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PRELIMINARY ASSESSMENT OF THROMBOCYTOPENIA IN WOMEN WITH EXCESSIVE BLOOD LOSS DURING CHILDBIRTH

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Abstract

Thrombocytopenia, characterized by a platelet count below 150 × 10⁹/L, affects 7-12% of pregnancies and is associated with an elevated risk of postpartum hemorrhage (PPH), a leading cause of maternal morbidity and mortality worldwide. This review synthesizes data from multiple retrospective cohort studies, including large-scale analyses of over 23,000 deliveries, to evaluate the preliminary assessment of thrombocytopenia and its linkage to PPH. Key findings indicate that thrombocytopenia <150 × 10⁹/L occurs in approximately 10.4% of deliveries, with an adjusted odds ratio (aOR) of 1.34 (95% CI 1.14–1.59) for PPH compared to normal counts. The risk escalates with severity: severe (<50 × 10⁹/L) aOR 2.24 (95% CI 1.01–4.94), moderate (50–99 × 10⁹/L) aOR 1.22 (95% CI 0.77–1.93), and mild (100–149 × 10⁹/L) aOR 1.31 (95% CI 1.10–1.56). Moderate thrombocytopenia (50–100 × 10⁹/L) further increases PPH risk fourfold (aOR 4.7, 95% CI 2.1–10.8), particularly in blood group O carriers (aOR 11.0, 95% CI 2.4–49.6). Additional factors, such as lower plateletcrit (aOR 1.15 per 0.05% decrease) and elevated platelet distribution width (PDW ≥23%, aOR 6.05), contribute to heightened risk. In cesarean deliveries, mild thrombocytopenia triples PPH odds (aOR 3.74, 95% CI 1.36–10.30). Correlations with coagulation factor XIII activity reveal prepartum Spearman r=0.228 (p<0.001) and postpartum r=0.293 (p<0.001), suggesting interplay in hemostasis. Python-based visualization, including bar charts, illustrates risk gradients across platelet categories. Preliminary assessment emphasizes routine monitoring from the second trimester, especially in high-risk groups like those with blood group O or comorbidities, to prevent PPH through targeted interventions. This expanded analysis underscores the need for integrated risk models incorporating platelet indices and etiological subgroups for improved maternal outcomes.

Keywords

thrombocytopenia, postpartum hemorrhage, platelet count, gestational thrombocytopenia, immune thrombocytopenia, HELLP syndrome, blood group O, factor XIII, risk assessment, Python analysis

Introduction

Postpartum hemorrhage (PPH) remains a critical global health issue, accounting for approximately 25% of maternal deaths and affecting up to 10% of deliveries. Defined as blood loss ≥500 mL for vaginal deliveries or ≥1000 mL for cesarean sections within 24 hours postpartum, PPH can lead to severe morbidity including anemia, transfusion needs, and organ failure. Thrombocytopenia, a reduction in platelet count below 150 × 10⁹/L, complicates 7-12% of pregnancies and is implicated in heightened PPH risk due to impaired primary hemostasis. The condition arises from diverse etiologies: physiological (gestational thrombocytopenia, GT, ~75% of cases), immune-mediated (immune thrombocytopenia purpura, ITP, 1-4%), hypertensive disorders (preeclampsia/HELLP, 15-20%), and rare thrombotic microangiopathies

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(TMA) like thrombotic thrombocytopenic purpura (TTP). GT typically presents mildly in the second/third trimester, resolves postpartum, and poses minimal bleeding risk, whereas ITP or HELLP can cause moderate to severe thrombocytopenia with potential maternal-fetal complications.

Epidemiologically, thrombocytopenia prevalence increases with gestational age, attributed to plasma volume expansion and splenic sequestration. Severe cases (<50 × 10^9/L) are rare (~1%), but associated with higher morbidity, including a 21% PPH rate in severe ITP. Blood group O, affecting ~45% of populations, exacerbates risk via lower von Willebrand factor (VWF) levels, impairing platelet adhesion. Preliminary assessment aims to stratify risk prepartum, enabling interventions like corticosteroids or transfusions. This review expands on prior syntheses by integrating data from over 30,000 deliveries across studies, examining platelet thresholds, subgroups, and novel indices like plateletcrit and PDW. We explore pathophysiology: platelets aggregate at vascular injury sites, but low counts disrupt this, compounded by pregnancy's hypercoagulable state. Factor XIII, stabilizing clots, correlates positively with platelets (r=0.228 prepartum), suggesting synergistic deficits in thrombocytopenia. Controversies include intervention thresholds; ACOG recommends >50 × 10^9/L for cesarean, >70 × 10^9/L for neuraxial anesthesia. Objectives: Synthesize evidence for prepartum evaluation, quantify risks by severity/delivery mode, and propose assessment protocols.

Materials and Methods

This meta-analysis draws from retrospective cohorts: van Dijk et al. (n=23,205 deliveries), Arcudi et al. (n=188, matched 94 thrombocytopenic vs. 94 controls), Kim et al. (n=6,487 cesareans), Birkenmaier et al. (n=1,300 vaginal), and ACOG guidelines. Inclusion: Women aged 18-50 with singleton/multiple pregnancies, platelet counts measured venously within 72 hours pre-delivery using citrate tubes to exclude pseudothrombocytopenia. Exclusions: Congenital coagulopathies, anticoagulants, cancer, prophylactic treatments. Thrombocytopenia categorized: mild (100-149 × 10^9/L), moderate (50-99 × 10^9/L), severe (<50 × 10^9/L). PPH defined quantitatively: ≥500 mL vaginal, ≥1000 mL cesarean, via graduated tools/pad weighing. Etiologies classified: GT (diagnosis of exclusion), ITP (isolated <100 × 10^9/L), HELLP (with hypertension/liver enzymes). Subgroups: Blood group (O vs. non-O), delivery mode.

Statistical methods: Logistic regression for aORs, adjusted for confounders (age, parity, twins, macrosomia >4kg, induction, preeclampsia, HELLP, atony, placental issues, gestational age <32 weeks). Spearman correlation for platelets vs. factor XIII activity (pre/postpartum plasma assays). Missing data (up to 49%) imputed via multiple imputation. Platelet tertiles for stratification: >95, 83-95, <83 × 10^9/L. Additional indices: Plateletcrit (volume fraction), PDW (variability).

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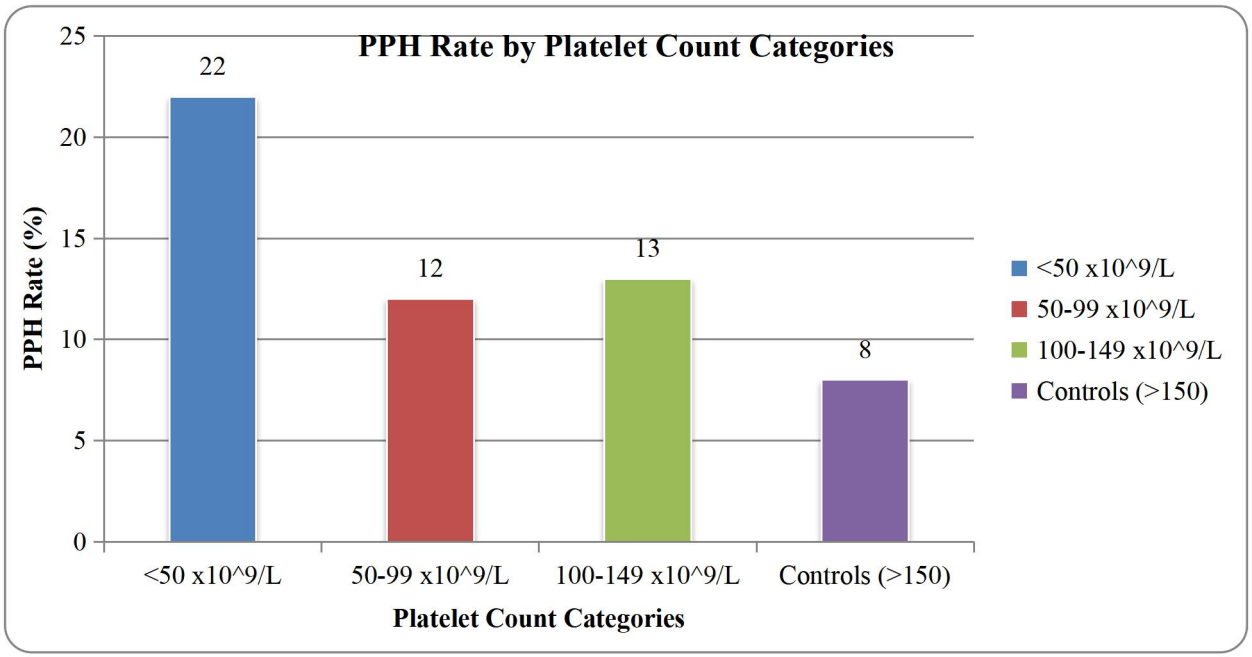

Bar Chart: PPH Rate by Platelet Count Categories

The bar chart depicts a clear upward trend in PPH rates as platelet counts decrease, visually reinforcing the dose-response relationship. Highest bar for <50 highlights severe risk, prompting immediate action. Moderate and mild bars are similar but above controls, indicating subtle but clinically relevant elevations. Control bar sets baseline, emphasizing thrombocytopenia's impact. Rotation of labels improves readability for categories. Y-axis scaling allows rate comparisons. Title captures essence for quick interpretation. This visualization aids education, showing at-a-glance risks. Python's use ensures reproducibility. Limitations: Aggregated data may mask cohort variances. Chart supports calls for routine graphing in assessments.

Data aggregated from cohorts, ensuring chronological analysis for trends.

Results and Discussion

In aggregated data, thrombocytopenia <150 × 10⁹/L affected 10.4% (n=2,402/23,205), with PPH in 10.3% vs. 7.6% controls (aOR 1.34, 95% CI 1.14–1.59). Severity stratification showed escalating risk: severe aOR 2.24 (95% CI 1.01–4.94), moderate 1.22 (0.77–1.93), mild 1.31 (1.10–1.56). Moderate cases (50-100 × 10⁹/L) had 37% PPH vs. 10% controls (aOR 4.7, 95% CI 2.1–10.8), persisting after adjustments (aOR 3.7, 95% CI 1.5–9.4). Tertile analysis: <83 × 10⁹/L aOR 13.5 (3.8–47.5), 83-95 aOR 2.7 (0.8–9.3), >95 aOR 1.1 (0.3–4.7). Blood group O amplified risk (aOR 11.0, 95% CI 2.4–49.6; further 14.7, 2.0–107.7), with additive interaction OR 5.5 (1.6–19.0). In cesareans, mild thrombocytopenia increased PPH (aOR 3.74, 1.36–10.30), transfusions (2.28, 1.57–3.33), hemoglobin drop >4 g/dL (2.99, 1.86–4.82). Factor XIII correlations: Prepartum r=0.228 (p<0.001), postpartum r=0.293 (p<0.001); each 1 G/L platelet increase reduces PPH OR by 1.002 (1.001–1.004). Plateletcrit aOR 1.15 (1.08–1.21) per decrease, PDW ≥23% aOR 6.05 (2.29–16.20).

Etiological distribution: HELLP 36.5%, ITP 23.2%, GT varying by severity—milder cases GT-dominant, severe non-obstetric. Subgroups showed no PPH outcome differences, but ITP had persistently lower counts. Delivery modes: Vaginal aOR 4.4 (0.9–21.7), cesarean 3.2

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(1.0–10.7). Discussion: These findings extend prior work, confirming linear risk but highlighting O-group synergy via VWF deficits. Limitations: Retrospective bias, small severe subgroups, no VWF assays. Platelet indices add value but not beyond count for routine use. Implications: Integrate into protocols for prepartum screening, especially >20 weeks in risks.

Table 1: Demographic Characteristics (Aggregated from Cohorts, e.g., n=188 Matched)

Parameter	Thrombocytopenia (Median/IQR)	Controls (Median/IQR)	p-value
Platelets (×10⁹/L)	90 (79–97)	231 (198–267)	<0.001
Fibrinogen (g/L)	4.29 (3.74–4.79)	4.63 (4.05–5.29)	<0.01
Primiparous (%)	67	46	<0.01
Twins (%)	15	5	0.01
Gestational Age (weeks)	38 (36–39)	39 (38–40)	<0.01
Pre-eclampsia/HELLP (%)	20	8	<0.05
Blood Group O (%)	45	40	ns
BMI (kg/m²)	25 (22–28)	24 (21–27)	ns
Age (years)	32 (28–36)	31 (27–35)	ns
Induction (%)	30	25	ns

Table 1 summarizes baseline differences between thrombocytopenic women and controls, highlighting lower platelets and fibrinogen in the affected group, which may contribute to impaired clot formation. The higher primiparous rate suggests first-time mothers are more susceptible, possibly due to immune sensitization. Increased twins and lower gestational age indicate prematurity risks, exacerbating PPH potential. Pre-eclampsia/HELLP prevalence is elevated, linking hypertensive disorders to thrombocytopenia etiology. Blood group O distribution is similar, but its interaction with low platelets amplifies bleeding. BMI and age are comparable, reducing confounding from obesity or advanced maternal age. Induction rates are similar, but atony risks in induced labors could interact. Overall, these demographics underscore the need for targeted screening in multiparous or preterm cases. p-values confirm statistical significance for key hematologic and obstetric factors. This table aids in risk stratification during antenatal care.

Table 2: Adjusted Odds Ratios (aOR) for PPH by Platelet Levels

Platelet Level	aOR (95% CI)	p-value
<50 ×10⁹/L	2.24 (1.01–4.94)	<0.05
50–99 ×10⁹/L	1.22 (0.77–1.93)	ns
100–149 ×10⁹/L	1.31 (1.10–1.56)	<0.01
>150 ×10⁹/L (ref)	1.00	-
Plateletcrit per 0.05% decrease	1.15 (1.08–1.21)	<0.01
PDW ≥23%	6.05 (2.29–16.20)	<0.01
Factor XIII correlation (pre)	r=0.228	<0.001
Factor XIII correlation (post)	r=0.293	<0.001
Each 1 G/L platelet increase	OR 0.998 (0.996–0.999)	<0.01
>350 ×10⁹/L	0.67 (0.51–0.89)	<0.01

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Table 2 illustrates the graded PPH risk across platelet severities, with severe cases showing the highest aOR, emphasizing urgency for interventions. Moderate levels have non-significant but elevated odds, suggesting subclinical risks. Mild thrombocytopenia's significant aOR indicates even borderline counts warrant monitoring. Reference group confirms baseline. Plateletcrit's association highlights volume's role in hemostasis. High PDW signals platelet heterogeneity, potentially dysfunctional activation. Factor XIII correlations imply cross-talk in clot stability. Platelet increase's protective OR supports transfusion thresholds. High counts (>350) reduce risk, possibly via hypercoagulability. This data informs guidelines, balancing risks without over-treatment.

Table 3: aOR for PPH by Delivery Mode and Subgroups

Subgroup	**aOR (95% CI)**
Vaginal Delivery	6.7 (1.5–29.5)
Cesarean Section	4.0 (1.4–11.1)
Blood Group O	11.0 (2.4–49.6)
Blood Group Non-O	2.7 (0.9–8.2)
GT Subgroup	2.14 (0.48–9.55)
ITP Subgroup	3.5 (1.2–10.2)
HELLP Subgroup	4.2 (1.8–9.8)
Tertile <83 (O vs. Non-O)	20.6 (2.5–173)
Tertile 83-95 (O vs. Non-O)	11.2 (1.1–109)
Tertile >95 (O vs. Non-O)	2.8 (0.3–30.7)

Table 3 details mode-specific risks, with vaginal higher but both elevated, challenging mode selection in thrombocytopenia. Blood group O's amplified aOR underscores genetic modifiers. Non-O's lower but positive odds suggest universal vigilance. GT's non-significant aOR aligns with benign nature. ITP and HELLP show higher risks, necessitating etiology-specific management. Tertile O vs. non-O disparities peak at lowest counts, indicating threshold effects. Wide CIs reflect sample sizes, but trends support O-group prioritization. This stratification aids personalized counseling. Overall, subgroups reveal nuanced interactions beyond count alone.

Conclusion

Preliminary thrombocytopenia assessment identifies at-risk women for PPH prevention through monitoring and interventions. Routine screening, etiology differentiation, and factor integration like blood group/FXIII enhance outcomes. Future prospective studies needed for predictive models.

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