

Analysis of Risk Factors and Establishment of a Risk Prediction Model for Severe Postpartum Haemorrhage

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Abstract

Aims/Background Severe postpartum haemorrhage (PPH) is a dangerous condition, characterized by rapid progression and poor prognosis. It remains the leading preventable cause of maternal death worldwide. This study aimed to investigate the risk factors for severe PPH and establish a prediction model to identify severe PPH early, allowing for early intervention reduce maternal death.

Methods Clinical data were collected from 784 patients diagnosed with PPH and delivered at the Second Affiliated Hospital of Anhui Medical University between December 2018 and December 2023. These cases were categorized into the training cohort. Severe PPH was diagnosed in 234 patients based on the criterion of the volume of vaginal bleeding volume exceeding 1000 mL within 24 hours after delivery; these patients were assigned to the experimental group. The remaining 550 patients with nonsevere PPH were assigned to the control group. Data from an additional 338 postpartum women from the same period were selected and classified into the validation cohort. Univariate and multivariate logistic regression analyses were performed to pinpoint the determinants associated with severe PPH. Additionally, these analyses were instrumental for developing and assessing a prediction model to forecast the risk of such complications.

Results Most of the PPH cases in this study stemmed from uterine atony, the leading aetiology. The second most common factor was soft birth canal lacerations and haematoma formation, accounting for 7.26% of the subjects in experimental group and 6.55% of those in the control group. Uterine rupture, uterine inversion, and amniotic fluid embolism were exclusively observed in the experimental group. In the univariate analysis, notable disparities were identified across various parameters, including maternal age, gravidity, parity, abortion frequency, gestational week at delivery, prothrombin time (PT), activated partial thromboplastin time (APTT), *in vitro* fertilisation, foetal position, caesarean delivery, pregnancy with anaemia, and hypertensive disorders of pregnancy ($p < 0.05$). A multivariate logistic regression model revealed that a parity of two or more, two or more abortions, *in vitro* fertilisation, gestational weeks at delivery, foetal position, caesarean delivery, pregnancy with anaemia, and hypertensive disorders of pregnancy were independent predictors of severe PPH ($p < 0.05$). The predictive model demonstrated excellent calibration for the training and validation datasets, with the areas under the curve (AUC) for receiver operating characteristic (ROC) curves measuring 0.799 and 0.759, respectively. Clinical decision curve analysis (DCA) confirmed a significant threshold exceeding 0.9, signifying a substantial net benefit and model precision.

Conclusion Parity ≥ 2 times, abortion ≥ 2 times, *in vitro* fertilisation, gestational week at delivery, abnormal foetal position, caesarean delivery, pregnancy with anaemia, and hypertensive disorders of pregnancy are independent risk factors for severe PPH. The predictive model established in this study accurately predicts the occurrence of severe PPH and has significant value for clinical application.

Key words: postpartum haemorrhage; severe postpartum haemorrhage; risk factors; nomogram; predictive model

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Introduction

Postpartum haemorrhage (PPH) is a prevalent obstetric complication that significantly jeopardises the well-being of both mothers and newborns. It is recognised as the foremost contributor to maternal mortality on a global scale and is responsible for 27.1% of such fatalities (Higgins et al, 2019; Pasha et al, 2018). Postpartum haemorrhage accounts for 8% of maternal mortality in developed countries and 20% of maternal mortality in developing countries (Say et al, 2014). In recent years, the incidence of severe postpartum haemorrhage has been increasing, partly due to the lack of timely detection and treatment. Well-designed cohort studies and randomised clinical trials have been conducted to evaluate interventions for PPH to predict, prevent, and manage severe PPH. In medical practice, PPH is diagnosed when there is a blood loss of 500 mL or more within the first 24 hours post-vaginal delivery or 1000 mL or more following a caesarean section. The current definition for the severe form of PPH is a blood loss of 1000 mL or greater or a blood loss with associated signs or symptoms of hypovolemia that occurs within 24 hours of delivery, regardless of the mode of delivery (Escobar et al, 2022). The causes of PPH include uterine factors, placental factors, lacerations, uterine rupture and coagulation defects (Bienstock et al, 2021). It is worth considering how to intervene in advance according to the cause and whether the occurrence of severe PPH can be reduced. Few studies have attempted to analyse the risk factors for severe PPH, but their results are mixed (Kramer et al, 2013; Nyfløt et al, 2017). Some studies have divided the risk factors for PPH into low-, medium- and high-risk categories for obstetricians' consideration. To date, an effective evaluation system for the early identification of pregnant women at high risk of severe PPH is lacking (Neary et al, 2021).

To mitigate the risk of severe PPH and its associated negative impacts on maternal and infant health, it is crucial to investigate its risk factors to enable early detection and intervention. This approach is essential for diminishing the prevalence of PPH and the maternal mortality rate linked to severe PPH, as well as for improving perinatal outcomes. Thus, we conducted a retrospective review of the clinical records of 784 patients who experienced PPH post-delivery at our institution from December 2018 to December 2023. Using a case-control study design, we analysed the causes of PPH, explored the independent risk factors for PPH, and summarised the findings to provide a reasonable and effective basis for clinical management.

Methods

Case Data

Clinical data were obtained from 784 postpartum women who experienced PPH and delivered at the Second Affiliated Hospital of Anhui Medical University between December 2018 and December 2023. These cases were allocated to the training cohort. According to the diagnostic criteria for PPH and severe PPH (defined as blood loss of ≥ 500 mL for vaginal delivery and ≥ 1000 mL for caesarean

delivery within 24 hours postpartum), 550 patients with nonsevere PPH were classified into the control group, whereas 234 patients with severe PPH were categorized into the experimental group. Additionally, clinical data from 338 postpartum women meeting the same criteria during the same period were selected and assigned to the validation cohort. In this study, the requirement of obtaining informed consent was exempted from the Ethics Committee of the Second Affiliated Hospital of Anhui Medical University. As it retrospectively analyzed the clinical data of patients without administering any steps that would adversely affect their health or rights, provided that the patients' privacy is protected. This study was approved by the Ethics Committee of the Second Affiliated Hospital of Anhui Medical University (YX2024-106 (F1)).

Inclusion and Exclusion Criteria

The inclusion criteria were based on the 2016 guidelines for the prevention and management of PPH (Sentilhes et al, 2016). Within 24 hours after delivery, a blood loss of ≥ 500 mL associated with a vaginal delivery of ≥ 1000 mL linked to a caesarean delivery is defined as PPH. Severe PPH refers to ≥ 1000 mL of blood lost within 24 hours after delivery. All patients were ≥ 24 weeks old, and their clinical data were completely preserved. Patients with severe hepatorenal dysfunction, haematopoietic system diseases, and a history of malignant tumours were excluded.

Research Methods and Observational Indicators

This study employed a case-control research method. Clinical data, including causes of haemorrhage, general conditions (including age, gravidity, parity, conception method, gestational weeks at delivery, and mode of delivery), and pregnancy-related complications, were collected from the control group and the experimental group.

Statistical Analysis

Data analysis was conducted using SPSS Statistics for windows, Version 26.0 (IBM Corp., Armonk, NY, USA) and R software, Version 4.1.2 (R Core Team, Auckland, New Zealand), with statistical significance set at $p < 0.05$. Quantitative data not conforming to a normal distribution are expressed as median values and interquartile ranges [M (Q1, Q3)] and were compared between groups using the Mann-Whitney U test. Categorical variables are presented as counts and percentages [n (%)], with statistical evaluations conducted via Pearson's chi-square test or Fisher's exact test for nonordinal categorical data. When the sample size was small and the expected frequency was low (< 5), Fisher's exact test instead of the chi-square test was used, whereas the Mann-Whitney U test was used for ordinal data. Variables that demonstrated statistical significance in the univariate analysis were incorporated into a multivariate logistic regression model to determine their collective influence on severe PPH. The predictive model was graphically represented through a nomogram, and its efficacy was appraised via calibration plots and receiver operating characteristic (ROC) curves, detailing the areas under the curve (AUC) alongside the 95% confidence interval (CI). The model's precision

Table 1. Analysis of the causes of postpartum haemorrhage in the control and experimental groups.

Causes	Total (n = 784)	Groups	
		Control group (n = 550)	Experimental group (n = 234)
Uterine atony	696 (88.78)	504 (91.64)	192 (82.05)
Placental abruption	6 (0.77)	5 (0.91)	1 (0.43)
Soft birth canal laceration + haematoma formation	53 (6.76)	36 (6.55)	17 (7.26)
Placenta previa	4 (0.51)	2 (0.36)	2 (0.85)
Dangerous placenta previa	17 (2.17)	3 (0.55)	14 (5.98)
Uterine rupture	6 (0.77)	0 (0.00)	6 (2.56)
Uterine inversion	1 (0.13)	0 (0.00)	1 (0.43)
Amniotic fluid embolism	1 (0.13)	0 (0.00)	1 (0.43)

Data are presented as n (%).

was additionally gauged via decision curve analysis (DCA), with a threshold for significance at $\alpha = 0.05$.

Results

Analysis of the Causes of PPH

A total of 784 PPH patients were included in this study, with 234 patients with severe PPH classified into the experimental group and 550 patients with non-severe PPH classified into the control group. The causes of PPH in both groups are summarised in Table 1. Uterine atony was the most common cause of PPH, accounting for more than 80% of cases in both groups. Soft birth canal lacerations and haematoma formation were the second most common causes, with 7.26% and 6.55% in the experimental and control groups, respectively. Dangerous placenta previa was less common in the control group but accounted for 5.98% of the cases in the experimental group, making it the third most common cause. Rare causes, such as uterine rupture, uterine inversion, and amniotic fluid embolism, were observed only in the severe PPH group.

Univariate Analysis of the General Conditions and Clinical Characteristics of Patients with PPH

Certain significant differences were found between the two groups in terms of general conditions and clinical characteristics, as shown in Table 2, including age, gravidity, parity, number of abortions, gestational weeks at delivery, prothrombin time (PT) and activated partial thromboplastin time (APTT) levels at admission, *in vitro* fertilisation (IVF), foetal position, caesarean delivery, pregnancy with anaemia, and hypertensive disorders of pregnancy ($p < 0.05$). No statistically significant differences in prepregnancy body mass index, fibrinogen, D-dimer levels, forceps delivery, macrosomia, pregnancy with thrombocytopenia, gestational diabetes mellitus, pregnancy with thyroid dysfunction, or intrahepatic cholestasis of pregnancy ($p > 0.05$) were detected between the groups.

Table 2. Univariate analysis of the general and clinical characteristics of patients with postpartum haemorrhage in the control and experimental groups.

Variables	Groups		Statistic	p-value
	Control group (n = 550)	Experimental group (n = 234)		
Age, n (%)			$\chi^2 = 17.649$	<0.001
<35	489 (88.91)	181 (77.35)		
≥35	61 (11.09)	53 (22.65)		
Gravidity, n (%)			$\chi^2 = 23.025$	<0.001
1	259 (47.09)	67 (28.63)		
≥2	291 (52.91)	167 (71.37)		
Parity, n (%)			$\chi^2 = 26.349$	<0.001
1	333 (60.55)	95 (40.60)		
≥2	217 (39.45)	139 (59.40)		
Number of abortions, n (%)			Z = -4.844	<0.001
0	366 (66.55)	113 (48.29)		
1	123 (22.36)	76 (32.48)		
≥2	61 (11.09)	45 (19.23)		
Gestational weeks at delivery	38.86 (37.86, 39.43)	40.00 (37.89, 40.71)	Z = -6.801	<0.001
Prepregnancy BMI	21.26 (19.53, 23.14)	21.28 (19.47, 23.39)	Z = -0.254	0.8
PT	10.40 (10.00, 10.80)	10.60 (10.20, 11.00)	Z = -3.939	<0.001
APTT	26.60 (25.30, 28.20)	26.20 (24.80, 27.60)	Z = 2.282	0.022
Fibrinogen	4.12 (3.55, 4.81)	4.20 (3.62, 4.85)	Z = -0.529	0.597
D-Dimer	2.14 (1.43, 3.31)	2.24 (1.54, 3.39)	Z = -0.754	0.451
IVF, n (%)			$\chi^2 = 10.332$	0.001
No	538 (97.82)	218 (93.16)		
Yes	12 (2.18)	16 (6.84)		
Foetal position, n (%)			$\chi^2 = 16.030$	<0.001
Normal	542 (98.55)	218 (93.16)		
Abnormal	8 (1.45)	16 (6.84)		
Forceps delivery, n (%)			$\chi^2 = 3.223$	0.073
No	523 (95.09)	229 (97.86)		
Yes	27 (4.91)	5 (2.14)		
Caesarean delivery, n (%)			$\chi^2 = 103.951$	<0.001
No	450 (81.82)	107 (45.73)		
Yes	100 (18.18)	127 (54.27)		
Macrosomia, n (%)			$\chi^2 = 0.020$	0.886
No	507 (92.18)	215 (91.88)		
Yes	43 (7.82)	19 (8.12)		
Pregnancy with anaemia, n (%)			$\chi^2 = 8.650$	0.003
No	399 (72.55)	145 (61.97)		
Yes	151 (27.45)	89 (38.03)		
Pregnancy with thrombocytopenia, n (%)			$\chi^2 = 0.178$	0.673
No	519 (94.36)	219 (93.59)		
Yes	31 (5.64)	15 (6.41)		

Table 2. Continued.

Variables	Groups		Statistic	p-value
	Control group (n = 550)	Experimental group (n = 234)		
Gestational diabetes mellitus, n (%)			$\chi^2 = 0.253$	0.615
No	386 (70.18)	160 (68.38)		
Yes	164 (29.82)	74 (31.62)		
Hypertensive disorders of pregnancy, n (%)			$\chi^2 = 10.330$	0.001
No	531 (96.55)	213 (91.03)		
Yes	19 (3.45)	21 (8.97)		
Pregnancy with thyroid dysfunction, n (%)			$\chi^2 = 0.808$	0.369
No	496 (90.18)	206 (88.03)		
Yes	54 (9.82)	28 (11.97)		
Intrahepatic cholestasis of pregnancy, n (%)			$\chi^2 = 0.512$	0.474
No	536 (97.45)	230 (98.29)		
Yes	14 (2.55)	4 (1.71)		

Abbreviations: APTT, activated partial thromboplastin time; BMI, Body mass index; IVF, *in vitro* fertilisation; PT, prothrombin time.

Multivariate Logistic Regression Analysis of Severe PPH

Significant factors identified in univariate analysis were included in the multivariate logistic regression analysis. A parity ≥ 2 times, number of abortions ≥ 2 , IVF, abnormal foetal position, caesarean delivery, pregnancy with anaemia, hypertensive disorders of pregnancy, and gestational weeks at delivery were found to be independent risk factors for severe PPH ($p < 0.05$; Table 3).

Risk Prediction Nomogram for Severe PPH

The results of the multivariate regression analysis were translated into a nomogram to evaluate the relationships among the risk factors in the prediction model, as shown in Fig. 1. Intuitively, degrees of influence on the occurrence of severe PPH vary across the risk factors. The predicted value for patients with severe PPH can be calculated through functional conversion of the total score and the probability of severe PPH.

Evaluation of the Effectiveness of the Predictive Model

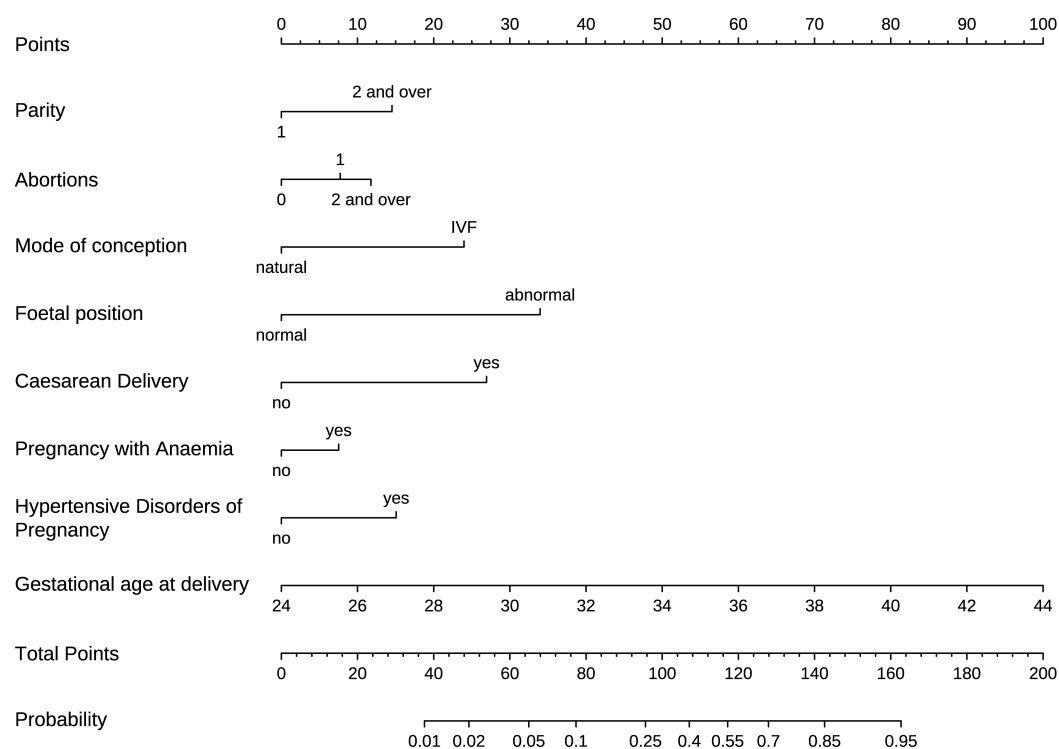
Calibration Curve of the Predictive Model

The predictive model's internal validity was confirmed through the bootstrap technique, which involved resampling the original dataset 1000 times. Subsequently, external validation was conducted using a separate validation cohort. The model's calibration plot, depicted in Fig. 2, is an indicator of its predictive accuracy, with closer proximity to the perfect curve signifying better performance. As observed

Table 3. Multivariate logistic regression analysis of severe postpartum haemorrhage.

	β	SE	Wald χ^2	p	OR	95% CI
Age (≥ 35)	0.401	0.264	2.309	0.129	1.493	0.890–2.503
Gravidity (≥ 2)	−0.061	0.372	0.027	0.869	0.941	0.453–1.951
Parity (≥ 2)	0.798	0.301	7.049	0.008	2.221	1.232–4.003
Abortions			4.446	0.108		
Number of abortions (1)	0.455	0.270	2.832	0.092	1.576	0.928–2.677
Number of abortions (≥ 2)	0.635	0.321	3.909	0.048	1.887	1.006–3.540
IVF	1.407	0.448	9.856	0.002	4.082	1.696–9.824
Foetal position	2.018	0.553	13.307	0.000	7.526	2.545–22.261
Caesarean delivery	1.618	0.187	74.596	0.000	5.041	3.492–7.277
Pregnancy with anaemia	0.433	0.195	4.935	0.026	1.541	1.052–2.257
Hypertensive disorders of pregnancy	0.871	0.390	4.985	0.026	2.388	1.112–5.128
Gestational weeks at delivery	0.304	0.056	29.084	0.000	1.355	1.213–1.513
PT	0.001	0.016	0.006	0.939	1.001	0.971–1.033
APTT	−0.025	0.037	0.452	0.501	0.976	0.908–1.048
Constant	−13.484	2.456	30.149	0.000	0.000	

Abbreviations: APTT, activated partial thromboplastin time; IVF, *in vitro* fertilisation; PT, prothrombin time.

**Fig. 1. Risk prediction nomogram for severe postpartum haemorrhage.**

in the illustration, the calibration plots for both the training and validation datasets exhibit good alignment with the ideal curve. Furthermore, the Hosmer–Lemeshow goodness-of-fit test yielded p -values of 0.183 for the training dataset and 0.286

for the validation dataset, both of which exceeded the threshold of 0.05, thereby suggesting that the model possesses satisfactory fit and predictive precision.

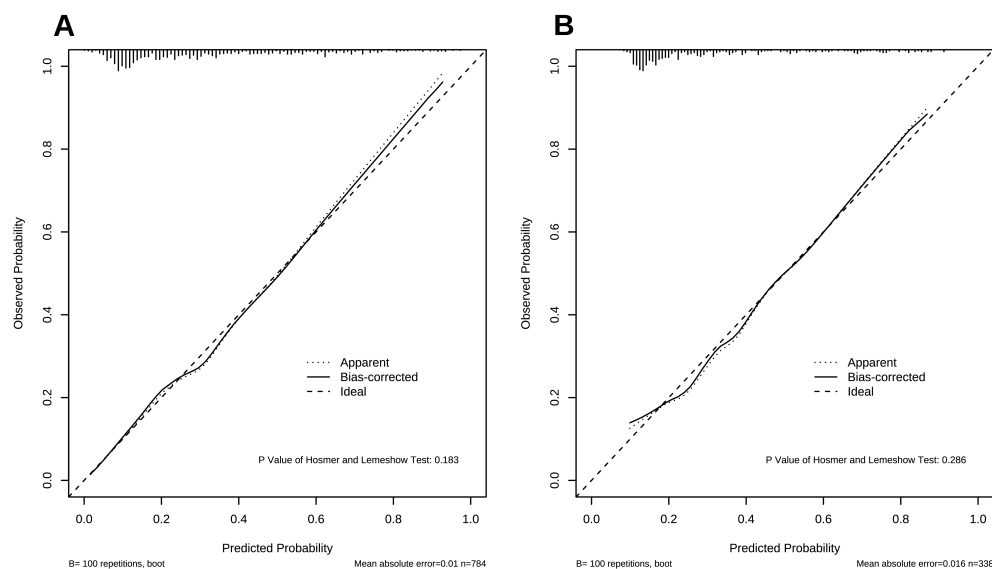


Fig. 2. Calibration curve of the predictive model. (A) Calibration curve of the training set. (B) Calibration curve of the testing set.

Receiver Operating Characteristic Curve of the Predictive Model

The ROC curve of the predictive model is shown in Fig. 3. The area under the ROC curve (AUC) ranges from 0.5 to 1. The closer the AUC is to 1, the better the prediction. The AUC values for the training and validation cohorts were 0.799 (95% CI: 0.764–0.833) and 0.759 (95% CI: 0.698–0.819), respectively, indicating good discrimination of the model. The DeLong test result was $D = 1.126$ ($p = 0.261$), suggesting no significant difference in the AUC between the training and validation cohorts. The model demonstrates good generalizability and high predictive power.

DCA Curve of the Predictive Model

The DCA curve of the predictive model is shown in Fig. 4. The DCA curve indicates the model has a high net benefit at a threshold above 0.9. These findings suggest that the model has good predictive accuracy.

Discussion

PPH is a severe obstetric complication featuring rapid progression. As of 2019, PPH accounted for 11.9% of maternal deaths in China. Therefore, reducing the incidence of severe PPH is crucial for lowering maternal mortality rates (Madar et al, 2021). Most maternal deaths from PPH are due to delays in diagnosis and treatment. The primary causes of PPH include uterine atony, birth canal trauma, placental factors, and coagulation disorders. These factors can coexist and inter-

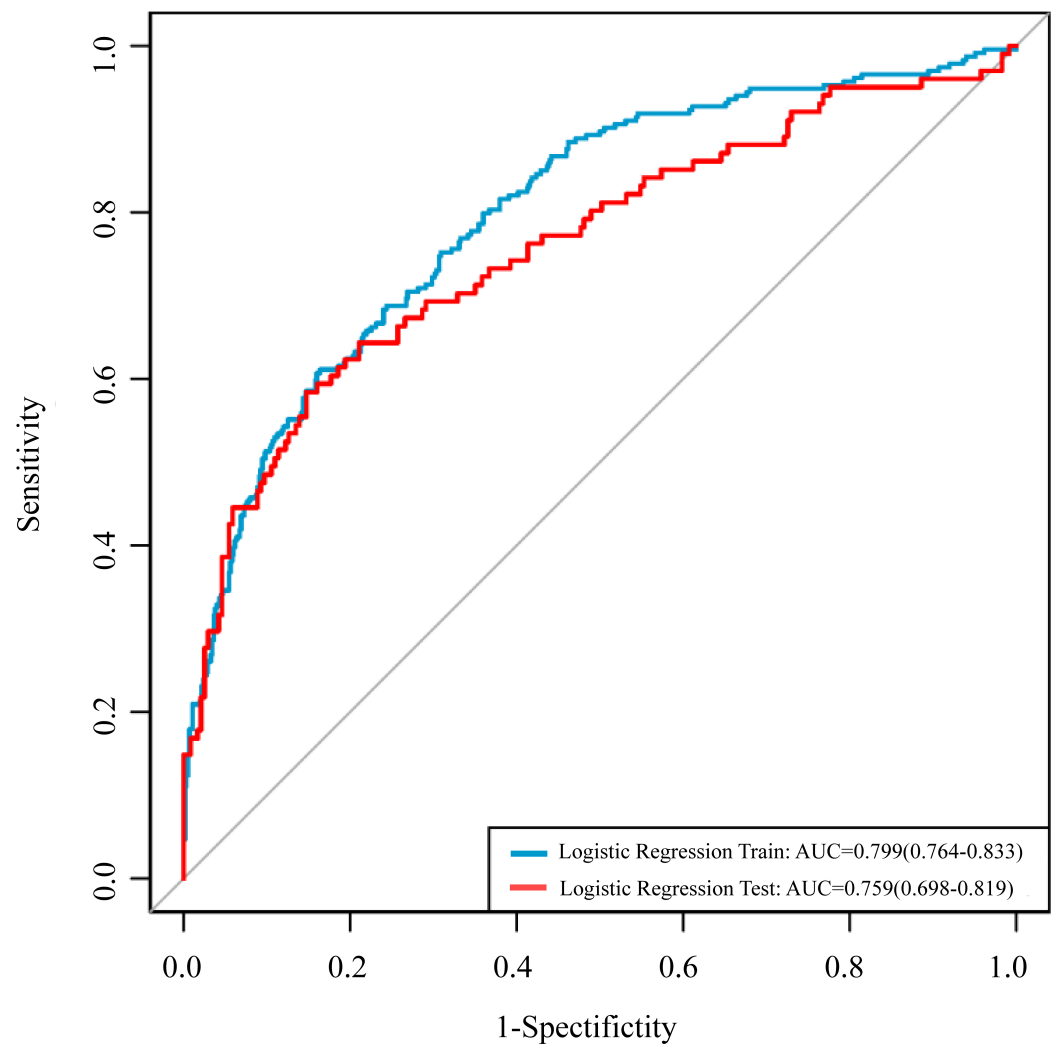


Fig. 3. Receiver operating characteristic (ROC) curves of the predictive model. (Blue curve) AUC of the training set. (Red curve) AUC of the testing set. Abbreviations: AUC, areas under the curve.

act, encompassing various causative and risk factors (Liu et al, 2021a). Enhancing prenatal care, treating underlying prenatal conditions, and implementing targeted perinatal preventive measures can effectively reduce maternal mortality and improve the quality of obstetric care. This study aimed to identify independent risk factors for severe PPH and develop a reliable clinical predictive model to provide effective guidelines for clinical practice.

Causes of the PPH

The main causes of PPH include uterine atony, soft birth canal lacerations, placental factors, and coagulation disorders. These causes can exist independently or coexist, influencing each other. Uterine atony is the most common cause of PPH and is responsible for 75% of cases in the United States (Marshall et al, 2017). In this study, uterine atony was the primary cause of PPH in the experimental and control groups, accounting for 91.64% and 82.05% of the cases, respectively. This is consistent with previous research (Patek and Friedman, 2023). Attention should be

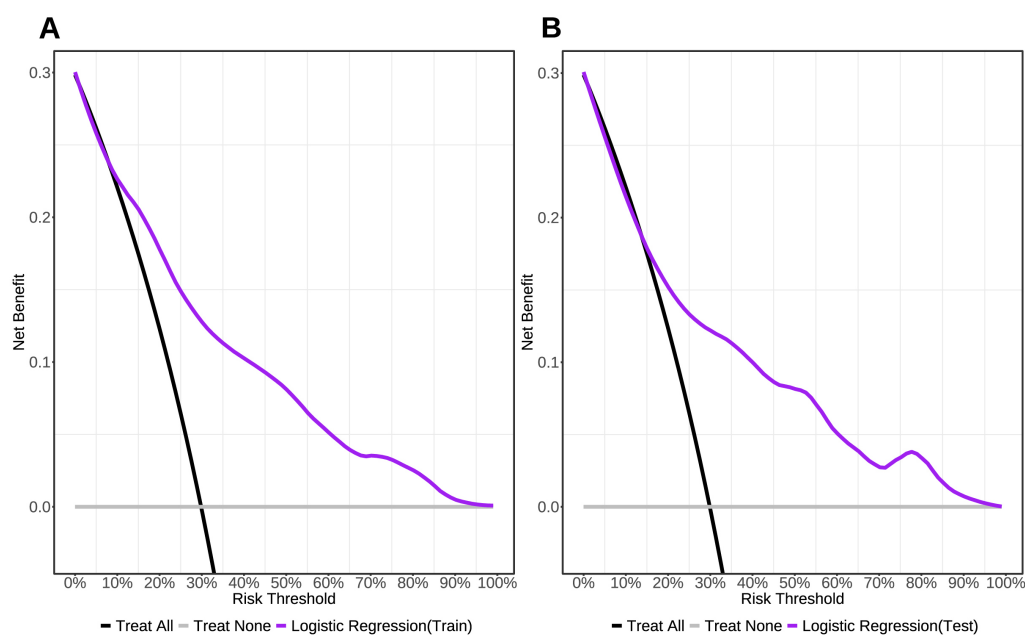


Fig. 4. DCA of the predictive model. (A) DCA curve of the training set. (B) DCA curve of the testing set.

given to diagnosing and treating uterine atony-related PPH. Soft birth canal lacerations primarily involve damage to the vagina and lower uterine segment. The major causes include pelvic narrowing, macrosomia, rapid labour progression, and abnormal foetal position, leading to obstructed labour. Excessive uterine cavity pressure can cause myometrial tears, resulting in significant postpartum bleeding (Ende et al, 2021). Improper use of oxytocin can lead to uterine rupture, and incorrect perineal protection techniques can cause perineal tears. Soft birth canal lacerations can lead to the formation of haematomas in the vaginal wall, lower uterine segment, and pelvic wall, even causing broad ligament haematomas, resulting in severe PPH. In this study, soft birth canal lacerations and haematoma formation were the second most common causes of PPH.

One case involved a full-term pregnant woman with vaginal delivery, soft birth canal lacerations, and incomplete uterine rupture, resulting in approximately 10,000 mL of blood loss and necessitating exploratory laparotomy and hysterectomy. This case underscores the importance of managing the second stage of labour, by carefully evaluating the foetal size, pelvic conditions and soft birth canal, and appropriately using oxytocin and induction drugs to prevent severe birth canal lacerations. Placenta previa, particularly in severe cases, poses a high risk for severe PPH. Placenta accreta is a major placental factor contributing to refractory PPH. The placental villi invade the myometrium due to inadequate decidualisation. After delivery, poor uterine contraction and open blood sinuses lead to massive, uncontrollable bleeding. In this study, dangerous placenta previa accounted for 5.98% of the haemorrhage cases in the experimental group, significantly higher than the 0.55% reported in the control group. Clinically, it is crucial to adhere strictly to cae-

sarean section indications, control caesarean section rates, and reduce unnecessary caesarean deliveries to lower the incidence of severe PPH.

Risk Factors for Severe PPH

A Norwegian study noted that risk factors for PPH include multiple pregnancies, surgical delivery, and chorioamniotic infection (Nyfløt et al, 2017). Lee et al (2018) reported that maternal age (≥ 35 years), prenatal haemorrhage, foetal position abnormality and placenta previa were risk factors for PPH. These findings are consistent with our findings; however, in the present study, we identified more risk factors for PPH. Our study revealed that parity (≥ 2), number of abortions (≥ 2), *in vitro* fertilisation, abnormal foetal position, caesarean delivery, gestational weeks at delivery, pregnancy with anaemia, and hypertensive disorders of pregnancy are independent risk factors for severe PPH. High parity can damage uterine muscle fibres, reduce their elasticity, and cause collagen fibre loss and degeneration, thereby impairing uterine contraction and leading to PPH. Women with multiple abortion histories may have undergone uterine procedures, resulting in endometrial damage, impaired decidua regeneration, abnormal placental attachment, and placenta accreta. Additionally, a history of uterine procedures increases the risk of endometrial infection, leading to poor or absent decidual development, insufficient placental blood supply, an enlarged placental area, and placenta previa, all of which contribute to an increased risk of PPH (Carusi, 2018). Therefore, it is essential to enhance family planning education for women of childbearing age, guide appropriate contraceptive use, and prevent unintended pregnancies and abortions to reduce endometrial damage and subsequent PPH. Although multivariate regression analysis in this study did not identify advanced maternal age as an independent risk factor for severe PPH, univariate analysis revealed that the proportion of patients aged ≥ 35 years was significantly greater in the severe PPH group than in the control group. With the implementation of pro-natalist policies and advancements in assisted reproductive technologies, a growing number of older and infertile patients would benefit from these technologies (Mavrides et al, 2017). Study has shown that assisted reproductive technology, such as *in vitro* fertilisation and embryo transfer, are independent risk factors for PPH, possibly due to endometrial asynchrony, low implantation of embryos, and abnormal placental attachment (Tang et al, 2023). In this study, the coefficient for IVF was 1.407, and the OR value was 4.082, indicating that patients who conceived through assisted reproductive technology had a 4.082-fold increased risk of severe PPH compared with those with natural pregnancies. Therefore, high-risk pregnant women treated with assisted reproductive technology require increased clinical attention, more frequent prenatal visits, early identification of other risk factors for severe PPH, and timely interventions. Abnormal foetal positions are often associated with uterine developmental anomalies (such as a septate uterus and bicornuate uterus) or abnormal placental positions (such as placenta previa). These uterine and placental anomalies frequently lead to abnormal uterine contraction polarity, reducing the sensitivity of oxytocin and its receptors, which in turn causes uterine atony and PPH. Additionally, these patients often face difficulties during foetal delivery, both surgically and vaginally, which

increases intraoperative and postoperative blood loss. Our multivariate regression analysis indicated that gestational weeks at delivery is an independent risk factor for PPH, with a coefficient of 0.304 and an OR of 1.355. [Butwick et al \(2021\)](#)'s study revealed that women who delivered at 41–42 weeks of gestation had the highest risk of PPH. Other international studies suggest that delivery at less than 26 weeks of gestation is closely associated with severe PPH ([Blanc et al, 2019](#); [Traoré et al, 2018](#)). These findings imply that when maternal and foetal safety allows, extending the gestational period appropriately reduces PPH incidence. Regrettably, this study did not further categorise gestational age groups to provide objective guidelines for optimal delivery timing, which warrants further research.

Caesarean section is a recognised risk factor for severe PPH ([Butwick et al, 2017](#); [Fukami et al, 2019](#); [Li et al, 2021](#)). A study by [Ashwal et al \(2021\)](#) revealed that the incidence of PPH during emergency caesarean section and elective caesarean section was 6.75% and 4.84%, respectively ($p = 0.007$), suggesting that emergency caesarean section is an independent risk factor for severe PPH. This finding also suggests that we can further study the factors related to emergency caesarean section to identify unknown risk factors for severe PPH. Study has shown that the probability of PPH in subsequent pregnancies with a scarred uterus and caesarean delivery is 14.8% ([Girault et al, 2018](#)). Caesarean sections can damage the endometrium and reduce the repair capacity of the myometrium. Additionally, the elasticity of scar tissue is poor, making it difficult for damaged blood vessels to close and increasing the risk of incision tears, ultimately affecting uterine contraction and leading to uterine atony and PPH. However, some researchers argue that caesarean section can be a protective factor against PPH. Compared with vaginal delivery, caesarean sections allow for rapid haemostasis through uterine artery ligation, B-Lynch sutures, and other techniques. Alternatively, vaginal delivery relies on medications, uterine tamponade, and interventional treatments, which may not achieve rapid haemostasis as effectively as caesarean section (does) ([Liu et al, 2021b](#)). In this study, out of the 234 patients in the experimental group, 127 patients (54.27%) delivered via caesarean section, whereas among the 550 patients in the control group, only 100 patients (18.18%) who delivered via caesarean section. The univariate analysis revealed a statistically significant difference between the two groups ($p < 0.001$). Multivariate regression analysis revealed a coefficient for caesarean section of 1.618 and an OR of 5.041, indicating that the risk of severe PPH is 5.041 times greater for caesarean deliveries than for vaginal deliveries. Therefore, clinicians must master caesarean section techniques, improve uterine incision suturing, and enhance postoperative management. Reducing the caesarean section rate is an important measure for lowering the incidence of PPH.

Anaemia is a common complication among pregnant women, and those with anaemia have reduced tolerance to blood loss, which increases their susceptibility to coagulation disorders and PPH. Study indicates that antenatal anaemia decreases oxygen delivery to uterine smooth muscle, increasing the incidence of uterine atony compared to nonanaemic patients ([Patek and Friedman, 2023](#)). Multiple studies investigating the relationship between antenatal anaemia and PPH have shown that antenatal anaemia is an independent risk factor for PPH ([Koh et al, 2018](#); [Omo-](#)

tayo et al, 2021). Our findings are consistent with these results. Therefore, in clinical practice, it is crucial to actively identify and correct the causes of antenatal anaemia in pregnant women to improve their tolerance to blood loss and reduce the incidence of PPH. Hypertensive disorders of pregnancy primarily involve impaired trophoblast invasion, uterine spiral artery remodelling, and inadequate placental perfusion. Severe cases can lead to placental abruption, liver and kidney dysfunction, and coagulation disorders, resulting in significant PPH that threatens maternal and foetal health (Turbeville and Sasser, 2020). Patients with hypertensive disorders during pregnancy have increased vascular fragility and ischaemic oedema of the uterine myometrium, affecting its ability to contract normally. Additionally, treatment for hypertensive disorders often involves antispasmodics, sedatives, and antihypertensive medications, which can reduce the excitability of uterine smooth muscle, inducing uterine atony and further leading to PPH. Study has confirmed that hypertensive disorders during pregnancy significantly increase the incidence of PPH (Durmaz and Komurcu, 2018). Our research indicates that such disorders during pregnancy are independent risk factors for severe PPH. Clinically, it is essential to manage hypertensive disorders and preeclampsia effectively to reduce severe complications, ensure maternal and foetal safety, and improve pregnancy outcomes.

Risk Prediction Model for Severe PPH

A nomogram-based prognostic model was constructed based on the independent predictors for severe PPH. The validation outcomes revealed that the AUC for the training cohort was 0.799, with a 95% CI ranging from 0.764 to 0.833, whereas the AUC for the validation cohort was 0.759, with a 95% CI ranging from 0.698 to 0.819. The calibration curve shows that the model fits well. DCA demonstrated a substantial net benefit at thresholds surpassing 0.9. Collectively, these results indicate that the model is highly accurate in forecasting the risk of severe PPH. Nevertheless, since the current research employed a retrospective observational study design and a small sample size, we consider there is a need to conduct randomised controlled studies with larger samples to provide more clinical evidence for corroborating our findings.

Conclusion

In summary, uterine atony is the primary cause of PPH and severe PPH. Early and timely use of oxytocin postdelivery can reduce the incidence of PPH. Additionally, it is critical to manage and treat placenta previa in a proactive manner, control the caesarean section rate, and avoid unnecessary caesarean deliveries. Also, providing prepregnancy education and relevant prepregnancy examinations is vital. Clinicians are required to be familiar with the risk factors for PPH and severe PPH, enabling early identification, intervention, and prevention, thereby reducing the incidence of severe PPH and improving maternal and foetal outcomes.

Key Points

- Uterine atony remains the main cause of postpartum haemorrhage among women who deliver through both vaginal and caesarean section. Therefore, attention must be given to diagnosing and treating uterine atony-related postpartum haemorrhage (PPH).
- Parity (≥ 2 times) and the number of abortions (≥ 2 times) are independent risk factors for severe PPH. Therefore, it is essential to enhance family planning education for women of childbearing age, guide appropriate contraceptive use, and prevent unintended pregnancies and abortions to reduce endometrial damage and subsequent PPH.
- *In vitro* fertilisation, abnormal foetal position, caesarean delivery, gestational weeks at delivery, pregnancy with anaemia, and hypertensive disorders of pregnancy are also independent risk factors for severe PPH. Reducing the caesarean section rate, managing hypertensive disorders and strengthening pregnancy care are crucial to reduce severe complications and improve pregnancy outcomes.
- Early identification, intervention and prevention are needed for controlling the rising incidence of severe PPH.

Availability of Data and Materials

The datasets analyzed during the current study are available from the corresponding author on reasonable request.

Author Contributions

JW and PH designed the research study. JW and YY performed the research. YZ, XQW and YHL provided help to collect the data. JW analyzed the data and drafted the manuscript. All authors contributed to editorial important changes in the manuscript. All authors read and approved the final manuscript. All authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

This study was approved by the Second Affiliated Hospital of Anhui Medical University ethics committee, YX2024-106 (F1) and all patients were exempt from informed consent. The study strictly adheres to the principles of the Declaration of Helsinki.

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Conflict of Interest

The authors declare no conflict of interest.

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