

Preeclampsia Severity and the Risk of Postpartum Hemorrhage: A Systematic Review

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Abstract

Postpartum hemorrhage (PPH) remains a leading cause of maternal mortality worldwide, and preeclampsia has long been recognized as a potential contributor to this risk, although evidence on how the risk varies with disease severity remains inconsistent. This review aimed to synthesize current evidence on the association between preeclampsia severity and PPH risk, and to examine whether the magnitude of risk differs between non-severe and severe disease. A systematic literature search was conducted in PubMed, ScienceDirect, and Google Scholar for observational studies, systematic reviews, and meta-analyses reporting this relationship; studies were screened for relevance, methodological quality, and reported effect estimates, and twelve studies meeting the inclusion criteria were retained for qualitative synthesis. Preeclampsia was consistently associated with a higher risk of PPH, with crude and adjusted odds ratios ranging from about 1.3 to 3.1; several large cohorts unexpectedly reported a higher adjusted risk in non-severe than in severe preeclampsia, a pattern linked to differences in labor induction, oxytocin augmentation, and timing of delivery, while uterine atony remained the dominant mechanism, compounded in severe disease by thrombocytopenia, endothelial dysfunction, and, in some cases, coagulopathy. Preeclampsia, regardless of its severity classification, is thus an important and largely modifiable contributor to PPH risk, warranting active management of the third stage of labor and severity-tailored anticipatory planning to reduce PPH-related morbidity in this population.

Key words: preeclampsia; postpartum hemorrhage; severity; uterine atony; systematic review

INTRODUCTION

Postpartum hemorrhage (PPH) remains one of the leading direct causes of maternal death worldwide, accounting for a substantial share of the global burden of maternal mortality, particularly in low- and middle-income settings (Say et al., 2014; Demissie et al., 2025). Uterine atony, defined as the failure of the myometrium to contract adequately after delivery, is responsible for most cases, with genital tract trauma, retained placental tissue, and coagulopathy accounting for the remainder (Yunas et al., 2025; Pettersen et al., 2025).

Preeclampsia, a multisystem hypertensive disorder of pregnancy characterized by new-onset hypertension after 20 weeks of gestation accompanied by proteinuria or evidence of end-organ dysfunction, has long been recognized as a contributor to adverse maternal outcomes, including PPH (Cunningham et al., 2022). The disorder is conventionally stratified into preeclampsia without severe features and preeclampsia with severe features, the latter defined by markers such as severe hypertension, thrombocytopenia, impaired liver function, renal insufficiency, pulmonary edema, or new-onset cerebral or visual disturbances (American College of Obstetricians and Gynecologists [ACOG], 2020; Cunningham et al., 2022).

Biologically, several mechanisms could link preeclampsia to PPH: widespread endothelial dysfunction and vasospasm may impair uterine vascular tone, magnesium sulfate used for seizure prophylaxis can blunt myometrial contractility, and, in more severe presentations, thrombocytopenia or overt disseminated intravascular coagulation may directly impair hemostasis (Cunningham et al., 2022; von Schmidt auf Altenstadt et al., 2013). Yet, the epidemiological evidence on how this risk scales with disease severity has been inconsistent.

Several large cohorts report the expected pattern of increasing risk with increasing severity, while others paradoxically report a higher risk of PPH among women with non-severe preeclampsia than among those with severe disease, a discrepancy that has not been fully reconciled in the clinical literature (von Schmidt auf Altenstadt et al., 2013).

Given these conflicting signals, and the practical importance of anticipating PPH in women with hypertensive disorders of pregnancy, this review was undertaken to systematically synthesize the available evidence on the association between preeclampsia severity and PPH risk, and to explore possible explanations for the observed heterogeneity across studies.

METHOD

This review followed a systematic approach informed by the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) framework. Eligible studies were observational studies (cohort or case-control), and systematic reviews or meta-analyses, published in English, that reported an association between preeclampsia (with or without severe features) and PPH, expressed as an odds ratio, adjusted odds ratio, relative risk, or descriptive frequency. Studies addressing PPH exclusively from causes unrelated to hypertensive disease, case reports, and non-peer-reviewed commentaries were excluded.

A literature search was performed across PubMed, ScienceDirect, and Google Scholar using combinations of the terms ("preeclampsia" OR "pre-eclampsia") AND ("postpartum hemorrhage" OR "postpartum haemorrhage") AND ("severity" OR "severe features" OR "risk factor" OR "uterine atony"), without restriction on publication year up to 2026. Titles and abstracts were screened for relevance, followed by full-text assessment of potentially eligible articles. Reference lists of included articles were hand-searched to identify additional relevant studies.

For each included study, the following data were extracted: first author and year, country, study design, sample size, definition of PPH used, definition and classification of preeclampsia severity, and the reported effect estimate with its 95% confidence interval. Methodological quality was judged qualitatively based on study design, sample size, and adequacy of confounder adjustment, drawing on quality domains comparable to the Newcastle-Ottawa Scale as applied in prior reviews on this topic (Gallardo Cerna et al., 2025; Li et al., 2021). Because of substantial heterogeneity in PPH definitions (blood loss thresholds of 500 mL versus 1000 mL, with or without a hemodynamic-compromise criterion) and in preeclampsia diagnostic criteria across different guideline eras, a narrative (qualitative) synthesis was performed rather than a pooled quantitative meta-analysis. Twelve studies met the inclusion criteria and were included in the final synthesis.

RESULT & DISCUSSION

Result

Twelve studies published between 2013 and 2025 met the inclusion criteria, comprising nationwide cohort studies, multicenter retrospective studies, hospital-based case series, and systematic reviews/meta-analyses conducted across Europe, the Americas, Asia, and Central Asia. The characteristics of the included studies are summarized in Table 1.

Table 1. Characteristics of studies included in the review

Author (Year)	Country/Region	Design	Sample size	Key finding on preeclampsia and PPH
von Schmidt auf Altenstadt et al. (2013)	Netherlands	Nationwide cohort	1,457,576	PPH risk higher with preeclampsia overall (aOR 1.53); non-severe preeclampsia carried a higher adjusted risk (aOR 1.67) than severe preeclampsia (aOR 1.32).
Gallardo Cerna et al. (2025)	Multinational (meta-analysis)	Systematic review & meta-analysis	2,102,987 pregnancies	Pooled OR 1.95 (95% CI 1.70–2.23); highest regional risk in Europe (OR 2.18), followed by the Americas (OR 1.95) and Asia (OR 1.67).
Yunas et al. (2025)	Global (327 studies)	Systematic review & meta-analysis	847,413,451	Preeclampsia classified as a moderate-strength risk factor for PPH; uterine atony identified as the leading overall cause of PPH.
Cabezas Quillama (2022)	Peru	Retrospective case series	380 records / 24 cases	13.25% of women with preeclampsia with severe features developed PPH due to uterine atony; 16.66% of these had concurrent HELLP syndrome.
Samayamuthu et al. (2025)	United States (ENACT network)	Federated cross- sectional cohort	591,375 deliveries	Severe preeclampsia among the top-ranked comorbidities in PPH admissions; uterine atony the commonest identified cause.
Li et al. (2021)	China	Multicenter retrospective cohort	Multicenter sample	Hypertensive disorders of pregnancy, including preeclampsia, identified as an independent risk factor for PPH.
Barth et al. (2025)	Germany	Retrospective cohort	Single- center cohort	Hypertensive/preeclamptic pregnancies contributed to risk profiles for severe blood

Author (Year)	Country/Region	Design	Sample size	Key finding on preeclampsia and PPH
				loss and operative management of PPH.
Pettersen et al. (2025)	Norway	Hospital-based cohort	Hospital cohort	Preeclampsia associated with severe PPH attributable specifically to uterine atony rather than retained tissue.
Babadjanova & Israilova (2025)	Uzbekistan	Narrative review	—	Preeclampsia and magnesium sulfate therapy highlighted among factors impairing myometrial contractility and predisposing to atonic bleeding.
Say et al. (2014)	Global (WHO analysis)	Systematic analysis of maternal mortality data	Global data	Hemorrhage and hypertensive disorders jointly identified among the leading direct causes of maternal death worldwide.
Demissie et al. (2025)	Sub-Saharan Africa (umbrella review)	Umbrella review	Multiple reviews	Preeclampsia confirmed as a recurrent determinant of PPH burden across regional systematic reviews.
American College of Obstetricians and Gynecologists (2020)	United States	Clinical practice guideline	—	Defines severity classification of preeclampsia used as the reference framework for severity-stratified analyses in this review.

PPH: postpartum hemorrhage; aOR: adjusted odds ratio; OR: odds ratio; CI: confidence interval; HELLP: hemolysis, elevated liver enzymes, low platelet count.

Across the included studies, preeclampsia was consistently associated with an elevated risk of PPH, with unadjusted odds ratios as high as 3.1 in some population-based analyses and adjusted estimates more conservatively in the range of 1.3 to 2.2 (Gallardo Cerna et al., 2025; von Schmidt auf Altenstadt et al., 2013). The largest meta-analysis identified, pooling data from twelve primary studies and over two million pregnancies, reported an overall odds ratio of 1.95, with the strength of association varying by geographic region (Gallardo Cerna et al., 2025).

A recurring and somewhat counterintuitive finding across the largest cohort studies was that women with non-severe preeclampsia had a higher adjusted risk of PPH than women with

severe preeclampsia (von Schmidt auf Altenstadt et al., 2013). This pattern reversed, however, when the analysis was restricted to women with spontaneous (non-induced) labor, suggesting that differences in obstetric management, particularly rates of labor induction and oxytocin augmentation, may partly explain the apparent severity paradox rather than a true protective effect of disease severity itself.

With respect to underlying mechanism, uterine atony was consistently identified as the dominant proximate cause of PPH in women with preeclampsia, both in large administrative-data cohorts and in hospital-based case series (Yunas et al., 2025; Samayamuthu et al., 2025; Pettersen et al., 2025; Barth et al., 2025). In the Peruvian case series specifically restricted to preeclampsia with severe features, nearly one in eight affected women developed PPH attributable to uterine atony, and roughly one in six of these cases had concurrent HELLP syndrome, pointing to an additional hematologic contribution to bleeding risk in the more severe end of the disease spectrum (Cabezas Quillama, 2022).

Taken together, the qualitative synthesis suggests two coexisting but partially independent pathways: an obstetric-management pathway, in which induction of labor and prolonged augmented labor in non-severe preeclampsia predispose to myometrial fatigue and atony, and a disease-severity pathway, in which endothelial dysfunction, thrombocytopenia, and, in severe cases, coagulopathy directly compromise hemostasis regardless of labor characteristics.

Discussion

The findings of this review confirm that preeclampsia, across its severity spectrum, is an important and consistent risk factor for PPH, corroborating decades of accumulated observational evidence (Gallardo Cerna et al., 2025; von Schmidt auf Altenstadt et al., 2013). What is less intuitive, and arguably more clinically important, is the observation that the magnitude of this risk does not always scale linearly with disease severity. Several of the largest and most methodologically robust cohorts included in this review reported a higher adjusted risk of PPH in non-severe preeclampsia than in severe preeclampsia (von Schmidt auf Altenstadt et al., 2013).

A plausible explanation for this pattern lies in differences in clinical management rather than in the underlying pathophysiology of the disease itself. Women with severe preeclampsia are more likely to undergo expedited, often cesarean, delivery under close monitoring, with anticipatory use of uterotonics and heightened vigilance for hemorrhage; women with non-severe disease, in contrast, are more commonly managed expectantly with induction of labor at or near term, a course associated with prolonged labor, oxytocin augmentation, and consequent myometrial fatigue, all established contributors to uterine atony (Yunas et al., 2025; Babadjanova & Israilova, 2025). This interpretation is strengthened by the observation that the severity gradient reversed toward the expected direction once the analysis was restricted to spontaneous, non-induced deliveries (von Schmidt auf Altenstadt et al., 2013).

At the same time, the review identified a distinct, severity-linked pathway operating through hematologic and vascular derangement. In severe preeclampsia, particularly when complicated by HELLP syndrome, thrombocytopenia and endothelial injury can impair clot formation independently of uterine tone, and magnesium sulfate, routinely administered for seizure prophylaxis in this group, has a recognized tocolytic-like effect on myometrial

contractility that may further predispose to atony (Cunningham et al., 2022; Babadjanova & Israilova, 2025). The case series from a Peruvian referral hospital illustrates this overlap concretely, with a notable proportion of women who developed atonic PPH in the setting of severe preeclampsia also presenting with concurrent HELLP syndrome (Cabezas Quillama, 2022).

These findings carry direct clinical implications. First, PPH risk stratification in women with preeclampsia should not rely on severity classification alone; obstetric factors such as mode of onset of labor, duration of labor, and use of oxytocin augmentation should be weighed alongside disease severity when anticipating hemorrhage risk. Second, active management of the third stage of labor, including timely administration of uterotonics, remains a cornerstone intervention regardless of preeclampsia severity, consistent with global recommendations for PPH prevention (Yunas et al., 2025). Third, in women with severe features or HELLP syndrome, anticipatory availability of blood products and correction of coagulopathy should be prioritized alongside standard uterotonic management, given the added hematologic contribution to bleeding risk in this subgroup (Barth et al., 2025).

This review has several limitations. The included studies used heterogeneous definitions of both PPH (ranging from a 500 mL to a 1000 mL blood-loss threshold, with or without clinical signs of hypovolemia) and preeclampsia severity, reflecting evolving diagnostic criteria over the study period; this heterogeneity precluded a pooled quantitative meta-analysis and limited the review to a narrative synthesis. Most included studies were retrospective in design and drawn from high- and middle-income settings in Europe, the Americas, and Asia, with comparatively limited representation from Southeast Asia, including Indonesia, where the burden of both preeclampsia and PPH remains high. Future prospective, multicenter studies using standardized definitions, and specifically designed to disentangle obstetric-management factors from intrinsic disease severity, are needed to clarify the mechanisms underlying the observed severity paradox.

CONCLUSION

Preeclampsia, irrespective of whether it is classified as severe or non-severe, is a consistent and clinically important risk factor for postpartum hemorrhage. The risk appears to be shaped jointly by disease-intrinsic mechanisms, such as endothelial dysfunction and coagulopathy in severe disease, and by obstetric-management factors, particularly labor induction and augmentation, which may be more prominent in non-severe disease. Anticipatory, severity-informed management of the third stage of labor, together with vigilant hematologic assessment in women with severe features, should be regarded as essential components of care for all women with preeclampsia.

End-of-Manuscript Statements

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