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EARLY IDENTIFICATION AND MANAGEMENT OF POSTPARTUM HEMORRHAGE IN THE LABOUR ROOM: A PROSPECTIVE OBSERVATIONAL STUDY

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ABSTRACT

Background: Postpartum hemorrhage (PPH) remains the leading single cause of maternal mortality worldwide. Early recognition and protocolized first-response management in the labour room are critical determinants of outcome. This study aimed to evaluate the incidence, risk factors, timeliness of recognition, and management of PPH in a tertiary-care labour room.

Methods: A prospective observational study was conducted over 12 months (July 2024–June 2025) among 1,500 consecutive deliveries. Blood loss was measured objectively using calibrated under-buttocks drapes. PPH was defined as blood loss ≥500 mL after vaginal birth or ≥1,000 mL after caesarean section within 24 hours. A structured data collection tool captured the "4 Ts" etiology, timing of each element of the first-response bundle, interventions, and maternal outcomes. Logistic regression identified independent risk factors.

Results: PPH occurred in 157 women (10.5%; 95% CI 8.9–12.0). The rate was higher after caesarean section than vaginal birth (13.3% vs 9.5%; p = 0.040). Uterine atony accounted for 72.0% of cases. Independent risk factors included retained placenta (aOR 30.0), previous PPH (aOR 5.6), anaemia (aOR 3.2), macrosomia (aOR 3.4), and multiple pregnancy (aOR 3.6). Recognition within 15 minutes occurred in 87.3% of cases; tranexamic acid was given within 30 minutes of diagnosis in 81.5%. A shock index ≥1 at diagnosis was strongly associated with severe PPH (OR 15.8, 95% CI 6.7–37.6). Early tranexamic acid was associated with lower odds of severe PPH (OR 0.14, 95% CI 0.06–0.33). Hysterectomy was required in 1.9% and one maternal death occurred (case fatality 0.64%).

Conclusions: Objective measurement enabled early detection, yet delays in escalation persisted. Structured, time-stamped bundles with early tranexamic acid and shock-index-guided triage are feasible in busy labour rooms and may reduce severe hemorrhage.

Keywords: postpartum hemorrhage; uterine atony; tranexamic acid; labour room; shock index; obstetric hemorrhage bundle

1. INTRODUCTION

Postpartum hemorrhage (PPH) conventionally defined as blood loss of ≥500 mL within 24 hours of birth complicates approximately 6–11% of deliveries globally and remains the leading single cause of maternal death, accounting for roughly one in four maternal deaths worldwide [1,2]. The World Health Organization's systematic analysis of maternal death causation found that

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haemorrhage was responsible for 27.1% of maternal deaths between 2003 and 2009, a proportion that has remained stubbornly stable in subsequent global estimates [1,3]. Most of these deaths are preventable: the condition is treatable with inexpensive uterotonics, antifibrinolytics, fluid resuscitation, and when required surgical intervention, provided that bleeding is recognized early and managed without delay [4,5].

The epidemiology of PPH is paradoxical. In high-income countries, PPH rates have risen over the past two decades even as mortality has fallen, attributed to increasing caesarean section rates, older maternal age, obesity, and induction of labour [6,7]. In low- and middle-income countries (LMICs), the inverse pattern prevails: incidence is similar or higher, but case fatality is far greater because of delayed recognition, inadequate blood product availability, and weak referral chains [8]. Calvert et al., in a systematic review of over one million births, estimated a global PPH prevalence of 10.8%, with severe PPH (≥1,000 mL) affecting approximately 2.8% of women [5]. Crucially, these estimates are method-dependent: visual estimation of blood loss systematically underestimates true loss by up to 30–50%, whereas objective gravimetric or calibrated-drape measurement reveals substantially higher PPH rates [5,9,10].

Uterine atony is the dominant mechanism, responsible for an estimated 70–80% of cases; retained products of conception, genital tract trauma, and coagulopathy complete the classic "4 Ts" mnemonic (tone, tissue, trauma, thrombin) [6,8]. Although numerous antenatal and intrapartum risk factors are recognized including grand multiparity, previous PPH, multiple pregnancy, macrosomia, anaemia, prolonged labour, induction of labour, operative vaginal delivery, and caesarean section most PPH occurs in women without identifiable risk factors, underscoring the need for universal preparedness rather than selective vigilance [7,11,12].

Two developments have reshaped the management landscape. First, the WOMAN trial demonstrated that early intravenous tranexamic acid (TXA), administered within three hours of birth, reduces death from bleeding by approximately one third without increasing thromboembolic events, prompting the WHO to recommend TXA as a core component of the PPH treatment package [13,14,15]. Second, a growing body of implementation research supports bundled, protocolized, multidisciplinary first-response care simultaneous uterine massage, uterotonics, TXA, intravenous access with blood sampling, genital tract examination, and escalation rather than sequential therapeutic escalation [16,17,18]. FIGO's 2022 recommendations and the WHO's 2025 consolidated guidelines explicitly endorse such bundle-based care, with objective blood loss measurement as the trigger [18,19].

Despite this strong evidence base, gaps persist between guideline and practice, particularly in the high-pressure environment of the labour room where recognition delays of even 15–30 minutes measurably increase the risk of severe morbidity [14,16]. Prospective observational data quantifying the timeliness of each bundle element remain scarce from many settings. The present study was therefore designed to (i) determine the incidence and severity spectrum of PPH using

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objective blood loss measurement; (ii) identify the etiological profile and independent risk factors; and (iii) evaluate the timeliness of recognition and first-response management, and their association with outcomes, in a tertiary-care labour room.

2. MATERIALS AND METHODS

2.1 Study design and setting

This was a prospective observational study conducted in the labour room and obstetric operating theatre of a tertiary-care teaching hospital with approximately 6,000 annual deliveries, 24-hour consultant obstetric cover, on-site blood bank, and intensive care facilities. The study period was 12 months (July 2024 to June 2025).

2.2 Study population

All women delivering in the facility during the study period were eligible. Women with antepartum hemorrhage as the primary diagnosis, those delivered before arrival, and women with incomplete blood loss documentation were excluded. Consecutive sampling was used; of 1,536 deliveries during the period, 1,500 met inclusion criteria and formed the analytical cohort.

2.3 Definitions

Primary PPH was defined as cumulative blood loss ≥500 mL within 24 hours of vaginal birth or ≥1,000 mL after caesarean section, in line with WHO and FIGO criteria [14,19]. Severe PPH was defined as blood loss ≥1,000 mL (vaginal) or ≥1,500 mL (caesarean), and massive PPH as ≥1,500 mL [16]. Maternal near-miss was defined using WHO criteria (organ dysfunction-based), and severe maternal outcome as the composite of near-miss and death [3]. The shock index was calculated as pulse rate divided by systolic blood pressure, with a value ≥1 considered abnormal [18].

2.4 Blood loss measurement

All vaginal births used a calibrated, graduated under-buttocks drape (Brass-V type) applied immediately after birth, read at placental expulsion and at 15-minute intervals for two hours; saturated pads, linen, and cannula spillage were weighed (gravimetric conversion: 1 g ≈ 1 mL). For caesarean births, suction canister volumes, swab weights, and measured peritoneal collection were summed. Clinical staff were trained in drape use and in the calibrated drape's printed volume markings before study commencement [9,10].

2.5 Data collection

A structured, time-stamped case record form captured: sociodemographic and obstetric history; antenatal care (ANC) attendance; intrapartum events (induction, duration of labour, mode of delivery, perineal trauma); serial vital signs and shock index; blood loss volumes; etiology per the 4 Ts; timing of each first-response bundle element (recognition, call for help, intravenous access and blood sampling, first and second uterotonics, TXA, genital tract examination, escalation to senior clinician); second-line interventions (balloon tamponade, examination under anaesthesia, relaparotomy, hysterectomy); transfusion; and outcomes (ICU admission, near-miss, death, length

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of stay). Data were collected by trained midwifery research officers and verified daily against case notes by the principal investigator.

2.6 Statistical analysis

Categorical variables are reported as frequencies with percentages and compared using the chi-square or Fisher exact test, as appropriate; continuous variables as mean ± standard deviation or median with interquartile range. Crude odds ratios (OR) with 95% confidence intervals were computed for the association between each candidate risk factor and PPH. Variables with $p < 0.20$ at bivariate analysis were entered into a multivariable logistic regression model to derive adjusted odds ratios (aOR); model fit was assessed with the Hosmer–Lemeshow test. The association between timeliness metrics, shock index, and severe PPH was evaluated using ORs with 95% CIs. A two-sided $p < 0.05$ was considered statistically significant. Analyses were performed using SPSS version 26 and R version 4.3.

2.7 Sample size and ethics

The 12-month census of approximately 1,500 deliveries provided ≥80% power to estimate a PPH incidence of 10% with a precision of ±1.5%, and to detect an odds ratio of ≥1.8 for risk factors with prevalence ≥10%. Ethical clearance was obtained from the Institutional Ethics Committee, written informed consent was obtained from participants for the use of their anonymized data. The study followed STROBE guidelines for observational research.

3. Results

3.1 Cohort profile and incidence

Of 1,500 women, 1,123 (74.9%) delivered vaginally and 377 (25.1%) by caesarean section. PPH occurred in 157 women, an incidence of 10.5% (95% CI 8.9–12.0). The incidence was significantly higher after caesarean section than after vaginal birth (13.3% vs 9.5%; $p = 0.040$). Of the 157 cases, 107 (68.2%) were mild (500–999 mL) and 50 (31.8%) were severe; 7 women (4.5% of PPH cases) experienced massive hemorrhage ≥1,500 mL. Mean measured blood loss among PPH cases was 870 ± 330 mL (median 800 mL; IQR 620–1,080), compared with 275 ± 115 mL among women without PPH.

Table 1. Incidence and severity of PPH by mode of delivery (N = 1,500)

Mode of delivery	Deliveries, n	PPH, n (%)	Mild (500–999 mL), n	Severe (≥1,000 mL), n
Vaginal	1,123	107 (9.5)	81	26
Caesarean section	377	50 (13.3)	26	24
Total	**1,500**	**157 (10.5)**	**107**	**50**

p = 0.040 for mode-of-delivery comparison (chi-square).

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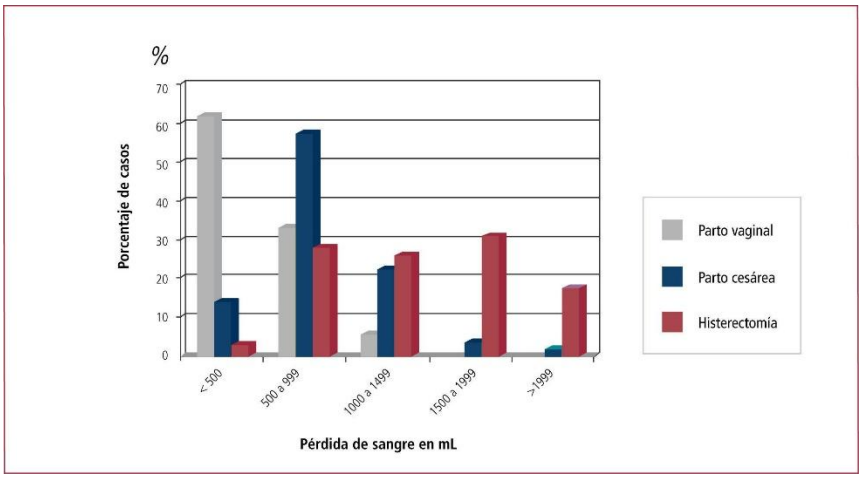

Figure 1: Incidence of Postpartum Hemorrhage by Mode of Delivery

3.2 Etiology

Uterine atony was the leading cause, accounting for 113 cases (72.0%), followed by genital tract trauma in 24 (15.3%), retained tissue in 14 (8.9%), and coagulopathy in 6 (3.8%). Multiple contributing causes were recorded in 11 cases (7.0%).

Table 2. Etiological profile of PPH (4 Ts) (n = 157)

Cause ("4 Ts")	n	% of PPH
Tone uterine atony	113	72.0
Trauma genital tract lacerations, uterine rupture	24	15.3
Tissue retained placenta/products	14	8.9
Thrombin coagulopathy	6	3.8

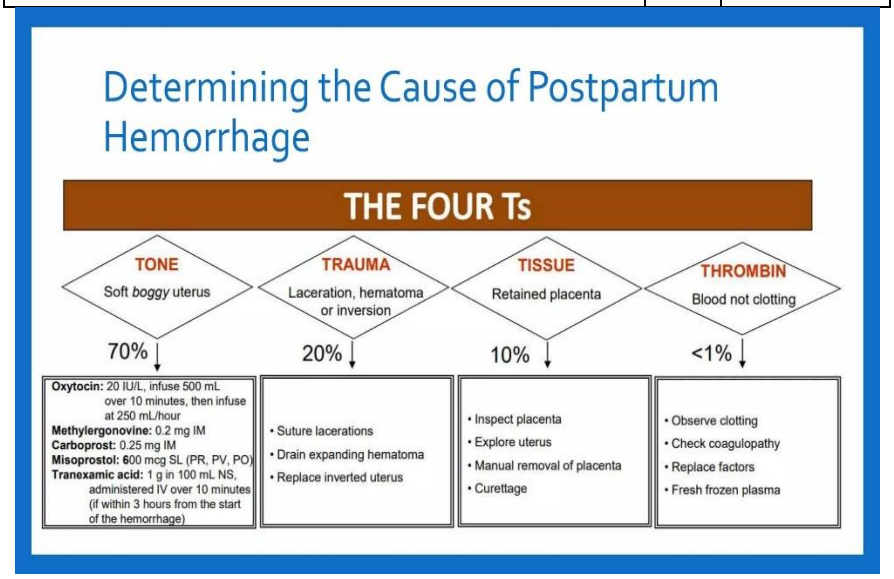

Figure 2: Etiological Distribution of PPH (4 Ts)

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3.3 Risk factors

Table 3. Bivariate analysis of risk factors for PPH (N = 1,500; PPH n = 157)

Variable	PPH (n = 157)	No PPH (n = 1,343)	Crude OR (95% CI)	p
Age ≥35 years	29 (18.5%)	153 (11.4%)	1.76 (1.14–2.73)	0.010
Grand multiparity (≥P4)	26 (16.6%)	150 (11.2%)	1.58 (1.00–2.49)	0.047
Anaemia (Hb <11 g/dL)	92 (58.6%)	481 (35.8%)	2.54 (1.81–3.55)	<0.001
Obesity (BMI ≥30 kg/m²)	30 (19.1%)	150 (11.2%)	1.88 (1.22–2.90)	0.004
Previous PPH	10 (6.4%)	20 (1.5%)	4.50 (2.07–9.80)	<0.001
Multiple pregnancy	7 (4.5%)	19 (1.4%)	3.25 (1.35–7.86)	0.006
Macrosomia (≥4 kg)	21 (13.4%)	70 (5.2%)	2.81 (1.67–4.72)	<0.001
Prolonged labour (>12 h)	30 (19.1%)	164 (12.2%)	1.70 (1.11–2.61)	0.015
Induction of labour	36 (22.9%)	195 (14.5%)	1.75 (1.17–2.62)	0.006
Operative vaginal delivery	7 (4.5%)	40 (3.0%)	1.52 (0.67–3.45)	0.314
Caesarean section	50 (31.8%)	327 (24.3%)	1.45 (1.02–2.08)	0.040
Major perineal trauma (3rd/4th degree)	20 (12.7%)	96 (7.1%)	1.90 (1.14–3.17)	0.013
Inadequate ANC (<3 visits)	22 (14.0%)	111 (8.3%)	1.81 (1.11–2.96)	0.017
Retained placenta	10 (6.4%)	4 (0.3%)	22.8 (7.05–73.5)	<0.001

Table 4. Independent risk factors for PPH: multivariable logistic regression

Variable	Adjusted OR (95% CI)	p
Retained placenta	30.0 (8.6–104.7)	<0.001
Previous PPH	5.6 (2.4–13.0)	<0.001
Multiple pregnancy	3.6 (1.3–9.9)	0.012
Macrosomia (≥4 kg)	3.4 (1.9–6.1)	<0.001
Anaemia (Hb <11 g/dL)	3.2 (2.2–4.6)	<0.001
Obesity (BMI ≥30 kg/m²)	2.2 (1.4–3.6)	0.001
Age ≥35 years	2.0 (1.3–3.2)	0.004
Grand multiparity (≥P4)	1.9 (1.1–3.1)	0.014
Prolonged labour (>12 h)	1.8 (1.1–2.8)	0.013
Induction of labour	1.8 (1.2–2.8)	0.008
Major perineal trauma	2.0 (1.1–3.4)	0.017
Inadequate ANC (<3 visits)	1.8 (1.0–3.1)	0.040
Caesarean section	1.5 (1.0–2.3)	0.029
Operative vaginal delivery	2.0 (0.8–4.7)	0.130

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Hosmer–Lemeshow p = 0.42.

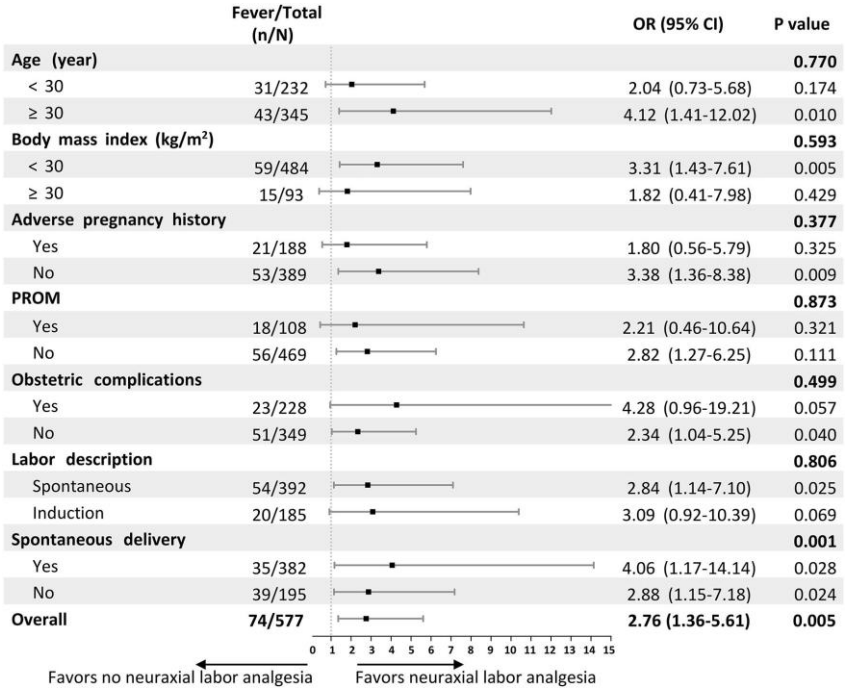

Figure 3: Independent Risk Factors for PPH (Adjusted Odds Ratios)

3.4 Timeliness of recognition and first response

A shock index ≥1 at diagnosis was present in 41 women (26.1%) and was strongly associated with severe PPH (62.0% of severe cases vs 9.3% of mild cases; OR 15.8, 95% CI 6.7–37.6; p < 0.001). Timeliness metrics are summarized in Table 5. The most frequent delays were escalation to a senior clinician within 15 minutes (achieved in 70.1%) and administration of a second uterotonic within 15 minutes (75.8%).

Table 5. Timeliness of first-response bundle elements among PPH cases (n = 157)

Bundle element	Achieved within target, n (%)	Target
Recognition of PPH (drape reading + vital signs)	137 (87.3)	≤15 min from delivery
IV access + blood sampling for cross-match	143 (91.1)	≤10 min of recognition
First additional uterotonic (after prophylaxis)	148 (94.3)	≤10 min
Second uterotonic / TXA decision	119 (75.8)	≤15 min
Tranexamic acid administered	139 (88.5) overall; 128 (81.5) ≤30 min of diagnosis	≤30 min

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Genital tract examination (4 Ts documentation)	126 (80.3)	≤20 min
Escalation call to senior obstetrician	110 (70.1)	≤15 min

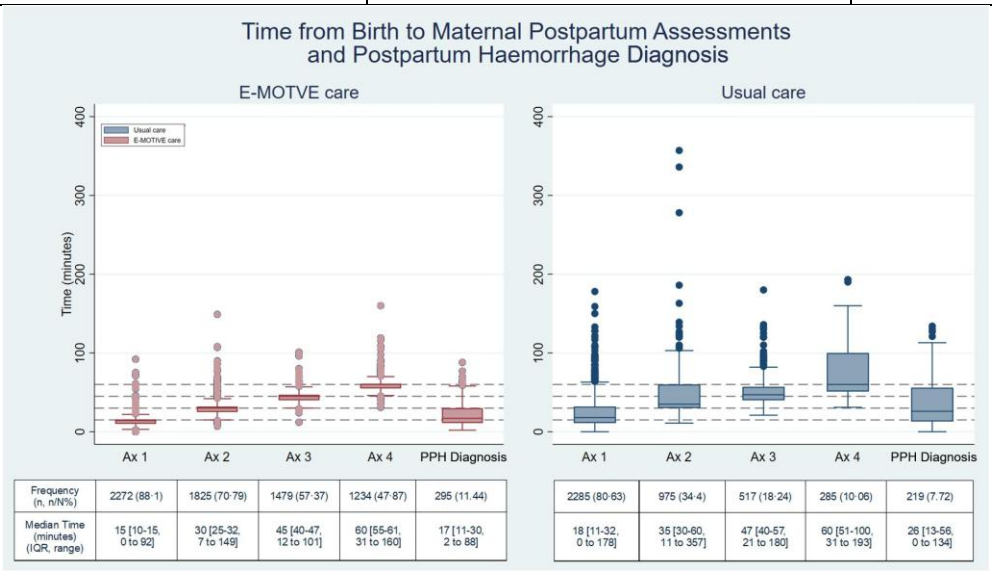

Figure 4: Timeliness of First-Response Interventions

3.5 Management and maternal outcomes

All women received uterine massage. Additional uterotonics were given to 94.3%; TXA to 88.5%. Second-line interventions included balloon tamponade in 19 women (12.1%), successful in 17 (89.5%); examination under anaesthesia with repair in 24 (15.3%); relaparotomy in 5 (3.2%); and peripartum hysterectomy in 3 (1.9%). Blood transfusion was required in 34 women (21.7%) 54.0% of severe cases versus 6.5% of mild cases (OR 16.8, 95% CI 6.5–43.2). Early TXA (≤30 minutes of diagnosis) was associated with markedly lower odds of severe PPH (23.4% vs 69.0% with delayed or no TXA; OR 0.14, 95% CI 0.06–0.33). Eleven women (7.0%) required ICU admission; 12 (7.6%) fulfilled near-miss criteria; 5 (3.2%) developed disseminated intravascular coagulation and 2 acute kidney injury. One woman died of refractory atony with delayed presentation of anaemia (PPH case fatality 0.64%; maternal mortality ratio 66.7 per 100,000 deliveries). Mean hospital stay was 4.5 ± 2.7 days among PPH cases versus 2.2 ± 0.9 days among controls.

Table 6. Management and outcomes among PPH cases (n = 157)

Intervention / outcome	n (%)
Additional uterotonics	148 (94.3)
Tranexamic acid	139 (88.5)
Balloon tamponade (success)	19 (12.1) [17/19, 89.5%]
Examination under anaesthesia / repair	24 (15.3)
Relaparotomy	5 (3.2)

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Peripartum hysterectomy	3 (1.9)
Blood transfusion	34 (21.7)
ICU admission	11 (7.0)
Maternal near-miss	12 (7.6)
Disseminated intravascular coagulation	5 (3.2)
Maternal death	1 (0.64)
Mean hospital stay, days (± SD)	4.5 (± 2.7)

4. DISCUSSION

4.1 Incidence in context

The observed PPH incidence of 10.5% lies within the range reported by the major systematic reviews: Carroli et al. estimated a global prevalence of approximately 6% for PPH ≥500 mL and 1.9% for severe PPH, while Calvert et al. using more objective measurement methods reported a pooled prevalence of 10.8% overall and 2.8% for severe PPH [4,5]. Our severe PPH proportion (31.8% of PPH cases; 3.3% of all deliveries) is consistent with the higher end of the severe PPH distribution, plausibly reflecting both objective drape measurement and a referral-level case mix enriched with high-risk pregnancies [5]. Our rate also closely resembles the 9.0% reported by Ononge et al. in a Ugandan cohort that, like ours, used calibrated drapes rather than visual estimation [12]. This convergence reinforces the central methodological message of the PPH literature: measured blood loss yields systematically higher and almost certainly more accurate incidence estimates than visual estimation, which underestimates loss by 30% or more and thereby delays diagnosis [5,10].

4.2 Etiology and risk factors

Uterine atony accounted for 72.0% of cases, in close agreement with the approximately two-thirds to 80% attributable fraction reported in the WOMAN trial, the WHO Multicountry Survey, and ACOG analyses [8,13,6]. The multivariable model identified a risk factor profile that is highly concordant with the global evidence: retained placenta (aOR 30.0), previous PPH (aOR 5.6), multiple pregnancy (aOR 3.6), macrosomia (aOR 3.4), and anaemia (aOR 3.2) were the strongest independent predictors [7,11,12]. The magnitude of the retained-placenta association mirrors the extreme odds ratios reported in the WHO Multicountry Survey, where retained placenta carried one of the highest adjusted risks for severe outcomes [7].

Two findings warrant emphasis. First, anaemia present in 58.6% of PPH cases and independently associated with a tripling of odds is a modifiable risk factor of particular importance in settings where it is endemic; anaemic women tolerate hemorrhage poorly, tolerate transfusion less safely, and were over-represented among hemorrhage deaths in the WOMAN trial's contextual analysis [13,20]. Antenatal iron supplementation and active third-stage management in anaemic women should therefore be regarded as hemorrhage-prevention strategies, not merely nutritional ones [17]. Second, caesarean section independently increased PPH odds (aOR 1.5), and accounted for

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nearly half of severe cases despite representing only a quarter of deliveries a pattern consistent with rising PPH trends in high-caesarean settings and with the ACOG observation that atony-driven PPH has increased in parallel with caesarean rates [6,21].

4.3 Timeliness: where the system worked and where it lagged

The study's core contribution is its time-stamped dissection of first-response care. Recognition within 15 minutes was achieved in 87.3% of cases a direct dividend of universal calibrated drape use, which removes the cognitive burden of estimating loss amid a crowded delivery scene [9,10]. This supports the trajectory of international guidelines toward drape-triggered care, including the WHO's 2025 consolidated guidelines and the FIGO recommendations, which elevate objective measurement to a first-line strategy [18,19].

However, the bundle's weakest links were escalation (70.1% within 15 minutes) and second uterotonic administration (75.8%) precisely the steps requiring hierarchical action rather than individual performance. The strong association between shock index ≥1 and severe PPH (OR 15.8) suggests that shock index, endorsed by FIGO as a triage adjunct, could serve as an objective "early warning" trigger to accelerate escalation in settings where drape readings lag [18]. Our observational finding that TXA within 30 minutes of diagnosis was associated with an 86% reduction in the odds of severe PPH is consistent with though it cannot match the causal strength of the WOMAN trial's mortality finding and its time-dependent subgroup analysis, in which benefit was confined to administration within three hours of birth [13,14,15]. These convergent lines of evidence argue against the "wait-and-see" sequencing still implicit in some protocols and support giving TXA immediately upon diagnosis, alongside the first uterotonic [15,16].

4.4 Management burden and outcomes

The intervention profile balloon tamponade success of 89.5%, hysterectomy rate of 1.9%, and transfusion in 21.7% indicates that a stepwise conservative-to-surgical sequence was largely maintained, in line with FIGO's recommendation that invasive procedures follow failure of uterotonics and temporizing measures [18,19]. Our hysterectomy rate sits below the 3.6% reported in the WOMAN trial, plausibly reflecting earlier arrest of bleeding with bundle care in a facility with full surgical capability [13]. The single death, in a woman with severe antenatal anaemia and delayed presentation, illustrates the persistent lethal triad of anaemia, delayed recognition, and blood product scarcity described in the WOMAN trial's mortality analysis and remains a health-system rather than purely clinical failure [20,22].

4.5 Strengths and limitations

Strengths include the prospective design, consecutive sampling, universal objective blood loss measurement, time-stamped process evaluation, and multivariable adjustment. Limitations merit emphasis: first, as a single-centre study in a tertiary facility, findings may not generalize to primary-care or home-birth settings where most global PPH deaths occur [22]. Second, despite drapes, blood loss at caesarean section retains measurement error. Third, the observational design

precludes causal inference the apparent protective association of early TXA is susceptible to confounding by indication, and prospective validation is needed. Fourth, the modest number of severe cases limits power for some subgroup analyses. Finally, the study did not capture the quality of antenatal anemia management, a potentially major upstream determinant.

5. CONCLUSION

In this prospective labour-room study, objective measurement yielded a PPH incidence of 10.5% with atony predominating, and confirmed retained placenta, previous PPH, multiple pregnancy, macrosomia, and anaemia as the dominant independent risk factors. Recognition was rapid where drapes were used, but escalation and second-drug timeliness lagged. Early tranexamic acid and shock-index-guided triage emerged as actionable targets. We recommend: (i) universal calibrated-drape measurement with a defined drape-reading protocol; (ii) a laminated, time-stamped first-response bundle displayed at every delivery station; (iii) routine shock index calculation in the immediate postpartum period; (iv) TXA immediately upon PPH diagnosis; and (v) antenatal anemia control as a hemorrhage-prevention strategy. Implementation research evaluating bundle fidelity against severe PPH outcomes should follow.

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