETING INTEREST The authors declare that they have no conflict of interest.

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SAŽETAK

Liječenje primarnog poslijeporođajnog krvarenja u tercijarnom perinatalnom centru

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Cilj ovog istraživanja bio je istražiti uzroke primarnog postporođajnog krvarenja (PPPH) i uspješnost njegova zbrinjavanja u tercijarnom perinatalnom centru.

Provedeno je kliničko retrospektivno istraživanje u tercijarnom perinatalnom centru tijekom desetogodišnjeg razdoblja (2010. – 2019.). Analizirani su perinatalni, maternalni i neonatalni podaci: podaci o rodilji i novorođenčetu, mogući uzroci krvarenja te terapijski pristup.

Od ukupno 29.543 poroda, PPPH je dijagnosticiran kod 215 (0,73 %) rodilja. Atonija maternice dijagnosticirana je u 72,09 % slučajeva, ruptura maternice u 1,39 %, kolpaporeksis u 0,47 %, a cervikovaginalna ruptura u 20 % rodilja. Zaostala posteljica zabilježena je kod 6,19 % rodilja, morbidno srasla posteljica kod 5,12 %, diseminirana intravaskularna koagulopatija kod 3,26 %, dok se opstetrički hemoragijski šok razvio kod 3,72 % rodilja. Farmakoterapija je bila dovoljna za zbrinjavanje PPPH-a kod 133 (61,86 %) rodilje. Abdominalni hemostatski postupci (B-Lynch, Cho, O'Leary) provedeni su u 32 slučaja (14,88 %), a kombinirane hemostatske metode i metode ligacije kod 10 (4,65 %) rodilja. Transvaginalni hemostatski postupci provedeni su kod 18,10 % rodilja: hemostatski šavovi (Hebisch Huch, Habek, Losickaja) u 14,88 % slučajeva, tamponada balonom ili gazom u 1,86 % te kombinirani hemostatski postupci i tamponada u 1,39 % slučajeva. Peripartalne histerektomije provedene su kod 11 (5,12 %) rodilja zbog refraktornog krvarenja i morbidno srasle posteljice. Nije bilo smrtnih ishoda kod majki uzrokovanih PPPH-om.

Pravilno prepoznavanje etiologije PPPH-a uz pridržavanje terapijskih smjernica izravno je povezano s perinatalnim ishodom.

KLJUČNE RIJEČI

Primarno poslijeporođajno krvarenje; Etiologija; Liječenje; Poštedna kirurgija maternice; Peripartalna histerektomija