


Article

Maternal Race and Clinical Vigilance in Obstetric Hemorrhage Management

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Abstract: Background/Objectives: Previous literature has described that non-white pregnant patients are at increased risk of severe morbidity from obstetric hemorrhage (OBH). Here, we investigate whether such disparities are secondary to delay in the administration of postpartum oxytocin for non-white patients compared to white patients. **Methods:** This is a retrospective cohort study of all deliveries from 2018 to 2019, comparing (1) Hispanic white or non-white race (HW/NWR) pregnant people and (2) non-Hispanic white (NHW) pregnant people. Our primary outcome was the time from delivery to the first dose of postpartum oxytocin, and our secondary outcome was the frequency of other hemorrhage interventions. **Results:** Out of 3832 patients with self-identified race and ethnicity recorded in their patient record, 644 patients identified as NHW, and 3188 patients identified as HW/NWR. We found no difference in time to first dose of postpartum oxytocin ($p = 0.51$), and there was also no difference in the frequency of other hemorrhage-related interventions. **Conclusions:** Our study found no delay in the administration of postpartum oxytocin for non-white patients.

Keywords: quantitative blood loss; maternal morbidity; postpartum oxytocin; pregnancy



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1. Introduction

Obstetric hemorrhage persists as the leading cause of maternal death on the day of birth worldwide, and is the cause of approximately 10% of maternal deaths in the United States [1]. Recent work has robustly demonstrated that being a non-white race is an independent risk factor for preventable morbidities, such as transfusion, disseminated intravascular coagulation (DIC) and peripartum hysterectomy, that can be associated with obstetric hemorrhage [2–5].

Systems-level and provider-level biases are known to contribute to poor obstetric outcomes for non-white pregnant people [6–9]. Few studies, however, have sought to quantify the effects of such biases on obstetric management by examining possible disparities related to vigilance in postpartum monitoring, clinical team coordination, and latency to hemorrhage-related pharmacologic or surgical interventions [10,11]. The primary objective of the present study was to assess for any delay in the administration of postpartum oxytocin or differences in the frequency of additional hemorrhage-related interventions for non-white and Hispanic pregnant people when compared to non-Hispanic white pregnant people.

2. Materials and Methods

This present study is a retrospective cohort study of all deliveries between 1 January 2018, to 1 January 2020. Institutional review board approval was obtained.

Our institution has a postpartum hemorrhage bundle protocol that was implemented in 2017 [Table S1] [12–14]. The components of this include, but are not limited to, a blood type and screen ordered for all patients admitted to labor, a hemorrhage risk assessment on admission and throughout the intrapartum course, and the institution of quantitative blood loss (QBL), which is measured in deliveries [Table S2]. Our institution also has a policy to universally administer postpartum oxytocin immediately after all deliveries, as prior research demonstrates that active management of the third stage of labor leads to decreased total blood loss and a lower risk of hemorrhage [Table S2] [15–17]. Finally, our bundle also contains an algorithm that specifies recommendations of interventions at specific QBL cutoffs (500–1000 cc, 1000–1500 cc, and >1500 cc).

Birth data was gathered from departmental “birth logs” that were collected daily for all deliveries, including information about maternal demographics, medical history, anesthesia type (if any), and QBL. Trained chart abstractors gathered additional information, which was stored in a secure database. We also abstracted self-identified race and ethnicity data from both patient registration information and prenatal provider notes. Categories of race included White, Black, Latino, Asian or Pacific Islander, Middle Eastern, Native American, Mixed Race, or Missing, whereas ethnicity was categorized as Non-Hispanic, Hispanic, or other self-identified ethnicity. We also collected information based on a survey for social determinants of health (“THRIVE” screener), where they could be considered low, medium, or high risk based, on how many areas of “high risk” were identified [18]. Finally, we also extracted information on whether patients elected to use our “birth sister” program, which is a free multi-cultural doula service [19].

The primary outcomes were time latency (minutes) from the time of delivery to the first dose of postpartum oxytocin, the frequency of receiving oxytocin within 1 min after delivery, and the frequency of receiving oxytocin within 5 min after delivery. Our retrospective sample size had 80% power to detect a 5-min difference in the time before the first dose of postpartum oxytocin in each exposure group, with a two-sided 0.05 significance level. The secondary outcomes were the frequency of additional hemorrhage-related interventions (including misoprostol, methergine, carboprost, tranexamic acid, procedural interventions such uterine artery embolization and Bakri balloons, and intra-op interventions such as uterine and uterine artery compression techniques).

The management of hemorrhages necessarily differs by mode of delivery; for our secondary analyses, we sought to parse out potential inequities in treatment delivery that could be specific to the clinical setting. Pharmacologic intervention for hemorrhages in the operating room are primarily managed by the anesthesia team, in contrast to hemorrhage management in labor rooms, where it is primarily the role of the nursing and obstetric team. Furthermore, we wanted to parse out any differences in latency to intervention, as per our proscribed institutional staged response that indicates heightened clinical vigilance and recommendations for additional treatment at specific QBL cutoffs of 500 cc, 1000 cc, and 1500 cc. We thus compared hemorrhage management by maternal race in the following four clinical scenarios: (1) all deliveries regardless of delivery mode with a QBL greater than 1000 cc, (2) vaginal deliveries with a QBL greater than 500 cc, (3) cesarean deliveries with a QBL between 1000 cc and 1500 cc, and (4) cesarean deliveries with a QBL greater than 1500 cc. For these stratified analyses, we also examined the time to additional pharmacologic interventions (e.g., time to first dose of misoprostol).

The two exposure groups (Hispanic white OR non-white race vs. non-Hispanic white race) were compared with descriptive and bivariate statistics using Student’s *t*-test

for continuous variables and chi-squared or 1-sided Fisher's exact test for categorical variables. Potentially confounding variables of the exposure-outcome association were identified in the stratified analyses. Multivariable logistic regression models for the primary outcomes were developed to estimate the effect of other factors on receiving oxytocin in less than 1 or 5 min. Clinically relevant covariates for initial inclusion in multivariable statistical models were selected using results of the stratified analyses, and factors were removed in a backward stepwise fashion, based on significant changes in the exposure adjusted odds ratio. All analyses were completed using STATA MP, version 16 (College Station, TX, USA) [20].

3. Results

Of 3832 patients who delivered a live infant between 1 January 2018 and 31 December 2019 with their self-identified race or ethnicity recorded in their patient record, 644 (16.8%) identified as non-Hispanic white race, 3188 (83.2%) identified as a non-white race. 1500 patients were excluded from the present study as they did not have a self-identified race or ethnicity recorded in their patient record. Of non-white identifying patients, 2003 (62.8%) identified as Black, 562 (17.6%) identified as Hispanic/Latino race, 361 (11.3%) identified as Hispanic ethnicity and white race, 195 (6.1%) identified as Asian or Pacific Islander, 40 (1.25%) identified as Middle Eastern, 16 (0.5%) identified as Native American/Aboriginal, and 11 (0.4%) identified as mixed race.

Maternal and obstetric characteristics, according to self-identified race, are shown in Table 1. The two groups (non-Hispanic white vs. Hispanic white/non-white) were not found to be significantly different with regards to social risk per THRIVE assessment, gestational age at time of delivery, historical obstetric hemorrhage, historical myomectomy, chronic hypertension, anemia, intrapartum intraamniotic infection, infant birth weight, delivery mode, or induction rate. Patients who identified as Hispanic white or non-white race (HW/NWR) were, however, associated with a younger age, a higher pre-pregnancy BMI, a higher use of the "birth sister" program, a higher proportion of a non-English primary language, higher parity, and a higher proportion of medium- or high-admission hemorrhage risk when compared to non-Hispanic white (NHW) race.

The primary and secondary outcomes are displayed in Table 2. There were no unadjusted differences by race in latency to the first dose of oxytocin ($p = 0.51$), or differences in frequency for patients receiving their first dose of postpartum oxytocin within 1 min ($p = 0.25$) or 5 min ($p = 0.08$) (Table 2). After adjusting for age, BMI, birth sister program use, and primary language, there were still no differences by race in the frequency of patients receiving their first dose of postpartum oxytocin within 1 min (adjusted odds ratio [AOR]: 0.9, 95% confidence interval [CI]: 0.57–1.43) or 5 min (AOR: 0.8, 95% CI: 0.49–1.40). There were no differences by race in total number of hemorrhage interventions or rate of individual pharmacologic or non-pharmacologic hemorrhage-related interventions (Table 2).

Table 1. Patient characteristics by race *.

	Non-White Race or Hispanic White (n = 3188)	White (Non-Hispanic) (n = 644)	p Value
Demographics			
Age	30.0 ± 6.1	31.4 ± 4.9	<0.01
Pre-Pregnancy Body Mass Index (kg/m ²)	28.8 ± 6.7	27.6 ± 6.1	<0.01
High-Risk Social Determinants	1117 (35.0)	221 (34.3)	0.73
Birth Sister Program	349 (11.0)	28 (4.4)	<0.01
Primary Language			
English	1747 (54.8)	550 (85.4)	<0.01
Other	1441 (45.2)	94 (14.6)	
Obstetric Characteristics			
Gestational Age	38.9 ± 2.1	38.9 ± 1.9	0.55
Preterm	292 (9.2)	72 (11.2)	0.11
Parity	1.1 ± 1.2	0.9 ± 1.1	<0.01
Chronic Hypertension	222 (7.0)	33 (5.12)	0.09
Intrapartum Intraamniotic Infection	114 (3.6)	21 (3.3)	0.69
Infant Birth Weight	3239.5 ± 600.5	3259.3 ± 556.6	0.44
Cesarean Delivery	1127 (35.4)	212 (32.9)	0.24
Induction	839 (26.3)	187 (29.0)	0.16
Hemorrhage-Related Characteristics			
Anemia (Admission Hematocrit < 28)	77 (2.4)	11 (1.7)	0.27
History of Obstetric Hemorrhage	89 (2.8)	15 (2.3)	0.510
History of Myomectomy	28 (0.9)	2 (0.3)	0.14
Total Quantitative Blood Loss (QBL)	603.4 ± 10.2	593.4 ± 24.0	0.69
Hemorrhage (QBL ≥ 1000)	542 (17.0)	93 (14.4)	0.11
Admission Hemorrhage Risk			
Medium	1597 (50.1)	350 (54.4)	0.05
High	486 (15.3)	71 (11.1)	<0.01

* Means with standard deviations (e.g., 10 ± 5.0 is a mean of 10 with a standard deviation of 5) are listed for all continuous variables, and were compared with a Student's *t*-test. The number of cases with the percentage or frequency in parentheses are listed for all categorical variables and compared with a chi-square test. *p* values for statistical significance are listed in the last column.

The results of a stratified analysis of only the patients who were identified as having an obstetric hemorrhage (total QBL greater than or equal to 1000 cc) are displayed in Table 3. There were no significant differences by race in rate of individual pharmacologic or non-pharmacologic hemorrhage-related interventions. There were also no differences in latency to pharmacologic interventions.

Table 2. Hemorrhage intervention by race.

	Non-White Race or Hispanic White (n = 3188)	White (Non-Hispanic) (n = 644)	aOR (95% CI)	p Value
Latency to postpartum oxytocin				
Time to oxytocin	5.9 ± 23.3	6.7 ± 34.7	--	0.51
Received oxytocin < 1 min	1604 (50.3)	308 (47.8)	0.9 (0.57–1.43) *	0.25
Received oxytocin < 5 min	2426 (76.1)	469 (72.8)	0.8 (0.49–1.40) *	0.08
Hemorrhage Interventions				
Misoprostol (Y/N)	426 (13.4)	94 (14.6)	--	0.40
Methergine (Y/N)	174 (5.5)	41 (6.4)	--	0.36
Carboprost (Y/N)	82 (2.6)	10 (1.6)	--	0.12
Tranexamic Acid (Y/N)	180 (5.7)	28 (4.4)	--	0.19
B-Lynch (Y/N)	9 (0.9)	3 (1.6)	--	0.36
O'Leary (Y/N)	16 (1.5)	2 (1.0)	--	0.61
Bakri (Y/N)	24 (1.0)	4 (0.8)	--	0.70
Uterine Art. Embolization (Y/N)	8 (1.7)	0 (0.0)	--	0.27
Transfusion (Y/N)	98 (20.3)	14 (16.3)	--	0.24

* adjusted for age, BMI, birth sister, primary language.

Table 3. Hemorrhage-related interventions by race: deliveries with hemorrhage (QBL > 1000) only.

	Non-White Race or Hispanic White (n = 549)	White (Non-Hispanic) (n = 96)	p Value
Oxytocin			
Time to oxytocin	6.5 ± 25.9	2.9 ± 4.3	0.20
Received oxytocin < 1 min	391 (71.2)	62 (64.6)	0.19
Received oxytocin < 5 min	461 (84.0)	76 (79.2)	0.25
Other Pharmacologic Interventions			
Misoprostol	64 (11.7)	14 (14.6)	0.42
Time to misoprostol (minutes)	55.1 ± 13.1	45.0 ± 14.9	0.73
Methergine	33 (6.0)	6 (6.3)	0.54
Time to methergine (minutes)	82.8 ± 25.4	31.0 ± 16.4	0.40
Carboprost	11 (2.0)	0 (0.0)	0.17
Time to carboprost (minutes)	132.5 ± 192.8	--	--
Tranexamic Acid (TXA)	23 (4.2)	5 (5.2)	0.65
Time to TXA (minutes)	51.0 ± 20.3	69.4 ± 39.2	0.70
Procedural Interventions			
Bakri	17 (3.5)	3 (3.6)	0.58
Return to operating room	37 (6.7)	5 (5.2)	0.38
B-Lynch (Y/N)	4 (1.1)	3 (5.3)	0.05
O'Leary (Y/N)	9 (2.4)	1 (1.8)	0.61
Uterine Art. Embolization (Y/N)	7 (1.5)	0 (0.0)	0.32
Other			
Transfusion (Y/N)	92 (19.8)	14 (14.6)	0.34

We further stratified the results by both quantitative blood loss and delivery mode and these findings are displayed in Table 4. There were no significant differences by race in the rate of hemorrhage-related interventions or in latency to interventions in the stratified group of patients who had a QBL greater than 500 cc in a vaginal delivery (total n = 674) or the stratified group of patients who had a QBL between 1000 and 1500 cc in a Cesarean delivery (total n = 281). Patients who identified as NHW, with a total QBL greater than 1500 in a Cesarean, more frequently had a uterine compression suture (B-Lynch) placed intra-operatively, compared to patients who identified as HW/NWR 4 (2.7%) versus 3 (13.6%), respectively, ($p = 0.05$). There were no other differences by race in the rate of or latency to individual pharmacologic or non-pharmacologic hemorrhage-related interventions among the 181 patients with a QBL greater than 1500 cc in a Cesarean delivery (Table 4).

Table 4. Hemorrhage interventions by race: stratification by delivery mode and QBL.

	Non-White Race or Hispanic White	White (Non-Hispanic)	<i>p</i> Value
Vaginal Deliveries QBL > 500	n = 544	n = 130	
Time to oxytocin (minutes)	5.6 ± 21.3	7.5 ± 25.3	0.42
Misoprostol	77 (14.2)	17 (13.1)	0.75
Time to misoprostol (minutes)	67.3 ± 176.3	97.5 ± 172.3	0.52
Methergine	30 (5.5)	6 (4.6)	0.68
Time to methergine (minutes)	33.6 ± 57.6	22.3 ± 3.9	0.64
Carboprost	16 (2.9)	1 (0.77)	0.16
Time to carboprost (minutes)	83.7 ± 156.4	--	--
Tranexamic Acid (TXA)	33 (6.1)	7 (5.4)	0.77
Time to TXA (minutes)	33.2 ± 66.6	25.6 ± 20.5	0.77
Bakri	9 (2.1)	1 (1.0)	0.47
Return to operating room	29 (5.3)	3 (2.3)	0.15
Transfusion (Y/N)	27 (20.5)	4 (13.8)	0.30
Cesarean 1000 < QBL < 1500	n = 242	n = 39	
Time to Oxytocin (minutes)	7.9 ± 33.1	2.9 ± 4.4	0.34
Misoprostol	24 (9.9)	4 (10.3)	0.57
Time to misoprostol (minutes)	51.8 ± 18.3	41.5 ± 22.8	0.83
Methergine	14 (5.8)	3 (7.7)	0.43
Time to methergine (minutes)	110.7 ± 199.7	43.3 ± 59.5	0.58
Carboprost	5 (2.07)	0 (0.0)	0.47
Time to carboprost (minutes)	--	--	--
Tranexamic Acid (TXA)	8 (3.3)	1 (2.6)	0.64
Time to TXA (minutes)	13.8 ± 4.2	--	--
Bakri	0 (0.0)	0 (0.0)	--
Return to operating room	2 (0.8)	0 (0.0)	0.74
B-Lynch (Y/N)	0 (0.0)	0 (0.0)	--
O'Leary (Y/N)	2 (0.9)	0 (0.0)	0.75
Uterine Art. Embolization (Y/N)	0 (0.0)	0 (0.0)	--
Transfusion (Y/N)	9 (4.7)	1 (3.0)	0.56

Table 4. Cont.

	Non-White Race or Hispanic White	White (Non-Hispanic)	<i>p</i> Value
Cesarean QBL > 1500	n = 156	n = 25	
Time to oxytocin (minutes)	5.8 ± 20.4	2.7 ± 3.7	0.47
Misoprostol	19 (12.2)	4 (16.0)	0.59
Time to misoprostol (minutes)	38.4 ± 63.2	13.8 ± 4.0	0.45
Methergine	5 (3.2)	2 (8.0)	0.25
Time to methergine (minutes)	99.4 ± 50.9	20.5 ± 3.5	0.40
Carboprost	1 (0.6)	0 (0.0)	0.86
Time to carboprost (minutes)	--	--	--
Tranexamic Acid (TXA)	4 (2.6)	0 (0.0)	0.42
Time to TXA (minutes)	124.3 ± 136.2	--	--
Bakri	8 (5.4)	3 (12.5)	0.19
Return to operating room	10 (6.4)	2 (8.0)	0.52
B-Lynch (Y/N)	4 (2.7)	3 (13.6)	0.05
O'Leary (Y/N)	7 (4.6)	1 (4.6)	0.73
Uterine Art. Embolization (Y/N)	4 (2.7)	0 (0.0)	0.58
Transfusion (Y/N)	57 (38.8)	9 (40.9)	0.51

4. Discussion

We found no delay in the administration of postpartum oxytocin when comparing HW/NWR patients to NHW patients. We also did not find any differences by race in the total number of hemorrhage interventions utilized or the rate of pharmacologic treatment of hemorrhage.

Prior research has primarily focused on delineating the racial disparities in obstetric outcomes. Multiple large national database studies have shown that non-white birthing people are at significantly higher risk for severe morbidity and mortality associated with postpartum hemorrhage [2,3]. Black patients have been found to have a higher risk of disseminated intravascular coagulation, transfusion, and maternal death from hemorrhage [2]. Hispanic and Asian or Pacific Islander (API) patients have also been found to have significantly increased odds of atonic postpartum hemorrhage, with Asian/Pacific Islander patients found to have the highest risk of requiring a hysterectomy [2,4,21]. Additionally, a recent systematic review and meta-analysis found that an Asian race and a Hispanic ethnicity were definite and likely risk factors, respectively, for hemorrhage secondary to uterine atony [5].

There is no scientific basis for the antiquated claim that biologic differences inherent to a socially constructed racial identity would make any patient physiologically at increased risk for obstetric hemorrhage [22,23]. However, there continue to be major gaps in our understanding of what systemic factors may be at the root of such racial disparities in obstetric outcomes. A major strength of our study was thus our choice in primary outcome. We deliberately chose a primary outcome that did not further engrain the racist myth that “biologic race” is an inherent risk factor for poor health; we chose instead a primary outcome—latency to intervention—that lent a more critical eye to the role of healthcare providers in providing equitable care, to detect a 5-min difference in time to first dose of postpartum oxytocin.

While we, ultimately, did not find any differences in the present study, future studies should continue to openly acknowledge and investigate the role that bias and systemic racism plays when forming clinical research questions. For example, while our cohort only

included deliveries after the implementation of our obstetric hemorrhage bundle, future studies could consider examining outcomes both before and after the obstetric bundle implementation to see if the standardization of the prevention, diagnosis and treatment of obstetric complications may serve a role in decreasing racial inequities [12–14]. Researchers could also consider disaggregating data by health literacy status, primary patient language, and other social determinants of health to ensure that patients do not experience a delay to care in obstetric emergencies as a result of provider or systemic biases.

We acknowledge there are limitations in the present study. As a non-randomized single institution cohort study over the span of two years, we had unequal sample sizes in our comparison groups; in addition, our study was underpowered for both the finer analyses investigating the frequency of non-oxytocin hemorrhage-related interventions and the stratified analyses that we conducted. There are also many limitations to engaging with the difficult conundrum of situating the role of race in investigative medical research. Limiting patients to self-identify into pre-determined discrete “categories” of race at the time of hospital registration may indirectly feed the influence of the harmful solipsistic social construct of race science.

Furthermore, to deconstruct differences in care provided to patients based on their self-identified race, we aggregated many non-white races and ethnicities, which may mask the subtler outcomes that may exist in more finely stratified data. In addition, as our patient population happens to have a low representation of API patients, we cannot comment on whether management of hemorrhage intervention may affect the disproportionate burden of hemorrhage experienced by API patients as has been documented in the literature [21,24].

5. Conclusions

We did not find any differences by race in the latency to hemorrhage intervention at our institution. Future studies should continue to move beyond simplistic descriptive studies that ask “if” clinical outcomes differ by race, and instead seek new ways to ask “how” systemic racism and biased human behavior may impact outcomes and patient care.

Supplementary Materials: The following supporting information can be downloaded at: <https://www.mdpi.com/article/10.3390/reprodmed6010001/s1>, Table S1: Obstetric Hemorrhage Bundle Components; Table S2: Obstetric Hemorrhage Risk Assessment Tool.

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