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Original Research

Postpartum hemorrhage is associated with neonatal body weight, pre-pregnancy body mass index, and maternal weight gain

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Summary

Purpose of Investigation: The purpose is to identify factors related to postpartum hemorrhage (PPH) that are evaluated during regular prenatal check-ups. *Materials and Methods*: Obstetric and neonatal data were collected retrospectively for 1,922 women with singleton pregnancies who delivered vaginally. *Results*: Overweight women exhibited more severe PPH cases compared to normal PPH ($p = 0.04$). Of pre-pregnancy body mass index (BMI), gestational weight gain (GWG), gestational week of delivery, and neonatal body weight, neonatal body weight showed a significantly elevated risk for severe PPH (adjusted OR = 1.156, $p < 0.001$). Infant body weight was correlated positively with pre-pregnancy BMI, GWG, and gestational week of delivery (r = 0.194, r = 0.189 and r = 0.364, respectively). Pre-pregnancy BMI, GWG, and gestational week of delivery were associated with neonatal body weight (adjusted B = 0.169, 1.206 and 1.181, respectively; $p < 0.001$). *Conclusion*: Management of maternal body weight induces a safe delivery through controlling neonatal body weight. *Content*: Severe postpartum hemorrhage is associated with neonatal body weight which is influenced by maternal body mass index and weight gain.

Key words: Postpartum hemorrhage; Neonatal body weight; Body mass index; Body weight gain.

Introduction

Postpartum hemorrhage (PPH) is the leading cause of maternal mortality globally [1]. Several recent publications have noted an increasing trend in incidence over time [2]. PPH is generally defined as blood loss in excess of 500 to 1,000 mL, and is classified as early or late onset [3]. In 2017, the American College of Obstetricians and Gynecologists revised their definition of PPH from the classic one described above to: cumulative blood loss $\geq$ 1,000 mL or bleeding associated with signs/symptoms of hypovolemia within 24 hours of the birth process regardless of delivery route [4]. Early PPH is defined as bleeding that occurs within 24 hours from birth [3]. Secondary PPH is generally defined as any significant uterine bleeding occurring between 24 hours and 12 weeks postpartum and the most common causes are retained products of conception, subinvolution of the placental bed and infection [4, 5]. It is important to evaluate PPH, in particular early PPH, in cases with predictable perinatal complications. However, predicting the risk of PPH is difficult in healthy pregnant women because four national guidelines announce that most women who experience PPH do not have any known risk factors [3]. Furthermore, because it is impossible to measure the amount of blood loss while caring for the pregnant woman in parturition, estimation of the risk of PPH before delivery is critical in clinical settings.

Risk factors for PPH include complications of pregnancy such as an atonic uterus due to fetal macrosomia, multiple pregnancy and polyhydramnios, antepartum hemorrhage, chorio-amnionitis, coagulation disorders, adenomyosis, induction of labor, obesity, hypertensive disorders of pregnancy, previous caesarean section delivery, past history of PPH, primigravity, and prolonged rupture of membranes and/or labor [6-8]. However, it is not clear which factors contribute to PPH in pregnant woman without obstetrical complications. 25% of maternal mortality is attributed to PPH and that 20% of people who develop PPH have no known risk factors. If prenatal check-ups for normal pregnant women can predict excessive blood loss, health outcomes for the mother will be improved.

The aim of this study was to determine whether maternal physique and infant body weight, both of which are easily assessed at prenatal check-ups, are associated with an increased risk for PPH.

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Table 1. — *Characteristics of the participants according to postpartum hemorrhage.*

	Normal PPH (< 1,000 mL)	Severe PPH (≥ 1,000 mL)	*p* value
Number (%)	1,796 (93.4%)	126 (6.6%)	
Age			0.89
< 20 years old	59 (3.3%)	3 (2.4%)	0.89
20-29 years old	723 (40.3%)	53 (42.4%)	0.89
30-39 years old	952 (53.1%)	64 (51.2%)	0.89
≥ 40 years old	60 (3.3%)	5 (4.0%)	0.89
Parity			0.52
0	898 (50.0%)	67 (53.2%)	0.52
1 and more	898 (50.0%)	59 (46.8%)	0.52
Pre-pregnancy BMI			0.52
Underweight	330 (19.3%)	26 (21.3%)	0.64
Normal	1,236 (72.4%)	79 (64.8%)	0.08
Overweight	141 (8.3%)	17 (13.9%)	0.04
Gestational diabetes mellitus			0.67
Presence	22 (1.2%)	2 (1.6%)	0.67
Absence	1,771 (98.8)	124 (98.4%)	0.67
Hypertensive disorders of pregnancy			0.36
Presence	17 (0.9%)	2 (1.6%)	0.36
Absence	1,776 (99.1%)	124 (98.4%)	0.36
Placenta Accreta			0.13
Presence	1 (0.1%)	1 (0.8%)	0.13
Absence	1,795 (99.9%)	125 (99.2%)	0.13

PPH, postpartum hemorrhage; BMI, body mass index.

Materials and Methods

Study design

An observational retrospective single-center cross-sectional study on data collected from the perinatal database of the National Hospital Organization Kyoto Medical Center was conducted. A total of 2,467 women with full term singleton pregnancies from January 2007 to December 2011 were enrolled in this study. Early PPH refers to excessive bleeding that occurs within 24 hours of delivery [3]. We selected full term singleton pregnancies that resulted in vaginal delivery, categorized the patients into two groups based on the amount of early PPH (≥ 1,000 mL or < 1,000 mL), and investigated the influence of clinical factors including maternal age, parity, pre-pregnancy body mass index (BMI), gestational weight gain (GWG), gestational week of delivery, and infant body weight on PPH. Based on the criteria of the American College of Obstetricians and Gynecologists, we designated ≥ 1,000 mL and < 1,000 mL cases as severe PPH and normal PPH groups, respectively. Women were classified into normal, overweight and obese groups according to conventional World Health Organization (WHO) BMI criteria; normal (18.5-24.9 kg/m^2), overweight (25.0-29.9 kg/m^2), and obese (≥ 30.0 kg/m^2) [9]. GWG was classified with the diagnostic criteria recommended by the Japan Society for the Study of Obesity; < 12.0 kg for underweight and normal weight women (BMI < 25.0 kg/m^2), and < 5 kg for overweight women (BMI ≥ 25.0 kg/m^2).

Statistical analyses

The χ^2 test was used for categorical variables, and Student's *t*-test was used to compare the means of continuous variables. Using binomial logistic regression analysis, we estimated the risk of severe PPH (1,000 mL and more than 1,000 mL) with pre-pregnancy BMI, GWG, gestational week of delivery, and neonatal body weight. Multiple regression analysis was used to evaluate the effect of pre-pregnancy BMI, GWG, and gestational week of delivery on neonatal body weight. Partial regression coefficient (B) of infant body weight was calculated for every additional 100 g. SPSS (version 23.0, IBM Corp., Armonk, NY, USA) was used for all analysis. $p < 0.05$ was considered significant.

Results

Characteristics of the participants

Of the 2,467 women with full term singleton pregnancies, vaginal delivery was identified in 1,931 women. Of the 1,931 cases, nine were excluded from the study because of insufficient data. There were 1,796 women (93.4%) with normal PPH (< 1,000 mL) and 126 women (6.6%) with severe PPH (≥ 1,000 mL) (Table 1).

There were no significant differences in maternal age or parity between the two groups ($p = 0.89$ and 0.52, respectively; Table 1). Although there were no significant differences between normal PPH and severe PPH in underweight and normal weight women, overweight women exhibited

Table 2. — *Effect of pre-pregnancy BMI, gestational weight gain, gestational week of delivery, and neonatal body weight on severe PPH.*

	Crude							Adjusted		
	B	S.E.	Wald	R^2	Odds ratio	95% CI	*p* value	Odds ratio	95% CI	*p* value
Pre-pregnancy BMI (kg/m2)	0.043	0.03	2.532	0.003	1.044	0.990-1.102	0.11			0.68
Excessive GWG	0.381	0.20	3.991	0.006	1.464	1.007-2.127	0.046			0.51
Gestational week of delivery	0.382	0.09	18.427	0.026	1.465	1.231-1.745	< 0.001			0.06
Neonatal body weight (every 100 g)	0.159	0.24	44.452	0.059	1.173	1.119-1.229	< 0.001	1.156	1.101-1.212	< 0.001

BMI, body mass index; GWG, gestational weight gain; R^2, Negelkerke R^2.

Table 3. — *Effect of pre-pregnancy BMI, gestational weight gain, and gestational week of delivery on neonatal body weight.*

	Crude				Adjusted		
	B	R^2	95% CI	*p* value	B	95% CI	*p* value
Pre-pregnancy BMI (kg/m^2)	0.206	0.031	0.153-0.259	< 0.001	0.169	0.120-0.218	< 0.001
Excessive GWG	1.634	0.045	1.287-1.980	< 0.001	1.206	0.883-1.530	< 0.001
Gestational week of delivery	1.276	0.143	1.137-1.416	< 0.001	1.181	1.041-1.320	< 0.001

BMI, body mass index; GWG, gestational weight gain; R^2, Negelkerke R^2.

significantly more cases of severe PPH compared to normal PPH (13.9% and 8.3%, respectively, $p = 0.04$; Table 1). The number of pregnant women with gestational diabetes mellitus or hypertensive disorders of pregnancy showed no significant differences ($p = 0.67$ and 0.36, respectively; Table 1). One case in each group was diagnosed with placenta accreta ($p = 0.13$). There were no women diagnosed with polyhydramnios, amniotic fluid embolism, or placenta previa. There were no differences in history of cesarean section between severe PPH and normal PPH groups.

Contribution of large neonatal body weight to PPH

To identify the factors affecting PPH, logistic regression analysis was performed using pre-pregnancy BMI, GWG, gestational week of delivery, and neonatal body weight. The crude odds ratio (OR) of pre-pregnancy BMI for severe PPH was 1.044 and was not statistically significant (95% CI = 0.990-1.102, $p = 0.110$; Table 2); however, crude OR of GWG, gestational week of delivery, and neonatal body weight were significantly higher for severe PPH (crude OR = 1.464, 95% CI = 1.007-2.127, $p = 0.046$; crude OR = 1.465, 95% CI = 1.231-1.745, $p < 0.001$; crude OR = 1.173, 95% CI = 1.119-1.229, $p < 0.001$; Table 2). In adjusted OR, although pre-pregnancy BMI, GWG, and gestational week of delivery did not show statistically significant risks for severe PPH, only neonatal body weight exhibited a significantly higher risk (adjusted OR = 1.156, 95% CI = 1.101-1.212, $p < 0.001$; Table 2).

Factors affecting the neonatal body weight

Since neonatal body weight was a significant factor, we then assessed possible factors that affect infant body weight to control clinically for these factors during maternal examination check-up system. Spearman rank correlation analyses indicated significantly positive correlations between pre-pregnancy BMI, GWG, and gestational week of delivery and infant body weight (r = 0.194, r = 0.189 and r = 0.364, respectively; $p < 0.001$; Figure 1A-C). Multiple regression analysis showed that crude B values of pre-pregnancy BMI, GWG, and gestational week of delivery to neonatal body weight were 0.206, 1.634, and 1.276, respectively (95% CI = 0.153-0.259, 1.287-1.980 and 1.137-1.416, respectively; $p < 0.001$; Table 3). Adjusted B values of pre-pregnancy BMI, GWG, and gestational week of delivery were also significant risk factors for higher neonatal body weight (adjusted B = 0.169, 1.206 and 1.181, respectively; 95% CI = 0.120-0.218, 0.883-1.530 and 1.041-1.320, respectively; $p < 0.001$; Table 3). These findings indicate that pre-pregnancy BMI, gestational GWG, and gestational week of delivery all influence neonatal body weight.

Discussion

The WHO defines PPH as blood loss of 500 mL or more within 24 hours after birth [10]. WHO classifies PPH as moderate when the blood loss is between 500 mL and 1,000 mL, and severe when the blood loss is over 1,000 mL [11].

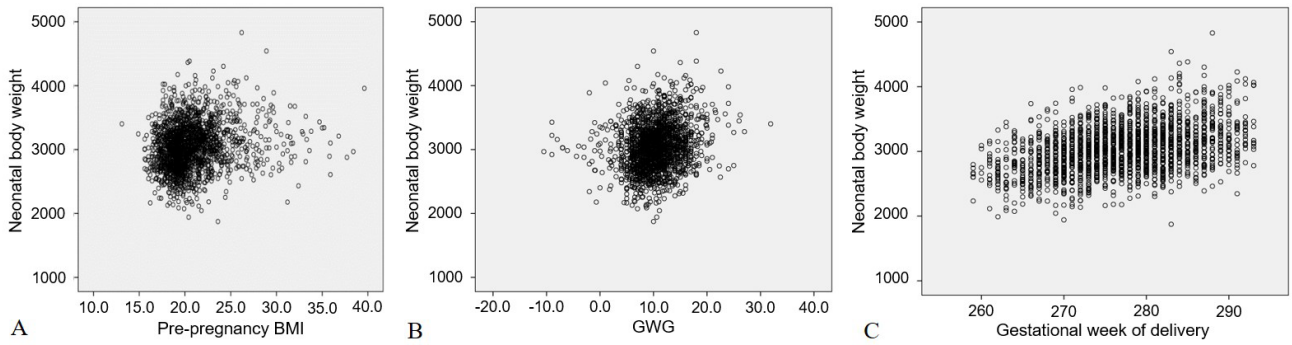

Figure 1. — Spearman rank correlation analyses indicated significantly positive correlations between pre-pregnancy BMI, GWG, and gestational week of delivery and infant body weight. Infant body weight was correlated positively to pre-pregnancy BMI (r = 0.194, a), GWG (r = 0.189, b), and gestational week of delivery (r = 0.364, c) ($p < 0.001$). BMI, body mass index; GWG, gestational weight gain.

However, we must assume that we are measuring less than half the amount of actual blood loss because of no preferred method to estimate blood loss [3]. In this study, severe PPH was defined as over 1,000 mL blood loss, because it may lead to a clinically lethal status. The prevalence of PPH worldwide is approximately 6% of all births, mostly in low-income settings [12]. However, it may vary between 5% and 18%, even within the same country. This study also showed that the prevalence of PPH is approximately 6.6% for all singleton deliveries, which is comparable to previous studies.

The primary etiologies of PPH have been shown to be uterine atony, placenta retention, cervico-vaginal lesions, polyhydramnios, placenta previa, placental accreta, placental abruption, and hypertensive disorders of pregnancy (HDP) [6-8, 13]. In this study, there were no significant differences between severe PPH and normal PPH groups in gestational diabetes mellitus, HDP, or placental accreta, indicating that these factors did not affect the results of this study. Although such obstetrical complications have been reported as risks for PPH, predictors for PPH in pregnant women without obstetrical complications remain unclear. It has been reported that fetal macrosomia causes an atonic uterus [14]. Collectively, our results suggest that neonatal body weight contributes to the amount of PPH possibly through an atonic uterus. The factors associated with PPH have been identified as ethnicity (Pacific island and Asian), past history of PPH, high multiparity, pre-pregnancy BMI, labor induction, episiotomy, the absence of managed placental delivery, an interval of more than 30 minutes between birth and placental delivery, and macrosomia [7, 13, 15]. Among these factors, however, it is uncertain which is the most influential factor on severe PPH. The risk of PPH increases rapidly with increasing BMI [6, 13]. After adjustment for covariates, obese women showed a two-fold increase for severe PPH compared to women with normal BMI [13, 16]. Even a modest decrease in pre-pregnancy BMI can reduce this risk. In this study, overweight women tended to be in the severe PPH group, which is similar to previous studies. However, the mechanism of how BMI influences PPH is unclear. Previous studies have suggested that the increase in risk of PPH in obese women can largely be explained by a concurrent increased caesarean section rate [17]. Nevertheless, some studies have shown that obesity is related to PPH regardless of cesarean section. This study also showed that obesity is a risk factor for PPH regardless of cesarean section, because only women who delivered vaginally were assessed. Overweight or obese women still showed an increased risk for severe PPH. Women with excessive GWG had an increased likelihood of PPH compared to women with normal weight gain [18]. Although the R^2 value of neonatal body weight is a little small ($R^2 = 0.059$), the p value showed statistical significant due to a large number of the cases. Wald test also indicated that neonatal body weight is the strongest contributor of severe PPH. Our results indicate that neonatal body weight carries the highest risk for severe PPH among GWG, gestational week of delivery, and neonatal body weight. These findings suggest that increased pre-pregnancy BMI may affect the risk of severe PPH through increased neonatal body weight.

Risk factors for increased neonatal body weight that can be evaluated in the prenatal check-up for pregnant women were also assessed. The known risk factors for macrosomia are male sex, high parity, maternal age and height, post-term pregnancy, obesity, large GWG, and pre-gestational and gestational diabetes [19]. In a prospective observational study of Han Chinese women with a singleton birth, the variables that showed a strong correlation with birth weight were placental weight, GWG, pre-pregnancy BMI, mid-pregnancy BMI, maternal fat free mass, and fat mass during the first and second trimesters [20]. GWG has been associated with shoulder dystocia and macrosomia [21]. Moreover, previous studies have reported that GWG above the recommendations and maternal obesity increase the risk of macrosomia [7, 22, 23]. This study also revealed that large neonatal body weight, which is a risk factor for PPH, is independently related to pre-pregnancy BMI, large GWG, and gestational week of delivery. Another study reported a 40% increase in risk for PPH with every 500 g increase

in birthweight in full-term infants [13]. Our findings indicate that infant body weight is independently associated with severe PPH, and that other factors including maternal BMI, GWG, and gestational week of delivery may also be related to severe PPH through infant body weight.

The prevalence of macrosomia in developed countries is between 5% and 20% [24]; however, an increase of 15-25% has been reported in the past two to three decades, mainly driven by an increase in maternal obesity and diabetes [24]. As the prevalence of diabetes and obesity in women of reproductive age increases in developing countries [25], a parallel increase in macrosomia may be expected. Our findings suggest that pre-pregnancy health care may decrease the incidence of severe PPH.

The limitations of this study include an inaccurate amount of blood loss and the absence of some variables in the dataset that may be important risk factors. Although the estimation of blood loss is difficult, over 1,000 mL for severe PPH is reasonable in a clinical setting. Another weakness is the limited number of women. In this study, there was no significant difference among HDP and duration of parturition (data not shown). The reason may be the small number of cases with obstetrical complications. Further research is necessary to evaluate PPH according to obstetrical complications. Another limitation is the single-center study. Racial differences were not compared in this study.

Conclusions

In summary, large neonatal body weight is a risk factor of severe PPH, especially in obese women. Pre-pregnancy BMI, inappropriate GWG, and gestational week of delivery are independent contributors to neonatal body weight. These findings indicate that prophylactic intervention of maternal body weight before and during pregnancy plays a possible role in a safe delivery through controlling neonatal body weight.

Ethics Approval and Consent to Participate

The study was approved by the ethics committee of the National Hospital Organization Kyoto Medical Center (approval number: 16-090). The requirement for written informed consent was waived due to the retrospective and anonymized nature of the analyses. This study was announced for the patients to give the opportunity to claim the refusal.

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Conflict of Interest

The authors declare no conflict of interest.

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