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Contemporary Approach to Postpartum Hemorrhage: Literature Review

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ABSTRACT

Obstetric haemorrhage—particularly primary postpartum haemorrhage—remains the leading preventable cause of maternal death worldwide. Despite pharmacological and surgical advances, its incidence remains high in low- and middle-income countries, where limited access to high-quality uterotonics, safe blood products, and multidisciplinary teams increases lethality. This article critically reviews the epidemiology, pathophysiology, and contemporary evidence-based diagnostic and treatment strategies published between 2000 and 2025. The Shock Index (≥ 0.9) is highlighted as an early marker of haemodynamic instability, and the importance of objective blood-loss quantification for activating rapid-response protocols is emphasised. Stepwise management integrates early haemostatic resuscitation, sequential and rational use of uterotonics, administration of tranexamic acid within the first three hours, intra-uterine tamponade, conservative surgical techniques, and—where available—selective arterial embolisation. A prevention bundle centred on active management of the third stage of labour with oxytocin or heat-stable carbetocin reduces postpartum haemorrhage incidence by more than 40 %. Disparities between high- and low-income countries demand systemic interventions that ensure supplies, train personnel, and optimise referral networks. Implementation of standardised obstetric bundles, together with wider availability of haemostatic technologies and universal access to heat-stable uterotonics, constitutes the most effective strategy for achieving the Sustainable Development Goal 2030 targets for maternal-mortality reduction.

Keywords
Postpartum Hemorrhage, Uterine Atony, Oxytocin, Tranexamic Acid, Hemorrhagic Shock.

Introduction

Obstetric hemorrhage—specifically postpartum hemorrhage (PPH)—remains the most feared complication of perinatal care. It is estimated that approximately 830 women die every day from causes related to pregnancy and childbirth, and about one-third of these deaths are due to obstetric bleeding, despite the fact that most could be prevented through timely medical interventions [1-4]. PPH affects more than fourteen million postpartum women

annually and causes nearly seventy thousand deaths, 88% of which occur acutely within the first four hours after delivery, often before the patient can be referred to a higher-level care facility [1,3,5]. These figures make PPH the leading direct cause of maternal mortality worldwide, surpassing hypertensive disorders of pregnancy and infectious etiologies [2,3,6]. Its clinical significance extends beyond mortality: in addition to acute blood loss leading to severe anemia, respiratory distress syndrome, and multiorgan dysfunction, PPH is associated with long-term psychosocial sequelae [4,6,7].

Over the past decade, organizations such as the World Health Organization (WHO) and the International Federation of Gynecology and Obstetrics (FIGO) have emphasized that reducing PPH is essential to achieving Sustainable Development Goal 3.1, which aims for a global maternal mortality ratio of fewer than 70 deaths per 100,000 live births by 2030 [3,5,8]. Early recognition and implementation of multidisciplinary care bundles have been shown to reduce mortality by up to 60% in high-performance centers [9,10]. Nevertheless, the gap between high- and low-income countries remains wide, as limited-resource regions often face shortages of heat-stable uterotonics, inconsistent blood banking systems, and delayed referral networks that perpetuate poor outcomes [4,8,11].

In this context, the present article reviews the most recent scientific evidence on the epidemiology, pathophysiology, diagnosis, and management of PPH, with the aim of providing robust and standardized recommendations for both frontline clinicians and researchers dedicated to this topic [1,2,5,6,12].

Epidemiology

The incidence of postpartum hemorrhage (PPH)—defined as blood loss exceeding 500 mL after vaginal delivery or 1000 mL after cesarean section—varies significantly depending on the availability of qualified obstetric services [3,4,6]. In Western Europe and North America, the frequency of blood loss greater than 1000 mL is estimated at 1–2 % of all deliveries; in sub-Saharan Africa and Southeast Asia, it ranges from 3–6 %, with peaks of up to 10 % in rural areas lacking access to high-quality uterotonics. Underreporting likely underestimates these figures [3,5,8,9].

The maternal mortality ratio due to hemorrhage remains close to 10 per 100,000 live births in high-income countries but exceeds 446 per 100,000 in low-income nations, reflecting striking regional inequities [3,5,8,11]. The WHO 2023–2030 Roadmap reports that obstetric bleeding accounted for 27 % of the 295,000 estimated maternal deaths in 2017 and warns that, without large-scale interventions, this proportion is expected to remain stable over the next decade [5]. Regionally, South America has reduced mortality due to PPH to 47 per 100,000 live births; however, pockets of high mortality persist in the Amazon basin and the Andean highlands—a phenomenon attributed to the "three delays" model: delays in recognition, decision-making, and transfer to referral-level care [1,3,8,10]. (Table 1).

Table 1: Epidemiology of Postpartum Hemorrhage.

Indicator	Low- and Middle-Income Countries	High-Income Countries
Incidence of PPH ≥ 500 mL	10–25 % of deliveries	5–8 % of deliveries
Incidence of PPH ≥ 1000 mL	3–6 %	1–2 %
Maternal mortality (2023)	446 / 100 000 live births in sub-Saharan Africa	10 / 100 000 live births in Western Europe
Proportion of maternal deaths due to PPH	≈ 30 %	< 10 %

Definition

The World Health Organization (WHO) and the International Federation of Gynecology and Obstetrics (FIGO) maintain the classical definition of primary postpartum hemorrhage (PPH) as blood loss of ≥500 mL after vaginal delivery or ≥1000 mL after cesarean section within the first 24 hours following birth [1-3,13].

The American College of Obstetricians and Gynecologists (ACOG) expands this concept to include any bleeding that results in hemodynamic instability, regardless of the estimated volume, since visual assessment can underestimate actual blood loss by up to 50% [9,14].

Massive hemorrhage is defined as blood loss exceeding 1500 mL or requiring transfusion of ≥4 units of blood products, while secondary postpartum hemorrhage refers to bleeding occurring between 24 hours and 6 weeks postpartum [4,6,14]. The broader term obstetric hemorrhage also encompasses antepartum bleeding (placenta previa, placental abruption, and vasa previa), which poses a life-threatening risk to both mother and fetus [3,5].

Pathophysiology

The pathophysiology of obstetric hemorrhage is classically summarized by the "4 Ts": tone (uterine atony), trauma, tissue, and thrombin [1,2,15]. (Figure 1)

At term, uteroplacental blood flow reaches 500–700 mL/min; following placental expulsion, myometrial contraction acts as a "physiologic suture" that compresses the venous sinusoids [3,4]. When this contraction fails, uterine arteries can bleed at rates of 300–500 mL/min, leading to hypovolemic shock within less than 10 minutes [15,16]. (Table 2)

In these cases, acute hyperfibrinolysis triggered by tissue activators and a fibrinogen level < 2 g/L double the risk of death [17,18]. Additionally, the placenta accreta spectrum adds a severe mechanical component by preventing spontaneous detachment from the placental bed and increasing flow through large-caliber vessels [6,19]. Finally, the combination of acidosis, hypothermia, and dilutional coagulopathy during resuscitation constitutes the so-called "lethal triad." [20].

Diagnostic Criteria

Early diagnosis depends on objective quantification of blood loss and continuous clinical assessment [3-5]. The use of

calibrated collection bags or pad weighing reduces the inherent underestimation of visual assessment, particularly in low-resource settings [8,21]. Among physiological parameters, the Shock Index (heart rate/systolic blood pressure) is the most sensitive marker: values ≥0.9 predict massive transfusion and severe adverse outcomes [22,23]. This index has been validated as the 95th percentile reference during the first 48 hours postpartum [23].

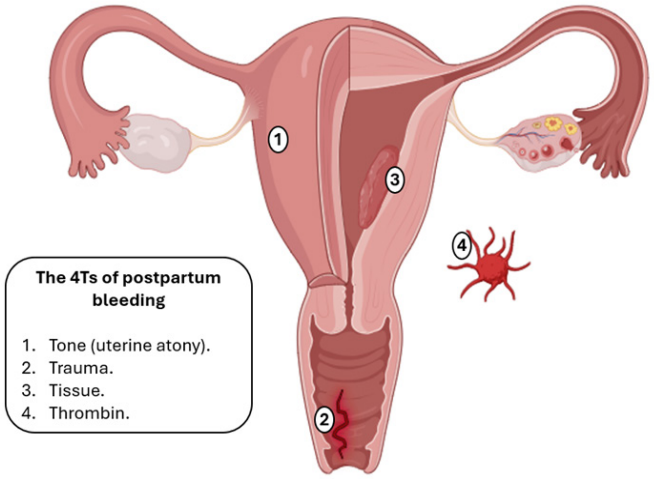

Figure 1: The "4 Ts" of obstetric hemorrhage. Source: Created with BioRender resources.

Table 2: The "4 Ts" of Obstetric Hemorrhage.

Mechanism	Physiological Detail
Uterine atony (≈ 80 % of PPH cases)	Uterine blood flow at term: 500–700 mL/min. In the absence of contraction, uterine arteries may bleed 300–500 mL/min.
Genital tract trauma	Cervical or soft birth canal lacerations.
Tissue retention	Interferes with uterine contraction and triggers release of tissue thromboplastin.
"Thrombin" – coagulation disorders	Early disseminated intravascular coagulation (DIC) due to activation of protein C and fibrinolysis.

In laboratory testing, fibrinogen <2 g/L and lactate >4 mmol/L are associated with poor outcomes, while viscoelastic assays (ROTEM/TEG) allow for targeted transfusion therapy [17,24]. According to FIGO 2022 and WHO 2023 recommendations, the "PPH code" should be activated in the presence of active bleeding with a Shock Index ≥0.9, progressive hypotension, or quantified blood loss exceeding 1000 mL [1,3,5,8].

Etiologic Classification

From a temporal perspective, obstetric hemorrhage is classified as antepartum, intrapartum, immediate postpartum, and late postpartum [1,3,5,6]. Antepartum hemorrhage includes placenta previa, placental abruption (abruptio placentae), and uterine rupture, which typically present with painless bright-red vaginal bleeding or severe abdominal pain with dark blood, depending on the underlying cause [6,9,19].

During labor, bleeding from lacerated cervical or vaginal vessels and extensive episiotomies contributes to the traumatic component [14,15,19]. In the first 24 hours postpartum, uterine atony predominates, accounting for up to 80% of cases, followed by retained placental tissue and the placenta accreta spectrum [3,6,19,20]. Between 24 hours and six weeks postpartum, the most frequent causes are endometritis, delayed placental bed involution, uterine artery pseudoaneurysms, and hereditary or acquired bleeding disorders [6,19]. (Table 3).

Table 3: Etiologic Classification According to the Timing of Bleeding.

Timing	Main Causes
Antepartum	Placenta previa, placental abruption (abruptio placentae), vasa previa, uterine rupture.
Immediate postpartum (< 24 h)	Uterine atony, genital tract lacerations, retained placental tissue, placenta accreta spectrum.
Late postpartum (24 h–6 weeks)	Endometritis, abnormal placental bed involution, uterine artery pseudoaneurysm.
Secondary to coagulopathy	Disseminated intravascular coagulation (DIC), acquired hemophilia.

Risk Factors

Risk factors are categorized as modifiable, non-modifiable, and iatrogenic [1,3,5,6]. Among modifiable factors, the most relevant are pre-gestational anemia (hemoglobin <11 g/dL), maternal obesity (BMI > 30 kg/m²), and inadequate antenatal care, all of which are associated with a higher likelihood of hemorrhage and adverse outcomes [4,5,8,14]. Non-modifiable factors include the placenta accreta spectrum, fetal macrosomia (>4500 g), multiple gestation, and grand multiparity (≥5 births), each increasing the risk of uterine atony and transfusion requirement [6,9,19,31]. Iatrogenic factors encompass prolonged induction or stimulation with oxytocin, instrumental delivery, and repeated cesarean sections, all of which heighten the probability of abnormal placental implantation [3,6,14,17]. Nevertheless, up to 60 % of women with PPH present no identifiable predisposing factors, underscoring the need for universal vigilance and readily available obstetric resources for every delivery [5,9,10,25].

Clinical Manifestations

Clinical manifestations depend on the volume of blood loss and the cardiovascular compensatory capacity [1,3,5]. In the early phase, the predominant signs include sinus tachycardia (>100 bpm), diaphoresis, pallor, and a sense of anxiety. These symptoms often appear before hypotension develops, owing to the expanded plasma volume of pregnancy [6,7,22].

As bleeding progresses, patients may develop systolic blood pressure <90 mmHg, oliguria (<30 mL/h), and distal coldness [3,4,22]. Altered mental status, ranging from restlessness to confusion, reflects cerebral hypoperfusion and correlates with elevated serum lactate levels [17,24].

Each etiology exhibits characteristic findings:

- A boggy, enlarged uterus suggests uterine atony.
- Painless bright-red bleeding points to placenta previa.
- Dark bleeding with uterine hypertonia and pain indicates

placental abruption.

- Passage of large clots after placental expulsion suggests retained products of conception.
- Deep cervical or perineal lacerations may cause persistent bleeding despite a firm, well-contracted uterus [6,15,19,25]. (Table 4).

Early recognition of these warning signs, combined with objective quantification of blood loss and a Shock Index ≥ 0.9, allows for prompt activation of rapid-response protocols [22,23,25].

Table 4: Clinical Manifestations of Postpartum Hemorrhage.

Phase	Key Signs	Etiologic Example
Early	Tachycardia, cold skin, anxiety.	Concealed bleeding due to retroplacental hematoma.
Late	Systolic blood pressure < 90 mmHg, oliguria < 30 mL/h, altered mental status.	Refractory uterine atony.
Specific findings	• Globular, flaccid uterus → uterine atony. • Painless bright-red bleeding → placenta previa. • Painful dark bleeding → placental abruption.	—

Treatment

The contemporary management of postpartum hemorrhage (PPH) follows a stepwise escalation strategy [1-3,9,17,25]. The first tier includes securing the airway, providing supplemental oxygen, establishing two large-bore (14–16 G) intravenous lines, and initiating resuscitation with warmed crystalloids while laboratory tests are obtained. If blood loss exceeds 1000 mL or hemodynamic instability develops, the massive transfusion protocol (1:1:1)—packed red blood cells, plasma, and platelets—should be activated and guided by viscoelastic testing (ROTEM/TEG) [24,25]. Tranexamic acid, administered as 1 g IV within 3 hours of bleeding onset, reduced hemorrhage-related mortality by 30% in the WOMAN trial [11,26], and its early use is now a Grade 1A recommendation by FIGO (2022) and WHO (2023) [1,3,5]. The etiologic treatment of uterine atony begins with oxytocin 10 IU IV/IM plus continuous infusion, followed—depending on availability—by methylergometrine 0.2 mg IM, thermostable carbetocin 100 µg IV, or misoprostol 800–1000 µg rectally [12,17,27].

The CHAMPION trial demonstrated non-inferior efficacy of thermostable carbetocin compared with oxytocin, with the added advantage of not requiring cold-chain storage [12]. If bleeding persists, an intrauterine balloon tamponade (Bakri or condom-type balloon) should be applied, achieving success rates of 80–90% [20,28,29]. If this measure fails, compression sutures (B-Lynch, Cho, Hayman) or selective arterial ligation may be employed; in equipped centers, radiologic embolization is preferred [19,30].

Early obstetric hysterectomy is reserved for uncontrolled hemorrhage or placenta accreta spectrum disorders [6,19,31]. Throughout the process, hypothermia and acidosis must be prevented, coagulopathy corrected, and adequate tissue perfusion maintained [20,25]. (Table 5).

Table 5: Therapeutic Approach to Postpartum Hemorrhage According to the Proposed Scenario.

Cause	First-Line Management	Second-Line Management	Third-Line Management
Uterine atony	Oxytocin 10 IU IV/IM + continuous infusion; uterine massage	Methylergometrine 0.2 mg IM, Carbetocin 100 µg IV, Misoprostol 800–1000 µg rectally	Intrauterine balloon tamponade (Bakri), B-Lynch suture, uterine/hypogastric artery ligation, hysterectomy
Genital tract trauma	Targeted surgical repair	Selective arterial embolization	—
Retained tissue	Manual removal or curettage	Intraumbilical oxytocin	—
Coagulopathy	Fibrinogen concentrate or cryoprecipitate to maintain > 2 g/L; PCC if INR > 1.5	Recombinant activated factor VII (rFVIIa) 90 µg/kg in refractory shock	—

Prevention

Prevention is grounded in the active management of the third stage of labor. Administration of oxytocin 10 IU IM/IV immediately after fetal delivery reduces the incidence of postpartum hemorrhage (PPH) by approximately 40% [1,3,5,17]. When cold-chain storage is unreliable, thermostable carbetocin or misoprostol 600 µg orally are safe and effective alternatives [12,17,27].

The FIGO 2022, WHO 2018/2023, and AIM-IHI 2024 guidelines recommend combining this practice with team simulation training, active monitoring, and universal availability of uterotonic agents [1,5,9,10,25]. Implementation of obstetric safety bundles has been shown to reduce PPH-related mortality by up to 50% through the integration of prevention, rapid response, and post-event evaluation components [9,25,32].

Discussion

Current consensus indicates that postpartum hemorrhage (PPH) is preventable when the three delays—recognition, decision-making, and transfer—are effectively addressed [3,5,8,9]. Integrated obstetric response bundles are more effective than isolated interventions, reducing mortality by up to 60% [9,10,25,32]. The Shock Index ≥0.9 has emerged as a universal, inexpensive, and validated tool for early detection of hemodynamic instability [22,23]. The availability of tranexamic acid and thermostable uterotonics has transformed PPH management in settings lacking blood banks or refrigeration capacity [1,3,5,12,26].

However, several areas of uncertainty remain: the optimal tranexamic acid dosing in obesity, the accuracy of portable

hemoglobin devices, and the ideal surgical timing in placenta accreta spectrum disorders [19,31]. The expansion of viscoelastic testing in obstetric units and the implementation of interventional radiology in secondary-level hospitals require sustained investment and training [24,25,30,31]. Future research should focus on community-level strategies to shorten referral times and on artificial intelligence–based systems capable of detecting subtle hemodynamic variations to enable earlier clinical intervention [32].

Conclusions

Obstetric hemorrhage remains the leading perinatal emergency and the primary direct cause of maternal death worldwide. Evidence supports a stepwise approach that integrates close clinical surveillance, use of the Shock Index, early administration of uterotonics and tranexamic acid, viscoelasticity-guided hemostatic resuscitation, and scalable surgical or radiologic techniques. Ensuring access to high-quality uterotonics, strengthening blood banks, training multidisciplinary teams, and implementing obstetric safety bundles are essential actions for countries to achieve, by 2030, the maternal mortality targets set by the World Health Organization. Bridging the gap between high- and low-income regions requires global and local strategies that combine sustainable funding, health system strengthening, and women's empowerment, so that no mother continues to die from preventable postpartum bleeding.

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