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REVIEW

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Updates in the perioperative management of postpartum hemorrhage

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Abstract

Background Postpartum hemorrhage (PPH) is the leading cause of maternal death all over the world. It could be primary or secondary with uterine atony being the cause in 80% of cases.

Main body For anticipated PPH, special antenatal preparation for abnormal placentation, inherited coagulation disorders, and Jehovah's Witnesses must be done. Optimal surgical management of placenta accreta is done by scheduling delivery in an appropriate surgical facility, by insertion of prophylactic embolization catheters in the uterine or internal iliac arteries, and by rapid diagnosis of PPH. The obstetric shock index (SI) is highly specific for PPH. Optimal anesthetic management is done by oxygen supply, using warming devices, ensuring wide bore intravenous (IV) access with adequate volume replacement, and blood product preparation. The non-pneumatic anti-shock garment (NASG) could be used as first-aid compression device. Permissive resuscitation, uterotonic administration, tranexamic acid, recombinant active factor seven (VIIa), and lyophilized fibrinogen concentrate are beneficial. Hemostatic reanimation to correct coagulopathy and cell saver auto-transfusion are applied. For unanticipated PPH, guidelines and regular skill training reduce the incidence of severe PPH.

Conclusions Anticipated PPH requires antenatal preparation, optimal anesthetic management with the implementation of permissive resuscitation, hemostatic reanimation, and optimal surgical management.

Keywords Postpartum hemorrhage, Obstetric shock index, Permissive resuscitation, Hemostatic reanimation

Background

In 2017, the American College of Obstetricians and Gynecologists (ACOG) defined PPH as blood loss ≥ 1000 ml or blood loss that is accompanied by symptoms or signs of hypovolemia, occurring within 24 h from delivery (Escobar et al. 2022). Severe maternal morbidity is indicated by intensive care unit (ICU) admission or massive transfusion of blood products. PPH accounts for half of obstetric ICU admissions (Carvajal et al. 2022).

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Main text

Classification of postpartum hemorrhage

Primary postpartum hemorrhage

It occurs within 24 h of delivery. It is due to the five Ts: tone (uterine atony; 80% of cases), trauma (lacerations or genital tract trauma), tissue (retained placenta), thrombin (abnormalities of coagulation), and turned inside out (uterine inversion). Also, serotonin-norepinephrine reuptake inhibitor (SNRI) intake in late pregnancy was associated with 1.6–1.9-fold increased risk of PPH. Factors increasing the incidence of uterine atony are as follows: maternal obesity, multiple gestations, advanced maternal age, increasing inductions of labor, and increasing cesarean sections (CS) (Hanley et al. 2016).

Secondary postpartum hemorrhage

It develops after 24 h of delivery. It is mostly due to severe infection leading to uterine atony, retained products

of conception, subinvolution of the placental site, and inherited coagulation defects (Abdul-Kadir et al. 2014).

Anticipated postpartum hemorrhage

- Pre-operative parturient's hematocrit < 32% should be treated by iron or erythropoietin to reduce the risk of peripartum blood transfusion (Beverley et al. 2015).
- Special antenatal preparation for:

1. *Abnormal placentation (placenta accreta, increta, and percreta)*: The estimated intra-partum blood loss is > 2.5 L in 60% of parturients, > 5 L in 22% of parturients, and > 10 L in 12% of parturients (Wright et al. 2011). Antenatal detection of placenta accreta is by ultrasonography in the first or second trimester, especially for parturients with prior uterine surgery. Magnetic resonance imaging (MRI) can be done to confirm the diagnosis when ultrasound is inconclusive and to detect the extent of invasion to the surrounding organs in case of placenta percreta (Weiniger et al. 2013). Parturients with suspected placenta accreta should be scheduled for delivery in an appropriate surgical facility, with a blood bank that can facilitate transfusion of large amounts of blood products (Eller et al. 2011).
2. *Inherited coagulation disorders*: History of postpartum or perioperative hemorrhage, significant menorrhagia, gingivorrhagia, or epistaxis helps to identify parturients at increased risk of PPH. Also, family history of inherited coagulopathy necessitates hematological consultation and available blood bank for optimal management. Ninety percent of inherited bleeding disorders are due to Von Willebrand disease, hemophilia A and B carriers, factor XI deficiency, and idiopathic thrombocytopenic purpura. Rare causes include inherited platelet disorders (Bernard-Soulier syndrome, Glanzmann thrombasthenia) (Abdul-Kadir et al. 2014).
3. *Acquired coagulation disorders*: This results from anticoagulant therapy, the occurrence of disseminated intravascular coagulopathy (DIC) after placental abruption, severe pre-eclampsia, intrauterine fetal death, sepsis, or amniotic fluid embolism (Evensen et al. 2017).
4. *Jehovah's Witnesses*: Antepartum consultation to review blood products alternatives and blood conservation strategies. Antepartum iron and erythropoietin are often acceptable to optimize the hematocrit, prior to delivery, and may be continued with PPH (Belfort et al. 2010).

Perioperative management of PPH

Rapid diagnosis of PPH is essential to successful management (Table 1). The Maternal Early Warning System suggests close monitoring, if the heart rate is > 120 bpm. An obstetric shock index (SI), heart rate in relation to systolic blood pressure (HR/SBP) ≥ 0.9, is highly specific for PPH, associated with an increased risk of blood transfusion, and is a reliable indicator of adverse maternal outcomes (El Ayadi et al. 2016).

Laboratory investigations

➢ Serial hematocrit, serum ionized calcium, serum potassium, serum creatinine, and arterial blood gas
➢ Serial coagulation tests to assess the development of coagulopathy. Conventional laboratory tests, platelets count, prothrombin time (PT), partial thromboplastin time (PTT), and fibrinogen level. Point of care testing (POCT) of viscoelastic coagulation by thromboelastography (TEG) and rotational thromboelastometry (ROTEM) (Carvajal et al. 2022)

Optimal anesthetic approach

➢ Oxygen supply by face mask at 10–15 l/min
➢ Ensure wide bore IV access and warming devices to ensure normothermia

Table 1 Clinical findings in PPH (Schuurmans et al. 2000)

Blood loss (ml)	SBP (mmHg)	Symptoms and signs	Degree of shock
500–1000	Normal	Tachycardia, palpitations, dizziness	Compensated
1000–1500	80–100	Weakness, tachycardia, sweating	Mild
1500–2000	70–80	Restlessness, pallor, oliguria	Moderate
2000–3000	50–70	Collapse, air hunger, anuria	Severe

SBP Systolic blood pressure

➢ Padding and positioning to prevent nerve compression injury

➢ Pre-operative antibiotic prophylaxis 1 h prior to surgery, repeated if surgery is prolonged ≥ 3 h or if severe bleeding occurs (Mercer et al. 2010)

➢ Use of the non-pneumatic anti-shock garment (NASG) as temporary compression device: it reduces the blood loss allowing stabilization of the hypovolemic shock, until blood transfusion, definitive surgical intervention, or transfer to a higher level facility. It is recommended by the World Health Organization (WHO) and the International Federation of Gynecology and Obstetrics (FIGO) in severe PPH. The pressure applied by the NASG is 20–40 mmHg. Direct compression of the abdomen, pelvis, and descending aorta decreases pelvic organ perfusion, thus increasing vital organ perfusion (heart, lungs, brain) and increasing the cardiac output, with decreasing of bleeding from the uterine arteries and the mesenteric vessels (Althabe et al. 2020)

➢ Permissive resuscitation with a mean arterial blood pressure of 50–60 mmHg helps to limit blood loss following delivery. It decreases the multiple organ dysfunction syndrome (MODS) and the acute respiratory distress syndrome (ARDS). Increased IV fluids leads to decreased concentrations of fibrinogen, hemoglobin, and platelets, with prolonged PT and PTT (Gillissen et al. 2018)

➢ IV volume replacement with initial crystalloids resuscitation: it decreases blood viscosity and improves the peripheral perfusion while maintaining oxygen delivery. However, excessive crystalloids resuscitation can result in dilutional coagulopathy and decreased oncotic pressure. Five hundred milliliters of bolus of balanced crystalloids (Ringer's lactate) is administered, owing to the risk of hyperchloremic acidosis and worsening of kidney function with normal saline (Pacheco et al. 2016)

➢ Hemostatic reanimation is early and aggressive blood product replacement: packed red blood cells (PRBC), fresh frozen plasma (FFP), and platelets (PLT) to correct the coagulopathy (Carvajal et al. 2022). Hemoglobin transfusion threshold is 7 g/dl; however, transfusion should proceed without waiting for the laboratory results. Transfusion ratios (FFP to PRBC and platelet to PRBC ratios of 1:1 to 1:2) increase survival in massively transfused patients. Evidence from the trauma literature suggests that the beneficial effect of plasma is in the first 2 h of resuscitation. The inclusion of platelets in massive transfusion occurs with transfusion of 8 units of PRBC. A low Fibtem A5 early in PPH is an indicator of platelet consumption and that the need of platelet transfusion is increased (McNamara et al. 2015; Jones et al. 2016)

➢ Ensure sufficient blood product supply: the blood bank delivers blood products in rounds to the operating theater. Rounds commonly consist of 6 units PRBC, 6 units FFP, 6 units PLT or 1 platelet apheresis, and 10 units of cryoprecipitate; the blood bank prepares and delivers 2–4 successive rounds of blood products until being inactivated (Kogutt and Vaught 2019)

➢ Lyophilized fibrinogen concentrate: 2–4 g maintain the fibrinogen level > 2 g/L. Most fibrinogen replacement is done with cryoprecipitate; one unit of cryoprecipitate contains 2 g fibrinogen/100 ml; 10 units of cryoprecipitate is estimated to raise the serum fibrinogen by 100 mg/dl (Cortet et al. 2012; Spahn et al. 2019)

➢ Damage control resuscitation (DCR); strategies to minimize PPH in parturients who may not survive surgery. It aims to prevent coagulopathy, acidosis, and hypothermia, with maximizing tissue oxygenation. It focuses on permissive resuscitation, hemostatic reanimation, and bleeding control by damage control surgery (DCS) with minimal operative time and damage control interventional radiology (DCIR) (Carvajal et al. 2022)

➢ The anesthetic technique, combined spinal epidural or epidural anesthesia, allows the mother to be awake for the delivery and may be extended for prolonged surgery. On the other hand, general anesthesia (GA) is preferred for cases with massive transfusion in the event of airway edema, fluid overload with pulmonary edema, or transfusion-associated lung injury (TRALI). Induction of GA can be done with 1.5–2 mg/kg of propofol; however, it has a vasodilator effect; 0.2–0.3 mg/kg of etomidate can be used, as it has less hemodynamic effect than propofol; however, it may also cause some vasodilation and hypotension, so 0.5–1 mg/kg of ketamine is better used, as it causes vasoconstriction. One to 1.5 mg/kg of succinylcholine as a muscle relaxant is used, unless contraindicated; for history of malignant hyperthermia or hyperkalemia, 0.6–1.2 mg/kg of rocuronium bromide can also be used. Maintenance of GA is done by 1% sevoflurane, as volatile anesthetics interfere with the uterine contraction. Total intravenous anesthesia (TIVA) with propofol infusion avoids the utero-relaxation of inhalational anesthetics (Ring and Landau 2019). The decision about the anesthetic technique weighs the anticipated blood loss, the operative plan, the availability of anesthesia team to assist with unplanned conversion to GA, and the anticipated

risk of difficult airway. For Jehovah's Witness parturients, epidural anesthesia is preferred, as an awake parturient may change her mind on facing impending death. Uterine inversion requires anesthesia and uterine relaxation to facilitate manual replacement (Weiniger et al. 2005). The effect of obstetric anesthesia on increasing the incidence of PPH was related to GA (Butwick et al. 2017); however, several studies found no influence of obstetric anesthesia on PPH or its severity (Anderson et al. 2022).

Drug therapy

The drug therapy used is as follows: uterotonic agents, prophylactic 10 IU oxytocin (IV/IM), followed by 5–10 IU/h IV infusion over 2 h. Infusion rate is ≥ 60 IU/h which increases the risk of oxytocin cold chain: serious hypotension and myocardial ischemia. Persistent bleeding with maximal oxytocin infusion indicates the use of second-line agents: Methergine 200 mcg IM if the parturient is not hypertensive, repeated once after 15 min; misoprostol 800–1000 mcg rectal or buccal; and prostaglandin F2α 250 mcg IM every 15–20 min up to 8 total doses, avoided in asthmatic parturients. The Society of Obstetricians and Gynecologists of Canada (SOGC) reinforced the use of carbetocin as a first-line uterotonic for PPH prevention. Also, carbetocin promotes blood coagulation (Gallos and Coomarasamy 2019).

The anti-fibrinolytic agent tranexamic acid, 1 g over 10 min IV on diagnosis of PPH and within 3 h of delivery, has wide therapeutic index; so, it can be repeated after 30 min if bleeding continues or if bleeding restarts within 24 h of the first dose; its effect lasts for 7–8 h (Althabe et al. 2020; Escobar et al. 2022).

Recombinant VIIa, dose ≤ 90 mcg/kg, is reported to have an 80% success rate to control PPH (Alfirevic et al. 2007).

Cell saver auto-transfusion

Cell saver with leukocyte reduction filters results in effective clearance of fetal squamous cells, phospholipid lamellar bodies, plasma heparin, cytokines, and other coagulopathic mediators. The use of a leukocyte depletion filter is associated with acute hypotension at the time of transfusion of cell-salvaged erythrocytes. Cell-salvaged blood contains up to 2% fetal red blood cells; rhesus negative parturients require anti-D administration. It is acceptable for Jehovah's Witness parturients (Barth et al. 2011; Rogers et al. 2013).

Optimal surgical management

Controlled cord traction (CCT) is the recommended method for placental delivery with CS. In cases of extensive placenta accreta, optimal surgical management is directed towards delivering the neonate, then closing the uterus with the placenta left in situ, followed by postpartum methotrexate for placental involution (Sentilhes et al. 2016).

Uterine conservation techniques include prophylactic pre-operative uterine and internal iliac artery balloon catheters insertion for embolization. Potential complications include the following: insertion site hematoma, abscess, tissue infection, and necrosis (Mercer et al. 2010). CS in the interventional radiology unit avoids catheter dislodgement and thus improves intra-arterial occlusion efficacy (Jeffrey and Clark 2011).

Uterine balloon tamponade (UBT) is the treatment of choice for refractory uterine atony after vaginal delivery (Mahankali 2017).

Stepwise uterine devascularization, pelvic vessel ligation, and uterine compression sutures, up to hysterectomy, must be planned (Mousa et al. 2014; Sentilhes et al. 2016).

Damage control surgery, intra-abdominal or pelvic packing, is indicated in critically ill parturients with persistent bleeding after hysterectomy. Indicators for early implementation of DCR and DCS are as follows: acidosis (base deficit > 8), blood loss > 1500 ml, and hypothermia (temperature < 35 °C). Other important parameters are SBP < 70 mm Hg, maternal blood pH < 7.1, and persistent bleeding despite massive blood transfusion (Schlembach et al. 2018; Carvajal et al. 2022).

Post-operative care

Interdisciplinary care of the coagulation and metabolic abnormalities is implemented. Severe anemia (Hb ≤ 4 g/dl) necessitates post-operative sedation, intubation, neuromuscular relaxants, and thermoregulation, to decrease oxygen consumption. Erythropoietin and iron are used to restore the red cell mass. Erythropoietin requires 48–72 h for a significant reticulocyte response in the peripheral blood and 10–14 days to increase hemoglobin levels (Rossi et al. 2010; Carvajal et al. 2022).

Unanticipated PPH

Guidelines and regular skill training decrease the incidence of severe PPH and are recommended by the Centre for Maternal and Child Enquiries in the UK. Simulation-based training for obstetric hemorrhage can reveal management deficits and facilitates targeted quality improvement. Calling for the obstetrician and the anesthesiologist within 10 min, administering oxytocin within 10 min, and exploring the uterus within 20 min decrease the incidence of severe hemorrhage from the uterine atony following vaginal delivery. Prompt placement of uterine compression sutures within 1 h of delivery

decreases the risk of peripartum hysterectomy. Group calling systems request an entire Obstetric Medical Emergency Team. An obstetric hemorrhage cart stores essential equipment. An obstetric hemorrhage drug pack containing fentanyl, oxytocin, methylergonovine, and a prostaglandin allows for efficient retrieval in the event of an emergency (Shields et al. 2011).

Unanticipated PPH with vaginal delivery can be due to vaginal lacerations. Surgical repair of laceration can be done with an indwelling epidural catheter or with small doses of intravenous opioids and local anesthesia to the repair site. For parturients with no indwelling epidural catheter, or PPH with more extensive surgical interventions, neuraxial anesthesia is avoided; however, induction of GA carries the risk of aspiration of gastric contents as parturients are considered to have delayed gastric emptying. In addition, the risk for difficult or failed intubation is present due to the expulsive Valsalva maneuvers, resulting in an edematous airway immediately after delivery (Ring and Landau 2019). For unanticipated PPH during CS, general guidelines and readiness for induction of GA as for anticipated PPH apply.

Conclusions

Optimal anesthetic and surgical management for PPH starts by antenatal detection of high-risk parturients, proper selection of institutional facility for delivery, early diagnosis, appropriate drug therapy, and hemostatic reanimation.

Abbreviations

ACOG	American College of Obstetricians and Gynecologists
ARDS	Acute respiratory distress syndrome
CCT	Controlled cord traction
CS	Cesarean sections
DCR	Damage control resuscitation
DCS	Damage control surgery
DCIR	Damage control interventional radiology
DIC	Disseminated intravascular coagulopathy
FFP	Fresh frozen plasma
FIGO	International Federation of Gynecology and Obstetrics
GA	General anesthesia
HR	Heart rate
ICU	Intensive care unit
IV	Intravenous
MODS	Multiple organ dysfunction syndrome
NASG	Non-pneumatic anti-shock garment
PLT	Platelets
POCT	Point of care testing
PPH	Postpartum hemorrhage
PRBC	Packed red blood cells
PT	Prothrombin time
PTT	Partial thromboplastin time
ROTEM	Rotational thromboelastometry
SBP	Systolic blood pressure
SI	Shock index
SNRI	Serotonin-norepinephrine reuptake inhibitor
SOGC	Society of Obstetricians and Gynecologists of Canada
TEG	Thromboelastography
TIVA	Total intravenous anesthesia
TRALI	Transfusion-associated lung injury
UBT	Uterine balloon tamponade
VIIa	Active factor seven

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