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Authors

Zullo, Fabrizio

Wiley, Rachel L

Ghose, Ipsita

et al.

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Original Article

Postpartum Hemorrhagic Morbidities with Livebirth versus Stillbirth

Fabrizio Zullo[1,2], Rachel L. Wiley[3], Ipsita Ghose[4], Giuseppe Rizzo[1], Antonella Giancotti[1], Hector Mendez-Figueroa[3], Daniele Di Mascio[1], Suneet P. Chauhan[4]

Affiliation addresses are listed at the end of the article.

ABSTRACT

Objective ACOG publications on stillbirth or postpartum hemorrhage (PPH) do not consider stillbirth as a risk factor for postpartum hemorrhagic morbidity. This study aimed to ascertain the likelihood of composite maternal hemorrhagic outcome (CMHO) among individuals who delivered vaginally with livebirth versus a stillbirth.

Study Design This was a retrospective cohort study of all parturients greater than 20 weeks gestation who delivered vaginally at a single level IV site within 24 months. Demographic differences and baseline PPH risks were analyzed. CMHO included any of the following: estimated blood loss ≥1,000 mL, use of uterotonics (beyond prophylactic oxytocin), Bakri balloon, surgical management of PPH, blood transfusion, hysterectomy, venous thromboembolism (VTE), admission to the intensive care unit (ICU), or maternal death. Statistical analysis included chi-squared, Kruskal-Wallis, and Poisson regression with robust error variance for risk ratios, adjusting for gestational age (GA), bleeding on admission, chorioamnionitis, and prior uterine surgery.

Results Of 8,623 consecutive vaginal births ≥20 weeks gestation, 89 (1.9%) were stillbirths. Maternal age, marital status, GA at delivery, and PPH risk stratification at admission differed significantly. Bleeding at admission ($p < 0.001$), prior uterine surgery ($p < 0.001$), magnesium sulfate use ($p = 0.006$), chorioamnionitis ($p < 0.001$), platelet count <100 ($p = 0.001$), platelet count <50 ($p < 0.001$), and retained products of conception ($p < 0.001$) were different in the two groups. CMHO was significantly higher with a stillbirth delivery (32.6 vs. 16.8%; aRR: 1.56, 95% CI: 1.01–2.46). After adjustment, the components of the CMHO that differed significantly were estimated blood loss ≥1,000 mL and ICU admission. Tamponade, surgical intervention, VTE, hysterectomy, and maternal death did not differ between the two groups.

Conclusion Pregnancies with stillbirth, compared with livebirth, had an increased risk of hemorrhagic related morbidity. In addition to being useful in shared decision-making, our results can be nidus for intervention trials to decrease the hemorrhagic morbidity associated with stillbirth.

Keywords blood loss, hysterectomy, intensive care unit, maternal mortality, tamponade, transfusion, uterotonic use

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Correspondence Suneet P. Chauhan, MD, Hon DSc, Delaware Center of Maternal-Fetal Medicine of ChristianaCare, 1 Centurian Drive Suite 312, Newark, Delaware 19713, United States, Email: schauhan@dcmfm.com

Introduction

Defined as delivery of a fetus at 20 weeks or more, without any breathing, heart rate, pulsation in the umbilical cord, or definite movement of the voluntary muscle, stillbirth occurs in approximately 1 in 160 births.[1,2] Although the antecedent risk factors and management of pregnancy with stillbirth are well delineated in national guidelines,[1,3,4] there is paucity on the maternal hemorrhagic adverse outcomes among those with livebirth versus stillbirth. Indeed, the ACOG practice bulletin on postpartum hemorrhage (PPH) does not identify stillbirth as a factor which influences whether a parturient is low-, medium-, or high-risk for PPH.[5]

We sought to ascertain if stillbirth, compared with livebirth, is associated with PPH (blood loss of at least 1,000 mL) for three reasons. First, ACOG recommends categorization of all individuals in labor into groups to mitigate adverse outcomes of otherwise unpredictable outcome.[5] Second, while some of the suggested factors are difficult to discern or uncommon (e.g., coagulation defect), stillbirth is not. Third, if stillbirth is risk factor for PPH, then it would be useful for counseling, and undertaking interventions to minimize maternal morbidity. We hypothesized that compared with livebirth, the composite maternal hemorrhagic outcome (CMHO) would be significantly higher among stillbirths.

The objective of the retrospective analysis was to compare the CMHO among individuals with livebirths versus stillbirth.

Key Points

- The risk of CMHO was significantly higher in the stillbirth group even after adjustment for potential confounders (32.6% vs. 16.8%).
- Stillbirth was associated with a significantly higher risk of blood loss of ≥1,000 mL.
- Stillbirth was also associated with higher likelihood of uterotonic use, transfusion, and admission to ICU.

Materials and Methods

Study Design and Participants

We conducted a planned secondary analysis of a retrospective cohort study[6] using data collected from all parturients with singleton pregnancies who were admitted to and delivered at our level IV center from March 1, 2020 to February 28, 2022. Cesarean deliveries were excluded as most stillbirths are delivered vaginally. Clinicians abstracted demographic characteristics, medical comorbidities, pregnancy, and neonatal outcomes from the electronic medical record. Entries were reviewed by senior clinicians for accuracy. This study was approved by University of Texas Health Science Center Houston Institutional Review Board (approval no.: HSC-MS-21–0383). The initial study included all singleton deliveries ≥14 weeks' gestation who delivered in the time period. This analysis was limited to singleton vaginal deliveries ≥20 weeks. We included pre-viable gestations based on the definition of stillbirth by ACOG.

Outcome Measures

The primary outcome was a CMHO consisting in any of the following cumulative estimated or quantified blood loss (EBL or QBL) of ≥1,000 mL, need for uterotonics (excluding prophylactic oxytocin) including additional doses of oxytocin boluses, methylergonovine, carboprost, tranexamic acid, or misoprostol administration during or after delivery, blood transfusion, surgical management of PPH (uterine artery ligation, uterine compression sutures, or uterine artery embolization), intrauterine balloon placement, hysterectomy, venous thromboembolism, admission to intensive care unit, or maternal death. This composite was established based on commonly reported interventions related to PPH in previous literature.

Maternal demographics data were obtained from the medical records and included maternal age, ethnicity, marital status, body mass index (BMI) at delivery, gestational age (GA) at delivery, insurance status, setting of prenatal care, smoking, parity, diabetes, hypertensive disorder of pregnancies, and PPH risk stratification at admission. Additionally, antepartum and intrapartum PPH-specific risk factors were also assessed including: abnormal vital signs at admission, hematocrit <30%, history of PPH, bleeding on admission greater than bloody show, known coagulopathy, prior uterine surgery, magnesium sulfate use, prolonged oxytocin use, fibroids, chorioamnionitis, greater than four prior births, platelet <100, platelet count <50, and retained products of conception.

Statistical Analysis

All data were abstracted from medical records and entered into the REDCap (Vanderbilt University, Nashville, Tennessee, United States) electronic data capture tool hosted at our institution. Differences in baseline characteristics were compared among the two groups using chi-squared tests for dichotomous variables, Kruskal-Wallis for categorical variables, and student *t*-test for averages according to data distribution. Multivariable Poisson regression models with robust error variance were used to estimate the association between stillbirth and CHMO with adjustment for confounders identified in the univariate analysis including GA, bleeding on admission, chorioamnionitis, and prior uterine surgery. All analyses were conducted on STATA statistical software version 18.0 (College Station, TX). The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines for reporting observational studies were followed throughout the manuscript.

Results

Of the 8,623 individuals admitted to the labor and delivery floor during the 24-month period, 4,572 were considered eligible for the analysis, of which 4,483 (98.0%) were livebirths and 89 (1.9%) stillbirths. Maternal baseline characteristics are listed in **Table 1**.

Table 1 Maternal demographics

	Livebirth		Stillbirth		
	n **= 4,483**	**%**	***n*** **= 89**	**%**	***p*-Value**
Maternal age (y)					**0.001**
<20	317	7.1	4	4.5	
20–34	3,504	78.2	59	66.3	
≥35	662	14.8	26	29.2	
Race/ethnicity					0.8354
Black	1,063	23.7	31	34.8	
White	1,086	24.2	21	23.6	
Hispanic	1,037	23.1	24	27.0	
Other	757	16.9	13	14.6	

(*Continued*)

Table 1 (*Continued*)

	Livebirth		Stillbirth		
	***n* = 4,483**	**%**	***n* = 89**	**%**	***p*-Value**
Marital status					**0.002**
Single	2,360	52.6	29	32.6	
Married	1,958	43.7	54	60.7	
Divorced/separated/widowed	40	0.9	2	2.2	
Unknown	125	2.8	4	4.5	
BMI at delivery					0.409
<30.0 $mg^{2/}$/kg	1,836	41.0	44	49.4	
30.0–39.9 $mg^{2/}$/kg	2,066	46.1	34	38.2	
≥40.0 $mg^{2/}$/kg	572	12.8	11	12.4	
Unknown	9	0.2	0	0.0	
Gestational age at delivery					**<0.001**
<32w0d	164	3.7	72	80.9	
32w0d–36w6d	512	11.4	14	15.7	
>37w0d	3,807	84.9	3	3.4	
Private insurance					0.450
No	2,594	57.9	48	53.9	
Yes	1,886	42.1	41	46.1	
Prenatal care provider					0.120
Academic	1,558	34.8	32	36.0	
Private practice	2,680	59.8	47	52.8	
Public clinic	130	2.9	1	1.1	
None	72	1.6	4	4.5	
Unknown	43	1.0	5	5.6	
Smoking during pregnancy					
Yes	120	2.7	1	1.1	0.366
No	4,363	97.3	88	98.9	
Nulliparous		0.0		0.0	0.126
Yes	1,717	38.3	27	30.3	
No	2,766	61.7	62	69.7	
Diabetes					0.686
Yes	398	8.9	9	10.1	
No	4,085	91.1	80	89.9	
Hypertensive disorder					0.386
Yes	1,454	32.4	25	28.1	
No	3,029	67.6	64	71.9	
ACOG PPH risk stratification at admission					**<0.001**
Low	2,515	56.1	32	36.0	
Medium	714	15.9	28	31.5	
High	1,254	28.0	29	32.6	
Maternal Covid					
Yes	188	4.2	3	3.4	0.701
Labored >24 hours					
Yes	365	8.1	9	10.1	0.502
Exposure to oxytocin					
Yes	3,916	87.3	80	89.9	0.475

Abbreviations: ACOG, American College of Obstetrics and Gynecology; BMI, body mass index; PPH, postpartum hemorrhage.

Table 2 Risk factors

	n = 4,483	%	*n* = 89	%	*p*-Value
Abnormal vital signs	715	15.9	12	13.5	0.529
Hematocrit <30%	571	12.7	12	13.5	0.834
History of PPH	82	1.8	1	1.1	0.622
Bleeding on admission	40	0.9	8	9.0	**<0.001**
Known coagulopathy	8	0.2	1	1.1	**0.046**
Prior uterine surgery[a]	157	3.5	22	24.7	**<0.001**
Magnesium sulfate use	351	7.8	14	15.7	**0.006**
Prolonged oxytocin use	400	8.9	6	6.7	0.474
Fibroids	70	1.6	3	3.4	0.178
Chorioamnionitis	144	3.2	17	19.1	**<0.001**
Greater than four prior births	156	3.5	3	3.4	0.956
Operative delivery	210	4.7	3	3.4	0.56
Platelet <100,000	44	1.0	4	4.5	**0.001**
Retained products of conception	29	0.6	10	11.2	**<0.001**

Abbreviation: PPH, postpartum hemorrhage.
Note: [a]Includes both myomectomy and previous cesarean delivery.

Among the two groups, there was a significant difference in maternal age ($p = 0.001$), marital status ($p = 0.002$), GA at delivery ($p < 0.001$), and PPH risk stratification ($p < 0.001$). Among the assessment of risk factors specific for PPH, there was a significant difference in bleeding at admission ($p < 0.001$), prior uterine surgery ($p < 0.001$), magnesium sulfate use ($p = 0.006$), chorioamnionitis ($p < 0.001$), platelet count <100 ($p = 0.001$), platelet count <50 ($p < 0.001$), and retained products of conception ($p < 0.001$). Abnormalities in coagulation were not significantly different between livebirths and stillbirths (**Table 2**).

The risk of CMHO was significantly higher in the stillbirth group even after adjustment for potential confounders (32.6 vs. 16.8%; aRR: 1.56, 95% CI: 1.01–2.46).

Regarding secondary outcomes, stillbirth was also associated with significantly higher risk of EBL or QBL of >1,000 mL (9.0 vs. 2.3%; aRR: 3.81, 95% CI: 1.45–10.54) and ICU admission (5.6 vs. 0.2%; aRR: 26.45, 95% CI: 4.11–169.89). Finally, the rate of tamponade, surgical intervention, VTE, hysterectomy, and maternal death were similar between the two groups (**Table 3**).

Table 3 Composite maternal morbidity

	Livebirth		Stillbirth							
	n = 4,483	%	*n* = 89	%	Unadjusted RR	95% CI		Adjusted RR	95% CI	
Composite maternal hemorrhagic outcome	752	16.8	29	32.6	**1.94**	**1.34**	**2.81**	**1.56**	**1.01**	**2.46**
EBL or QBL ≥ 1,000 mL	102	2.3	8	9.0	**3.95**	**1.92**	**8.11**	**3.81**	**1.45**	**10.54**
Uterotonic use[a]	693	15.5	23	25.8	**1.67**	**1.10**	**2.53**	1.37	0.83	2.26
Transfusion	84	1.9	6	6.7	**3.60**	**1.57**	**8.24**	1.83	0.65	5.15
Tamponade	15	0.3	3	3.4	x	x	x	x	x	x
Surgical intervention[b]	3	0.1	0	0.0	x	x	x	x	x	x
Venous thromboembolism	2	0.0	0	0.0	x	x	x	x	x	x
ICU admission	8	0.2	5	5.6%	**31.48**	**10.30**	**96.23**	**26.45**	**4.11**	**169.89**
Hysterectomy	5	0.1	0	0.0%	x	x	x	x	x	x
Maternal death	0	0.0	0	0.0%	x	x	x	x	x	x

Abbreviations: CI, confidence interval; EBL, estimated blood loss; ICU, intensive care unit; QBL, quantitative blood loss; RR, risk ratio.
Notes: Adjusted for gestational age, bleeding on admission, chorioamnionitis, and prior uterine surgery. Bold values are significant.
[a]Uterotonic use defined as additional doses of oxytocin boluses, methylergonovine, carboprost, tranexamic acid, or misoprostol administration.
[b]Surgical intervention includes uterine artery ligation or placement of compression sutures.

Discussion

Main Findings

Compared with livebirths, individuals who had stillbirth and delivered vaginally had almost 2-fold higher likelihood of composite maternal hemorrhagic outcomes. Blood loss >1,000 mL and ICU admission were the statistically significant components of the composite outcome.

Results in the Context of What Is Known

Previous studies[7–9] explored the likelihood of severe maternal morbidity (SMM) in individuals with stillbirth. Nyarko et al[7] demonstrated a higher likelihood of SMM with stillbirth irrespective of GA. Similarly, Wall-Wieler et al[8] reported roughly a 4-fold increase in the relative risk of SMM among individuals with stillbirths compared with those with livebirth deliveries in over 6 million deliveries in California. Finally, Lewkowitz et al[9] found increased odds of SMM among individuals with stillbirths even after adjusting for maternal sociodemographic characteristics and mode of delivery. However, all these evidence were hampered by few methodological limitations. First, population studies based on ICD-9 and ICD-10 are susceptible to inaccuracies and to underreporting bias.[10] Additionally, although SMM serves as a valuable composite for adverse maternal outcomes, it may not encompass all severe complications such as ICU admission, hysterectomy, and PPH.

Also, another cohort of 543 stillborn deliveries reported by Gold et al[11] showed higher rates of shoulder dystocia and PPH, but without reporting a livebirth population to compare for baseline characteristics and specific outcomes, thus limiting the strength of these findings.

Recently, Beyene et al[12] conducted a small unmatched case–control study involving 333 parturients. They found that women with intrauterine fetal death were 4.2 times more likely to develop PPH compared with those without intrauterine fetal death. Again, significant biases limit the power of these findings due to the absence of a matched control population.

Clinical and Research Implications

Every hour in the United States of America parents grieve the loss of one baby with profound consequences on family's mental health and wellbeing.[13]

Although there is a lack of convincing evidence explaining the main pathophysiological mechanism of bleeding in stillbirths, national guidelines currently consider stillbirth a potential trigger for coagulation abnormalities, as noted in the ACOG bulletin on PPH.[5]

Anecdotally, stillbirth has been associated with increased risks of bleeding potentially due to a consumptive coagulopathy but evidence supporting this association is very scarce. The biological mechanism proposed to explain stillbirth-related coagulopathy involves the release of tissue thromboplastin from the fetal circulation into the maternal circulation. Thromboplastin is thought to stimulate the maternal coagulation system, thus causing intravascular consumption of clotting factors and platelets. A corresponding increase in activity of the fibrinolytic pathway leads to breakdown of fibrinogen to form fibrin degradation products (FDPs) and fibrin-fibrin dimers (D-dimers).[14] However, basic science data demonstrating the causality relation between stillbirth and acquired coagulopathies is still lacking and further research is needed to better understand molecular mechanisms behind this biological hypothesis.

From the findings of this study, we could speculate that the rationale behind the increased risk of CMHO could not be entirely explained by coagulation abnormalities. Other PPH-specific risk factors such as chorioamnionitis, retention of products of conception, bleeding at admission, or the use of magnesium sulfate must be considered when stratifying this subset of pregnancies according to the risk of PPH.

In our cohort, we observed higher rates of chorioamnionitis among the stillbirth population. Chorioamnionitis is recognized as one of the leading cause of stillbirths worldwide, occurring in 10 to 25% of stillbirths in high-income countries and in up to 50% in low- and middle-income countries, particularly when considering preterm stillbirths.[15] It is known that chorioamnionitis is a major determinant of PPH because of the impairment on uterine contractility,[16] thus leading to increased risks of blood transfusion and uterine atony.[17] In this scenario, early evaluation of clinical findings, despite nonspecific, and a prompt management are recommended in pregnancy complicated by stillbirth.

It is also noteworthy that individuals who experienced stillbirth in our cohort were more likely to be diagnosed with retained products of conception. This is not surprising, as individuals who had a stillbirth in our cohort delivered earlier than those with livebirth and retention of placenta is a common complication of preterm deliveries, with the risk decreasing as GA increases.[18] Therefore, an active management of the third stage of labor and postpartum bed-side ultrasound to rule out retained products of conception might be reasonable for patients delivering a stillbirth.

Strengths and Limitations

Our study has several strengths that increase the relevance of our findings. First, the inclusion of a large case cohort gives robustness to our conclusions, and our results are representative of a broad obstetric population. Additionally, we had a stillbirth rate of 1.9%, significantly higher than the national average, giving us a comparably large population to examine. We also included a racially diverse group, with the majority of individuals being of non-white ethnicity, allowing us to generalize our findings more broadly. The cohort includes both private and academic providers, allowing greater diversity in practice styles, which additionally improves generalizability. The dedicated focus on the analysis of PPH-specific risk factors and outcomes allows our findings to shed light on a relatively understudied topic in maternal fetal medicine. Finally, the utilization of adjusted models in our analysis aimed at controlling for potential confounding variables that could otherwise alter the interpretation of our findings. Incorporating these adjusted models allowed us to isolate the specific impact of stillbirth on PPH risk and outcomes, minimizing the influence of confounding variables and enhancing the internal validity of

our results. Consequently, our findings are more robust and reliable, providing greater confidence in the associations identified between stillbirth and maternal hemorrhagic adverse outcomes.

However, our study is not without limitations. As a retrospective cohort study, biases in data collection and analysis can potentially impact the accuracy and reliability of our findings. The lack of available cause of death or pathology reports data for stillbirths represents a significant limitation, limiting our ability to explore potential associations between specific etiologies, particularly placental abruption, a common cause of stillbirth and a known independent risk factor for PPH. Furthermore, the absence of estimated time of death information limits our ability to assess the timing of stillbirth events, potentially impacting the interpretation of maternal outcomes. Our institution is affiliated with a fetal care center, and as such, the etiologic profile of stillbirths in our cohort may differ from that of the general obstetric population, potentially limiting the generalizability of our results. Although we corrected for several cofounders, including bleeding greater than show at admission, there is the potential for residual bias. Although our study has a large percentage of stillbirths, the overall number is small, and our ability to detect minor difference in rare outcomes (such as ICU admission, transfusion) may contribute to a type II error. Finally, the management of these cohorts at a well-resourced quaternary care center may not be widely applicable to community hospitals, where the majority of births in the United States occur.

Conclusion

Based on our results, the risk of hemorrhagic adverse outcomes was 2-fold higher among patients with stillbirth, compared with those delivering a livebirth. Further research should focus on either basic science, trying to understand the biological plausibility of the association between stillbirth and hemorrhagic outcomes, or evidence-based strategies to tailor and stratify the management of this subset of pregnancies.

Author Affiliations

1 Department of Maternal and Child Health and Urological Sciences, Sapienza University of Rome, Italy

2 Department of Obstetrics and Gynecology, ChristianaCare Health System, Newark, Delaware, United States

3 Department of Obstetrics, Gynecology and Reproductive Sciences, University of California San Diego, San Diego, California, United States

4 Department of Obstetrics, Gynecology and Reproductive Sciences, McGovern Medical School at The University of Texas Health Science Center at Houston (UTHealth), Houston, Texas, United States

Statements and Additional Information

Conflict of Interest The authors declare that they have no conflict of interest.

Contributors' Statement F.Z., R.L.W., I.G., A.G., G.R., D.D.M., H.M.F., and S.P.C. contributed to conceptualization; F.Z. and R.L.W. contributed to data curation; F.Z. and R.L.W. contributed to formal analysis; F.Z., R.L.W., and S.P.C. contributed to investigation; F.Z., R.L.W., and S.P.C. contributed to methodology; F.Z., R.L.W., and S.P.C. contributed to resources; S.P.C., D.D.M., and G.R. contributed to supervision; F.Z., R.L.W., D.D.M., and S.P.C. contributed to validation; F.Z., R.L.W., D.D.M., and S.P.C. contributed to visualization; F.Z., R.L.W., and S.P.C. contributed to writing—original draft; F.Z., T.C.L., D.D.M., G.R., A.G., I.G., H.M.F., A.C.S., and S.P.C. contributed to writing—review and editing.

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