American Journal of Emergency Medicine 103 (2026) 65–72

Contents lists available at ScienceDirect

American Journal of Emergency Medicine

journal homepage: www.elsevier.com/locate/ajem

High risk and low incidence diseases: Postpartum hemorrhage

Gianna Petrone [a], Amy Mariorenzi [a], Alex Koyfman [b], Brit Long [c,*]

[a] Department of Emergency Medicine, Brown University Health & Warren Alpert Medical School of Brown University, USA
[b] Department of Emergency Medicine, UT Southwestern, Dallas, TX, USA
[c] Department of Emergency Medicine, University of Virginia School of Medicine, Charlottesville, VA, USA

ARTICLE INFO

Article history:
Received 12 November 2025
Received in revised form 4 January 2026
Accepted 28 January 2026

Keywords:
Obstetrics
Gynecology
Postpartum
Hemorrhage
Bleeding
Shock
Postpartum hemorrhage

ABSTRACT

Introduction: Postpartum hemorrhage (PPH) is a leading cause of maternal morbidity and mortality worldwide, with rising incidence in the United States. Diagnosis can be particularly challenging in the emergency department (ED), where time-sensitive decisions are required and maternal physiologic adaptations may obscure early warning signs. Emergency physicians must be equipped to recognize subtle presentations, initiate resuscitation, and coordinate care across specialties.
Objective: This review highlights the pearls and pitfalls of PPH, including presentation, diagnosis, and management in the ED based on current evidence.
Discussion: PPH presents variably, and patients can deteriorate rapidly, with pregnancy-related physiologic changes often masking early signs of blood loss. Emergency physicians should consider this diagnosis in appropriate clinical scenarios, recognizing that hypotension is a late finding and subtle symptoms may represent early hemorrhage. Initial evaluation begins with resuscitation, followed by identifying the etiology using the "4 Ts" framework: tone (uterine atony), tissue (retained placenta), trauma (tears, lacerations, uterine inversion), and thrombin (coagulation abnormalities). Pelvic examination is essential, with laboratory studies and ultrasound serving as adjuncts to confirm retained tissue, uterine rupture, or intra-abdominal bleeding. Timely recognition and targeted intervention are critical to preventing morbidity and mortality.
Conclusion: Emergency physicians must remain prepared to rapidly identify and treat PPH, as early recognition and intervention are vital to survival. Applying the "4 Ts" framework and engaging a multidisciplinary team are keys to reducing morbidity and mortality in this high-risk condition.

1. Introduction

This article series highlights high risk but uncommon diseases encountered in the emergency department (ED). For many of these conditions, the primary literature is limited and not focused on emergency medicine, leaving emergency physicians with little direct evidence to guide diagnosis and management. Each article defines the disease or clinical presentation, reviews the current states of knowledge, and concludes with practical pearls and pitfalls presented in question-and-answer format. This review specifically focuses on the ED recognition and management of postpartum hemorrhage (PPH), emphasizing rapid diagnostic strategies, ED relevant imaging considerations, critical decision points, and time-sensitive escalation pathways unique to emergency care.

1.1. Definition

PPH is the leading cause of maternal death worldwide and accounts for approximately 11–12% of maternal deaths in the United States [1,2]. PPH is classified as either primary, occurring within the first 24 h after delivery, or secondary, occurring 24 h to 12 weeks postpartum [3]. While definitions of primary PPH have varied across organizations, the American College of Obstetricians and Gynecologists (ACOG) most recently defined it as blood loss exceeding 1000 mL associated with signs or symptoms of hypovolemia, following either spontaneous vaginal delivery (SVD) or cesarean section [3].

1.2. Pathophysiology

During pregnancy uterine blood flow increases dramatically, reaching to nearly 600 mL per minute at full-term, compared with only about one-tenth that amount in the non-pregnant state [4]. The profound maternal physiologic changes are critical to consider when evaluating a patient with PPH. Pregnancy is associated with increased

* Corresponding author.
E-mail address: Brit.long@yahoo.com (B. Long).

https://doi.org/10.1016/j.ajem.2026.01.048

circulating red blood cell mass, total blood volume, and cardiac output, all of which can initially mask the classic vital sign changes seen with hemorrhage, making timely recognition of PPH especially challenging [5-7].

Immediately following delivery, mechanical and hormonal changes trigger contraction of the uterine myometrium, reducing uterine size and volume, prompting placental separation, and decreasing blood flow [8]. At the same time, a series of complex hemostatic adaptations occur, including increased production of clotting factors, alterations in platelet activity, and enhanced fibrinolysis [8].

The etiology of PPH is classically categorized into the "4 Ts": tone (uterine atony), tissue (retained placenta), trauma (tears, lacerations, uterine inversion), and thrombin (coagulation abnormalities) [4]. Uterine atony is the most common cause, accounting for nearly 70% of cases. Trauma-related hemorrhage, such as lacerations, represents approximately 20% of cases, while retained placental tissue contributes roughly 10% [9]. Coagulopathy and uterine inversion are rare but important considerations that should not be overlooked [3].

If not appropriately recognized and managed, PPH can result in life-threatening complications including severe anemia, disseminated intravascular coagulopathy (DIC), multisystem organ failure, acute respiratory distress syndrome (ARDS), and ultimately death [3].

1.3. Epidemiology

Risk factors for PPH are commonly grouped into three main categories: maternal, pregnancy-related, and fetal. Notable contributors include uterine anomalies, prior uterine surgery, high parity, multiple gestation, placental abnormalities, and prolonged labor [10]. However, a substantial proportion of patients who develop PPH have no identifiable risk factors, underscoring the importance of maintaining a high index of suspicion in every case [11].

The incidence of PPH has been rising both in the U.S. and globally. In the U.S., rates increased from 2.7% in 2000 to 4.3% in 2019. This trend is thought to reflect a parallel rise in underlying risk factors for PPH, particularly the increased prevalence of cesarean deliveries and placenta accreta spectrum (PAS) disorders. Despite the overall increased rates of PPH, the use of blood transfusion and need for peripartum hysterectomy have declined in recent years [12].

Consistent with broader patterns of racial disparities in maternal health [13], minority women including Black, Asian, and Hispanic patients continue to experience disproportionately higher rates of PPH, as well greater risks of severe maternal morbidity and mortality related to hemorrhage [10].

2. Discussion

2.1. Presentation

The clinical presentation of PPH is highly variable, and it should remain on the differential for any patient presenting to the ED during labor or following a precipitous delivery, given its unpredictable onset and potential for rapid decompensation [14]. Pregnancy-related physiologic adaptations can further obscure early signs of hemorrhage: up to one liter of blood may be concealed within the uterus, and compensatory mechanisms may initially preserve normal vital signs [15]. As a result, hypotension represents a late and worrisome indicator of severe hemorrhage. Clinicians should maintain a high index of suspicion and avoid dismissing subtle symptoms such as anxiety, lightheadedness, fatigue, or pallor which may represent the earliest signs of ongoing blood loss [16].

2.2. ED evaluation

The initial evaluation of suspected PPH begins with a focused assessment of the patient airway and hemodynamic status, recognizing that even subtle abnormalities may indicate impending decompensation. This should be followed by a prompt review of vital signs and immediate establishment of large-bore intravenous (IV) access to facilitate immediate resuscitation, laboratory evaluation, and preparation for potential transfusion [17]. Importantly, vital signs must always be interpreted in the context of pregnancy-related physiologic changes, which can mask early hemodynamic instability.

Interpretation of vital signs in patients with PPH must account for pregnancy-specific physiologic norms, as reliance on nonpregnant thresholds may delay recognition of maternal deterioration. Literature suggests that blood pressure does not significantly decrease during pregnancy, and systolic blood pressure is rarely below 95 mmHg in any trimester [18]. As a result, relative hypotension, narrowing pulse pressure, or systolic blood pressure below 100 mmHg should be considered abnormal and concerning in pregnant and postpartum patients, particularly in the setting of suspected hemorrhage, even when absolute values appear deceptively normal [18]. Heart rate interpretation also requires caution, as mild tachycardia is common in normal pregnancy, with median heart rates increasing from the low 80s early in gestation to the low 90s near term. However, persistent heart rates above 110–115 beats per minute or a rising trend out of proportion to pain, fever, or exertion should raise concern for occult hemorrhage and early shock in the postpartum patient [18]. Emergency physicians should prioritize physiologic trends over isolate measurements when evaluating for PPH.

Accurate assessment of hemorrhage severity is foundational to ED decision-making, yet this remains one of the most challenging aspects of PPH recognition [19,20]. Visual estimation of blood loss is unreliable and consistently underestimates true volume by up to 30%, making early recognition dependent on physiologic changes and serial assessment rather than visual appearance alone. While gravimetric techniques, colorimetric tools, and calibrated drapes are recommended for improved accuracy, these techniques may be difficult to implement in the ED due to time constraints and resource limitations [14,21]. When feasible, even partial use of these tools (e.g., weight pads, visual aids) can aid in earlier recognition of hemorrhage severity and guide more appropriate and timelier interventions [3,19]. In cases where quantification is not possible, clinicians must rely on vital signs and physical examination and maintain a high index of suspicion to avoid under-triaging.

Identification of the underlying etiology of PPH should occur concurrently with resuscitation. The pelvic examination is one of the highest-yield diagnostic interventions and should include both visual inspection and bimanual examination to assess uterine size and tone, evaluate for genital tract trauma, and identify retained placental tissue [21]. Emergency physicians should approach the diagnostic evaluation using the "4 Ts" framework (i.e., Tone, Tissue, Trauma, and Thrombin), while recognizing that multiple etiologies may coexist and that definitive diagnosis may evolve over time.

Uterine atony should be presumed in cases of brisk bleeding with an enlarged, boggy uterus on bimanual examination and treated immediately, as it is the most common and readily reversible cause of PPH. During bimanual examination, one hand is inserted into the vaginal canal and the other is placed on the lower abdomen to assess uterine size and tone. A normal involuting uterus should feel firm and well contracted; a soft or enlarged uterus is highly suggestive of uterine atony [18]. In cases of suspected uterine atony, it is important to ensure the bladder is decompressed, as a full bladder can prevent uterine contraction [20].

If the uterus is firm and bleeding persists, trauma-related hemorrhage should be suspected, prompting careful visual inspection for vaginal, cervical, or perineal lacerations and expanding hematoma. This should include adequate lighting and visual inspection but may require speculum examination [3,11].

Retained placental tissue should be considered in patients with ongoing bleeding, particularly when the placenta is known to be incomplete or delivery occurred precipitously. If the placenta has been delivered, it should be inspected carefully to ensure it remained intact. The bladder should be decompressed with a foley catheter, and any clots should be manually removed [20].

Thrombin-related causes, including coagulopathy, should be suspected when bleeding is disproportionate to examination findings or refractory to initial mechanical and pharmacologic interventions.

Adjunctive diagnostic testing, including laboratory studies and imaging, can further support evaluation but should not delay definitive management. Point-of-care-ultrasound may be useful for identifying intrauterine echogenic material suggestive of retained tissue, uterine rupture, or free intraperitoneal fluid. However, it cannot reliably distinguish retained products from blood clots. Rare but life-threatening etiologies such as uterine rupture or inversion should be considered in patients with severe pain, hemodynamic instability, abnormal uterine contour, or a history of prior uterine surgery. In these cases, emergent obstetric consultation should not be delayed for imaging [18].

Additional diagnostic testing including laboratory studies and imaging can further support the evaluation of patients with suspected PPH. Ultrasound is particularly valuable, as it may reveal retained placental tissue within the uterus, signs of uterine rupture, or evidence of intra-abdominal bleeding [22].

2.3. ED management

Management of PPH in the ED requires rapid, parallel execution of resuscitative and etiologic interventions, including early airway and hemodynamic assessment, large-bore IV access, and mobilization of multidisciplinary resources. Treatment should be directed toward the underlying cause, with particular attention to uterine atony and genital tract trauma, which together account for nearly 90% of cases [11]. In patients with suspected ongoing hemorrhage, therapeutic escalation should follow a time-sensitive decision-making pathway rather than a sequential trial of isolated interventions (Fig. 1). Immediate management includes bimanual uterine massage with concurrent administration of uterotonics agents, early consideration of tranexamic acid (TXA), and initiation of blood products as indicated [3,4,23].

Uterine massage consists of firm pressure applied to the uterine fundus through the abdominal wall (Fig. 2). If hemorrhage persists, bimanual uterine compression should be performed by placing one hand, in the shape of a fist, into the vaginal canal to compress the anterior aspect of the uterus while the other hand is placed on the lower abdominal wall over the uterine fundus externally to apply counter-pressure [24]. Downward traction or massage of the uterus should be avoided, as this may precipitate uterine inversion [24].

Ongoing hemorrhage or hemodynamic instability should prompt early escalation of care, including a low threshold for activation of massive transfusion protocol (MTP). Blood product administration should be guided by physiologic response and anticipated transfusion needs, with emphasis on balanced transfusion of packed red blood cells, plasma, platelets and early fibrinogen replacement via cryoprecipitate of fibrinogen concentrate [17]. Failure of initial medical and mechanical interventions necessitates progression to advanced therapies. These include uterine tamponade devices, consultation with interventional radiology (IR) for uterine artery embolization in hemodynamically stabilized patients, or operative management, such as uterine compression sutures or hysterectomy, in patients with refractory hemorrhage or persistent instability [3,19]. Early involvement of obstetrics, anesthesia, blood bank, and IR is critical and should occur concurrently with resuscitative efforts rather than as a delayed response to treatment failure.

3. Pearls and pitfalls

3.1. How is postpartum hemorrhage defined?

PPH is most commonly defined as a cumulative blood loss ≥1000 mL within 24 h of delivery, or any amount of blood loss associated with signs or symptoms of hypovolemia regardless of the delivery method [3]. In the ED, clinical concern and physiologic change should prompt intervention even before numeric thresholds are reached, given the unreliability of visual blood loss estimation and potential for occult hemorrhage [21]. PPH may present as primary (within the first 24 h) or secondary (24 h to 12 weeks postpartum), and delayed presentations should not be dismissed as benign bleeding [21].

Although uncommon, post-abortion hemorrhage should be considered in patients presenting with significant vaginal bleeding after spontaneous or induced abortion. Risk factors for and etiologies are similar to PPH including uterine atony, trauma, retained products of conception, and coagulopathy [3].

3.2. What are essential components of the initial evaluation?

Early recognition of PPH includes assessment of vital signs, airway, mental status, and the degree of bleeding with physiologic trends rather than absolute values. Failure to recognize high-risk patients, such as those with prior PPH, anemia, multiple gestations, placenta previa, coagulopathy, uterine fibroids, or previous operative delivery, may delay escalation and definitive management [25].

A focused physical examination is essential in identifying the etiology of hemorrhage [3]. Bimanual examination assesses uterine tone, size, and the presence of retained, while visual inspection evaluates for vaginal or cervical lacerations, hematomas, bleeding at the episiotomy site, and signs of uterine inversion. Relying on imaging alone (e.g., ultrasound) without performing a pelvic examination may miss common and immediately treatable causes of hemorrhage.

Focused testing can assist clinicians. Point-of-care-ultrasound (POCUS) can supplement the physical examination by identifying retained products, uterine rupture, or free intraperitoneal fluid [22]. Initial laboratory testing should include complete blood count (CBC), coagulation panel, fibrinogen level, and type and screen or cross match. A fibrinogen level < 200 mg/dL suggests severe hemorrhage or evolving coagulopathy and should prompt early escalation of care. Lactate and renal function are helpful to assess for hypoperfusion and end organ damage but may not be relevant to the initial ED evaluation [11]. Importantly, delaying intervention while awaiting laboratory results, particularly in unstable patients, can worsen outcomes; management should proceed in parallel with diagnostic testing.

3.3. What are the initial management priorities of postpartum hemorrhage in the ED?

Initial management of PPH in the ED requires rapid recognition, immediate resuscitation, and early multidisciplinary activation [17]. Obstetrics, anesthesia, blood bank, and operating room resources should be notified early rather than sequentially [3,17,21]. Delaying consultation or resource activation while attempting prolonged bedside management can result in delayed definitive hemorrhage control.

Clinicians should work quickly to identify and address the “4 T's”: Tone (uterine atony), Tissue (retained placenta/ products), Trauma (laceration or uterine rupture), and Thrombin (coagulopathy). Of these 4 etiologies, uterine atony is the most common and should be treated immediately with bimanual uterine massage and uterotonics.

Early consideration for transfusion initiation with low threshold to initiate MTP and when clinically indicated, balanced transfusion using a 1:1:1 ratio of PRBCs, plasma, and platelets is recommended [3,26,27]. Transfusion should be initiated with uncrossmatched type O- PRBCs unless crossmatched blood has already been prepared and is

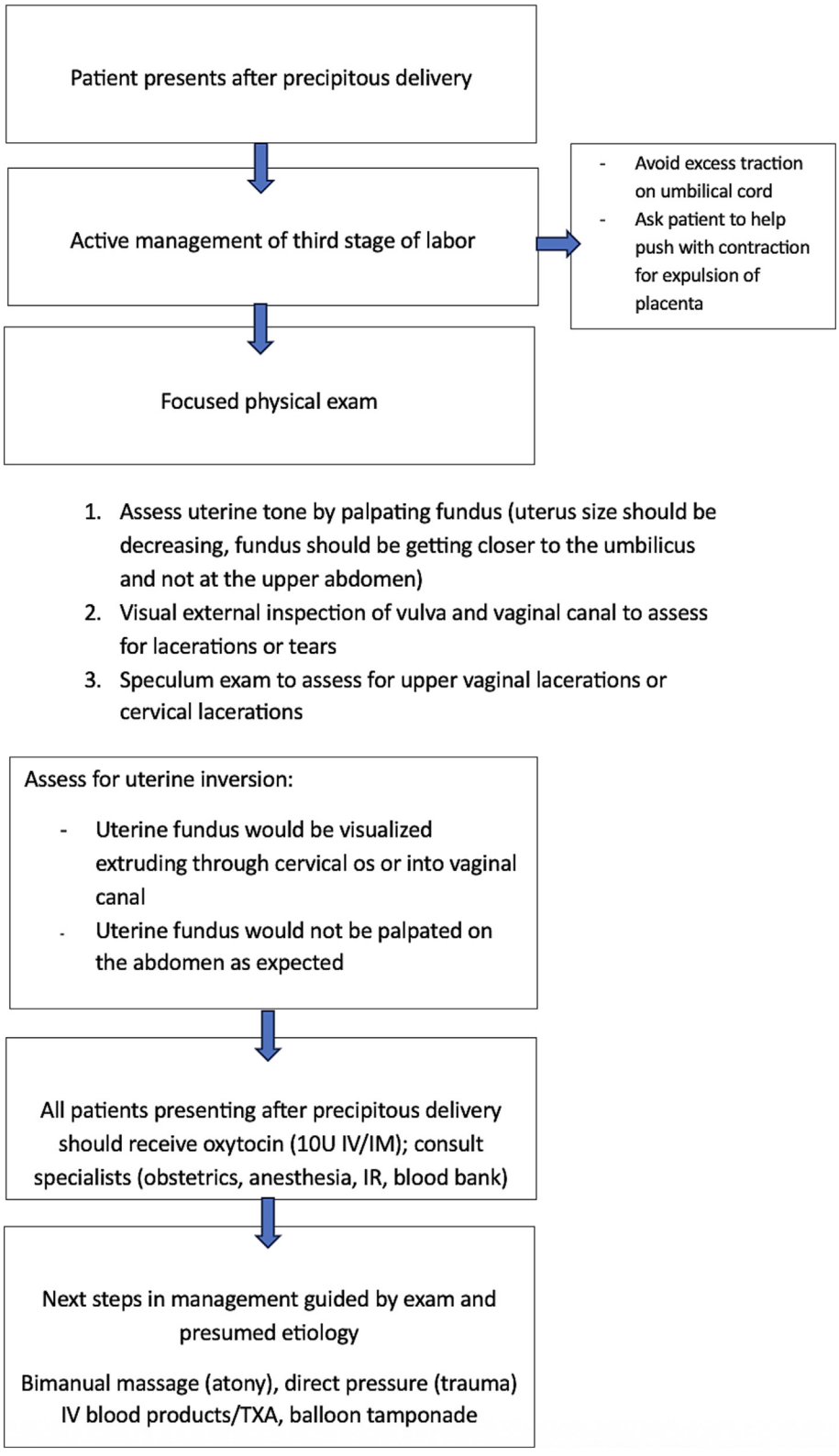


Fig. 1. PPH evaluation.

immediately available. The trigger for initiating MTP is not standardized but is generally recommended after 4 units of PRBCs have been administered [28]. Similar to the management of trauma patients, physicians should consider initiating MTP when there are severe vital sign derangements such as systolic blood pressure < 90 mmHg or sustained heart rates >120 beats per minute [29,30].

3.4. What is the treatment for uterine atony?

Uterine atony is the most common cause of PPH and should be presumed and treated immediately. Bimanual uterine massage remains the first-line intervention and can be initiated rapidly at the bedside with minimal resources.

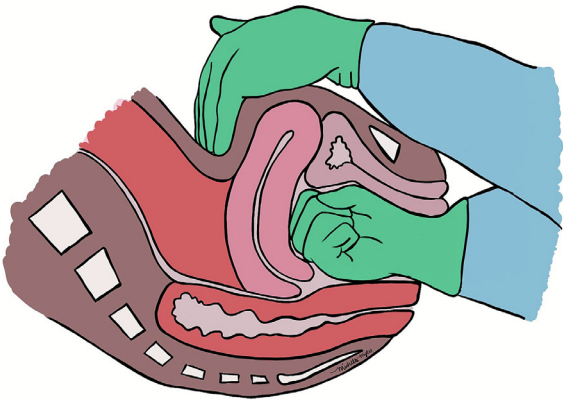


Fig. 2. Bimanual massage is a critical first intervention in the management of PPH. One hand is inserted into the vaginal canal pushing against the body of the uterus while the other hand is placed on the lower abdominal wall, attempting to compress the uterus between the two hands. Created by Michelle Myles, MD.

Oxytocin (Pitocin) is the first-line pharmacologic therapy and should be promptly administered at a dose of 10–40 units IV or intramuscular (IM) (Table 1). Administration should not be delayed by lack of IV access, as this medication can be safely administered via the IM route. If bleeding persists, clinicians should escalate to a second-line agent based on the patient's past medical history and contraindications: methylergonovine (avoid in hypertension), carboprost (avoid in asthma); and misoprostol (which is particularly useful when IV access is limited as it is administered rectally) [3].

Tranexamic acid (TXA) should be administered early in patients with PPH as a dose of 1 g IV over 10 min, ideally within 3 h of hemorrhage onset, as this has been associated with reduced mortality when given in conjunction with uterotonics [31,32]. If after 30 min bleeding persists, a second dose of 1 g IV over 8 h should be administered [11].

If medical therapy fails to achieve hemostasis, clinicians must escalate to mechanical tamponade. These therapies include the placement of a balloon tamponade device, which places direct pressure on the walls of the uterine cavity (Table 2) [19,33]. Additional advanced therapies to consider include uterine artery embolization by IR and surgical management by the obstetric specialist with uterine compression sutures, uterine artery ligation, or peri-partum hysterectomy [3,17]. Uterine packing is generally avoided due to risk of intrauterine infection but could be considered if advanced therapies such as IR or surgical intervention are not available [34,35].

3.5. What is the treatment for severe trauma (tears/lacerations)?

Trauma-related PPH should be suspected when bleeding persists despite a firm, well contracted uterus. In the ED, careful visual inspection during the secondary survey is critical to identify vaginal, cervical, perineal, or vulvar lacerations. If active bleeding is identified, immediate direct pressure should be applied as a temporizing measure until definitive management can be arranged. Most lacerations will require surgical repair performed by the obstetric specialist or surgical team, often in the operating room. If these resources are not available, targeted hemostatic sutures should be attempted. Alternatively, clinicians can consider vaginal packing as a temporizing intervention to achieve hemostasis and stabilization until operative intervention is available [3].

Delaying hemorrhage control while awaiting operative resources can result in further patient decompensation. If surgical repair is not immediately available, temporary measures such as targeted hemostatic sutures or vaginal packing may be used.

Table 1
Commonly Used Medications in PPH.

Medications	Dose	Contraindications
First Line:		
Oxytocin	10–40 IU/500–1000 mL of lactated ringer 5–10 IU IM up to 4 doses	None
Second Line:		
Methylergonovine maleate	0.2 mg every 2–4 h IM, max of 5 doses	Hypertension, cardiovascular disease
Carboprost tromethamine	250 μg every 15–90 min IM, max 8 doses	Asthma
Misoprostol	600–1000 μg PO or PR	Allergy to misoprostol
Tranexamic Acid	1 g IV over 10 min, can repeat dose after 30 min	Known hypersensitivity or allergy

3.6. What is the treatment for retained placental tissue?

Retained products of conception should be suspected in patients with ongoing bleeding despite a firm uterus or incomplete placental delivery. In the ED, manual uterine exploration may be performed when clinically appropriate, but ultrasound is often a useful adjunct to support the diagnosis and guide escalation of care. The presence of intrauterine echogenic material, with or without Doppler flow, should raise suspicion, though findings must be interpreted in clinical context [22].

Reliance on uterotonics alone may delay definitive management, as retained tissue frequently requires surgical evacuation. When retained products are suspected, early obstetric consultation and timely dilation and curettage should not be delayed while awaiting response to medical therapy.

3.7. What is the treatment for coagulopathy?

Early recognition of coagulopathy in the PPH patient is essential. The potential coagulopathies to evaluate for include underlying preeclampsia, HELLP syndrome, inborn errors of coagulation, antiplatelet or anticoagulant medications, and DIC. Treatment will be dictated based on the specific etiology [33].

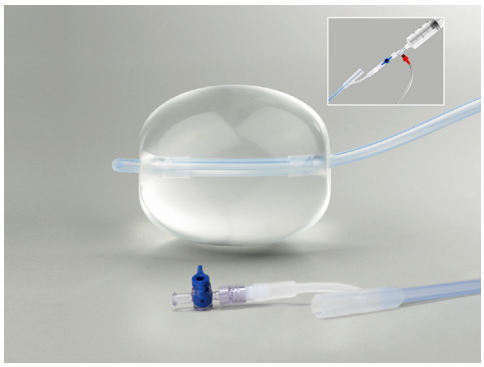
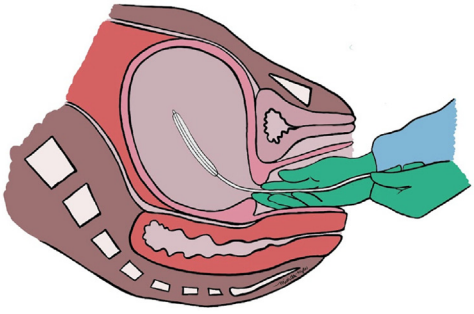
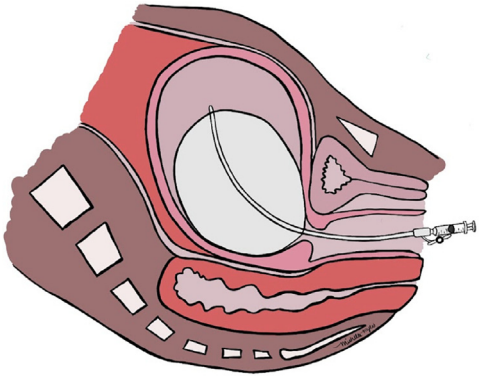
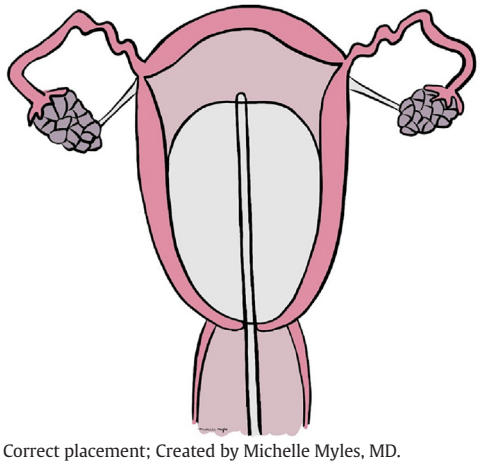
DIC should be suspected in the setting of ongoing bleeding with abnormal coagulation studies, thrombocytopenia, or hypofibrinogenemia. Diagnostic testing should include coagulation studies, including PT/INR, aPTT, platelet count, and fibrinogen. A fibrinogen level < 200 mg/dL is a strong predictor of progression to severe PPH. Most guidelines recommend targeting a fibrinogen level of >300–400 mg/dL. In patients with low fibrinogen levels, fibrinogen concentrate 4 g IV should be administered if available. If this is not available, cryoprecipitate should be administered [32,33].

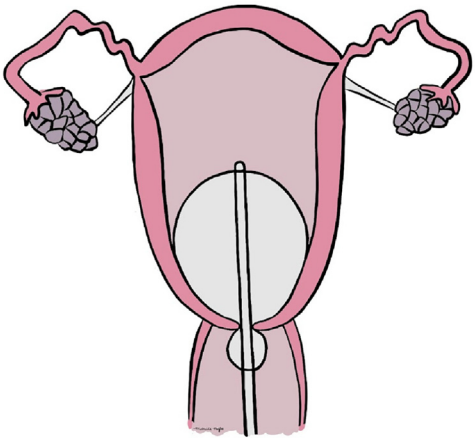
3.8. What is the treatment for uterine inversion and uterine rupture?

Uterine inversion is a rare but potentially fatal cause of PPH. Uterotonics should be discontinued immediately upon the diagnosis, and the clinician should promptly attempt manual replacement of the uterine fundus. The use of uterine relaxants such as nitroglycerin, terbutaline magnesium sulfate, or anesthesia to assist with relaxation of a constricted cervix may be helpful. To reduce the uterus, gentle manual pressure should be placed on the uterine fundus to return it to the abdominal cavity. If manual techniques are not effective, then surgical intervention is required [3,17,34,35]. Once the uterus is reduced, uterotonics should be restarted to maintain uterine tone and prevent reinversion. To avoid uterine inversion, gentle traction techniques should be used when attempting to remove an entrapped placenta, as forceful traction of the umbilical cord when resistance is met may lead to uterine inversion.

In contrast, uterine rupture is a surgical emergency that requires immediate recognition and operative management. Clinicians should maintain a high index of suspicion in patients with known risk factors, particularly those with a history of prior uterine surgery such as cesarean delivery or myomectomy. Although extremely rare, occurring in

Table 2
Insertion of uterine balloon tamponade device.

Bakri™ Postpartum Balloon (Cook Medical)	 https://cdnnamsseuspwsprod.azureedge.net/data/images/800x600/G24237_P_001-2.jpg Cook Medical
1. Insert deflated balloon portion into upper uterine segment through cervical os.	 Created by Michelle Myles, MD.
2. Instill sterile fluid into balloon using syringe to inflate the balloon to desired level of tamponade (maximum 500 mL).	 Created by Michelle Myles, MD.
3. While instilling fluid, use hand to palpate the end of balloon and confirm balloon is inflating inside the uterus and not protruding into cervical or vaginal canal.	 Correct placement; Created by Michelle Myles, MD.



Incorrect placement; Created by Michelle Myles, MD.

approximately 1:2500 to 1:5000 deliveries, the condition carries substantial risk of maternal and fetal morbidity and mortality [36,37].

The clinical presentation of uterine rupture can be subtle and is often difficult to recognize in the ED. The most sensitive early indicator is abnormal fetal heart rate patterns, including bradycardia, reduced variability, and recurrent decelerations. However, because patients in the ED are rarely on fetal monitoring, these findings may not be detected. Patients without regional anesthesia may report sudden severe or constant abdominal pain, sometimes described as a tearing or ripping sensation. Other clinical signs include hemodynamic instability, pain out of proportion to labor, loss of uterine tone, and fetal distress [36,37].

If a diagnosis of uterine rupture is suspected, aggressive resuscitation should be initiated while simultaneously arranging definitive operative management. Ongoing intraperitoneal bleeding may be from a concealed uterine rupture and should prompt emergent obstetric consultation and operative intervention. Management is typically surgical, for direct repair if the patient is hemodynamically stable with a desire to maintain fertility. In unstable patients, definitive management may require peripartum hysterectomy as a life-saving treatment. Uterine artery embolization may be an alternative option for stable patients desiring future fertility. Ultimately the management will depend on multiple factors, including stability of the patient, desired fertility, and available resources [3,17]. Table 3 provides a summary of pearls and pitfalls for PPH.

4. Conclusions

PPH is a leading cause of maternal morbidity and mortality and can be challenging to recognize in the ED due to unreliable visual estimation of blood loss and physiologic changes that mask early shock. Emergency

Table 3
Summary of PPH Pearls and Pitfalls.

Domain	Pearls	Pitfalls
Recognition and Definition	Treat PPH based on physiology, not volume alone; intervene for any bleeding with signs of hypovolemia. Delayed PPH can occur up to 12 weeks postpartum.	Waiting to meet numerical blood loss thresholds or dismissing delayed PPH.
Definition and Timing	PPH includes >1000 mL blood loss or any bleeding with signs of hypovolemia. Primary PPH occurs within 24 h; secondary PPH can be up to 12 weeks postpartum	Waiting to reach numeric blood loss thresholds or dismissing delayed PPH as benign.
Initial Evaluation	Prioritize vital sign trends, mental status, and bleeding severity. Perform pelvic examination in all suspected cases.	Relying on visual blood loss estimation or imaging alone without pelvic examination.
Physical Examination	Pelvic examination and bimanual examination to assess uterine tone and size. Visual inspection for lacerations/ hematomas or inversions.	Relying on imaging alone without performing a pelvic examination.
Etiology (4Ts)	Evaluate Tone, Tissue, Trauma, Thrombin in parallel; presume and treat uterine atony first.	Fixation on a single cause while missing concurrent etiologies.
Laboratory testing and Adjuncts	Obtain CBC, coagulation studies, fibrinogen, and type and screen. Fibrinogen <200 mg/dL signals severe hemorrhage.	Delay treatment while awaiting laboratory results in unstable patients.
Early Management	Activate OB, anesthesia, blood bank, IR, and OR early; management should be parallel, not sequential.	Delayed consultation or prolonged bedside management before escalation.
Transfusion and MTP	Maintain a low threshold for blood products; initiate balanced transfusion (1:1:1) when indicated; use uncrossmatched blood if needed.	Waiting for fixed transfusion volumes or strict criteria before activating MTP.
Uterine Atony	Immediate bimanual uterine massage and oxytocin; escalate quickly to second-line uterotonics and TXA.	Delaying escalation beyond oxytocin despite ongoing hemorrhage.
Mechanical and Definitive Therapy	Balloon tamponade is first-line mechanical therapy; escalate to IR embolization or surgery based on stability and resources.	Prolonged medical management when mechanical or surgical intervention is required.
Trauma (Lacerations)	Identify and apply direct pressure promptly; most lacerations require operative repair.	Failure to perform careful visual inspection or delaying surgical management.
Retained Products	Manual uterine exploration is first-line; ultrasound can assist; definitive management is surgical (D&C).	Relying on uterotonics alone when retained tissue is present.
Coagulopathy/ DIC	Suspect DIC with abnormal coagulation studies or hypofibrinogenemia; target fibrinogen >300–400 mg/dL with fibrinogen concentrate or cryoprecipitate.	Under-recognizing evolving coagulopathy or delaying correction of low fibrinogen.
Uterine Inversion	Stop uterotonics and attempt immediate manual replacement; use uterine relaxants if needed; restart uterotonics after reduction.	Risk factors include continue traction on the umbilical cord or delayed recognition.
Uterine Rupture	Maintain high suspicion in patients with prior uterine surgery; initiate aggressive resuscitation and emergent operative management.	Miss rupture due to subtle presentation or lack of fetal monitoring in the ED.

physicians must maintain a high index of suspicion and rapidly initiate resuscitation while identifying the underlying cause using the "4 Ts": Tone, Tissue, Trauma, and Thrombin. Management requires prompt physical examination, early transfusion of blood products, and targeted therapy, including uterotonics such as oxytocin, methylergonovine, carboprost, and misoprostol, with adjunctive use of TXA when indicated. Definitive management may require laceration repair, removal of retained products, balloon tamponade, and IR or operative intervention. Optimal outcomes depend on early recognition and a coordinated multidisciplinary collaboration between emergency medicine, obstetrics, IR, anesthesia, and critical care.

CRediT authorship contribution statement

Gianna Petrone: Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Resources, Conceptualization. **Amy Mariorenzi:** Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Resources, Conceptualization. **Alex Koyfman:** Writing – review & editing, Visualization, Validation, Supervision, Resources, Conceptualization. **Brit Long:** Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Resources, Conceptualization.

Declaration of competing interest

None.
No AI program was used in the construction of this manuscript.

Acknowledgements

GP, AM, AK, and BL conceived the idea for this manuscript and contributed substantially to the writing and editing of the review. This manuscript did not utilize any grants, and it has not been presented in abstract form. This clinical review has not been published, it is not under consideration for publication elsewhere, its publication is approved by all authors and tacitly or explicitly by the responsible authorities where the work was carried out, and that, if accepted, it will not be published elsewhere in the same form, in English or in any other language, including electronically without the written consent of the copyright-holder.

References

[1] Say L, Chou D, Gemmill A, Tunçalp Ö, Moller A-B, Daniels J, et al. Global causes of maternal death: a WHO systematic analysis. Lancet Glob Health. 2014;2(6):e323–33.
[2] Creanga AA, Syverson C, Seed K, Callaghan WM. Pregnancy-related mortality in the United States, 2011 to 2013. Obstet Anesth Digest. 2018;38(1):16.
[3] American College of Obstetricians and Gynecologists. Practice bulletin: postpartum hemorrhage. Clinical management guidelines for obstetrician-gynecologists. Obstet Gynecol. 2017;130:e168–86.
[4] Dobiesz VA, Robinson DW. In: Walls RM, Hockberger RS, Gausche-Hill M, editors. Rosen's emergency medicine: Concepts and clinical practice. Philadelphia: Elsevier; 2017.
[5] Lockwood C, Moore T, Copel J. Creasy and Resnik's maternal-fetal medicine: Principles and practice. Philadelphia: Elsevier; 2020.
[6] Pritchard JA. Changes in the blood volume during pregnancy and delivery. Anesthesiology. 1965;26(4):393–9.
[7] Pouta AM, Räsänen JP, Airaksinen KE, Vuolteenaho OJ, Laatikainen TJ. Changes in maternal heart dimensions and plasma atrial natriuretic peptide levels in the early puerperium of normal and pre-eclamptic pregnancies. Br J Obstet Gynaecol. 1996;103(10):988–92.
[8] Arulkumaran S. A comprehensive textbook of postpartum Haemorrhage: An essential clinical reference for effective management. London: Sapiens Publishing; 2012.
[9] Bateman BT, Berman MF, Riley LE, Leffert LR. The epidemiology of postpartum hemorrhage in a large, nationwide sample of deliveries. Anesth Analg. 2010;110(5):1368–73.
[10] Gyamfi-Bannerman C, Srinivas SK, Wright JD, Goffman D, Siddiq Z, D'Alton ME, et al. Postpartum hemorrhage outcomes and race. Obstet Anesth Digest. 2019;39(2):61–2.
[11] Bienstock JL, Ahizechukwu C, Hueppchen N. Postpartum hemorrhage. N Engl J Med. 2021;384(17):1635–45.
[12] Corbetta-Rastelli C, Friedman A, Sobhani N, Arditi B, Goffman G, Wen T. Postpartum hemorrhage trends and outcomes in the United States, 2000–2019. Obstet Gynecol. 2023;141(1):152–61.
[13] Wang E, Glazer KB, Howell EA, Janevic TM. Social determinants of pregnancy-related mortality and morbidity in the United States: a systematic review. Obstet Anesth Digest. 2021;41(1):12.
[14] Schorn MN. Measurement of blood loss: review of the literature. J Midwifery Womens Health. 2009;55(1):20–7.
[15] Tan EK, Tan EL. Alterations in physiology and anatomy during pregnancy. Best Pract Res Clin Obstet Gynaecol. 2013;27(6):791–802.
[16] Mhyre JM, D'Oria R, Hameed A, Lappen JR, Holley SL, Hunter SK, et al. The maternal early warning criteria: a proposal from the National Partnership for maternal safety. J Obstet Gynecol Neonatal Nurs. 2014;43(6):771–9.
[17] Royal College of Obstetricians and Gynaecologists. Prevention and management of postpartum haemorrhage: Green-Top Guideline No. 52. BJOG. 2017;124(5):e106.
[18] Green LJ, Mackillop LH, Salvi D, et al. Gestation-specific vital sign reference ranges in pregnancy. Obstet Gynecol. 2020;135(3):653–64. doi:10.1097/AOG.0000000000003721.
[19] American College of Obstetricians and Gynecologists. Clinical practice update: use of nonsurgical hemorrhage-control devices for postpartum hemorrhage management. Obstet Gynecol. 2025;146(4):569–71.
[20] Turkoglu O, Friedman P. Evaluation during postpartum hemorrhage. Clin Obstet Gynecol. 2023;66(2):357–66.
[21] American College of Obstetricians and Gynecologists. Quantitative Blood Loss in Obstetric Hemorrhage: Committee Opinion No. 794. Obstet Gynecol. 2019.;134(6)e150–56.
[22] Mappa I, Patrizi L, Maruotti GM, Carbone L, D'Antonio F, Rizzo G. The role of ultrasound in the diagnosis and management of postpartum hemorrhage. J Clin Ultrasound. 2023;51(2):362.
[23] Kroh E, Waters JH. Blood product replacement in postpartum hemorrhage. Clin Obstet Gynecol. 2023;66(2):357–66.
[24] Roberts JR, Custalow CB, Thomsen TW, Hedges JR. Roberts and Hedges' clinical procedures in emergency medicine and acute care. 7th ed., Philadelphia, PA: Elsevier; 2019.
[25] Post S, Rood K, Kiefer M. Interventions of postpartum hemorrhage. Clin Obstet Gynecol. 2023;66(2):367–83.
[26] Al Kadri HM, Al Anazi BK, Tamim HM. Visual estimation versus gravimetric measurement of postpartum blood loss: a prospective cohort study. Arch Gynecol Obstet. 2011;283(6):1207–13.
[27] Patel A, Goudar SS, Geller SE, Kodkany BS, Edlavitch SA, Wagh K, et al. Drape estimation vs visual assessment for estimating postpartum hemorrhage. Int J Gynaecol Obstet. 2006;93(3):220–4.
[28] Patil V, Shetmahajan M. Massive transfusion and massive transfusion protocol. Indian J Anaesth. 2014;58(5):590–5.
[29] Shields LE, Wiesner S, Fulton J, Pelletreau B. Comprehensive maternal hemorrhage protocols reduce the use of blood products and improve patient safety. Am J Obstet Gynecol. 2015;212(3):272–80.
[30] American College of Surgeons trauma quality improvement program. Massive transfusion in trauma guidelines [internet]. Chicago: American College of Surgeons; 2025. Available from: https://web4.facs.org/tqipfiles/Massive%20Transfusion%20in%20Trauma%20Guildelines.pdf.
[31] Escobar MF, Sandoval E, Tobón YA, Riaño-Medina CE. Postpartum hemorrhage in the emergency department. Int J Emerg Med. 2022;15(1):1–6.
[32] Wang M, Oyelese Y. Postpartum hemorrhage. Matern Fetal Med. 2025;7(1):38–48.
[33] Overton E, D'Alton ME, Goffman D. Intrauterine devices in the management of postpartum hemorrhage. Am J Obstet Gynecol. 2024;230(3 Suppl):S1076–88.
[34] Shakur H, Roberts I, Fawole B, Chaudhri R, El-Sayed H, Qureshi Z, et al. Hysterectomy and other morbidities in women with postpartum hemorrhage (WOMAN): an international, randomized, double-blind, placebo-controlled trial. Lancet. 2017;389(10084):2105–16.
[35] Kroh S, Waters JH. Obstetrical hemorrhage. Anesthesiol Clin. 2021;39(4):597–611.
[36] Togioka BM, Tonismae T. Uterine rupture. StatPearls [internet]. Treasure Island. FL: StatPearls Publishing; 2025 PMID: 32644635.
[37] Abdulmane MM, Sheikhali OM, Alhowaidi RM, Qazi A, Ghazi K. Diagnosis and management of uterine rupture in the third trimester of pregnancy: a case series and literature review. Cureus. 2023;15(6):e39861.