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Blood Type Association with Bleeding Outcomes at Delivery in a Large Multi-Center Study

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Abstract

Background.—Postpartum hemorrhage is a leading cause of maternal death globally. Recent studies have associated Type-O group to increased risk of bleeding. We aimed to determine if women with Type-O blood are at higher risk of PPH.

Methods.—This is a retrospective cohort analysis of a multi-center database included women admitted to labor and delivery from January 2015 to June 2018. All deliveries resulting in live birth were included. Association between Type-O and non Type-O were examined using chi-square test and fishers exact test. Prevalence of postpartum hemorrhage, estimated blood loss, drop in hematocrit and red blood cell transfusion were compared.

Results.—The matched sample included 40,964 Type-O and the same number of no Type-O. The overall prevalence of postpartum hemorrhage was 6.4 %, and there was no difference in the prevalence of PPH among Type-O compared to non Type-O (6.38% vs. 6.37% respectively; p=0.96). There was no difference in hematocrit drop and estimated blood loss between Type-O and non Type-O in all deliveries. However, in cesarean delivery there was a significant difference in blood loss among the two groups Finally, Type-O had 1.09 fold increased risk for transfusion compared to non Type O (95% CI, 0.9 - 1.34).

Conclusion.—There is an association between Type-O group and risk of bleeding in women undergoing cesarean delivery. More prospective studies, taking into account coagulation profile,

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platelet count and tissue factors, are needed to draw a conclusion on whether ABO system can be considered a heritable risk of postpartum hemorrhage.

Keywords

ABO; Postpartum hemorrhage; von Willebrand disease; blood group

Introduction

Postpartum hemorrhage (PPH) is a leading cause of maternal mortality and morbidity globally, both in low and high resource countries, and it accounts for more than two-thirds of hemorrhage-related deaths in the obstetrical setting [1–5]. In addition, peripartum hysterectomy and postpartum intensive care support are major morbidities resulting from PPH [6,7]. Recently, the American College of Obstetricians and Gynecologists defined PPH as the cumulative blood loss greater than or equal to 1000 mL with signs or symptoms of hypovolemia within 24 hours after delivery regardless of the delivery method. This is an update to the previous definition of a 500 mL blood loss in vaginal delivery and 1000 mL in cesarean delivery, yet they still recognize that blood loss greater that 500 mL in vaginal delivery warrants further evaluation [8].

Most cases of PPH occur as a result of disturbance in one or both mechanisms that exist to maintain hemostasis including contraction of the myometrium and local decidual hemostatic factors. The former mechanism is the most common cause of PPH in the United States, responsible for at least 75 % of the cases [9]. Other causes include trauma, laceration, placental abnormalities, uterine inversions, and inherited or acquired blood diathesis [8].

The most common inherited bleeding disorder is von Willebrand Disease (vWD), caused by a quantitative deficiency (type 1 and 3) or a qualitative defect of von Willebrand factor (vWF) [10]. vWF plays a pivotal role in primary hemostasis through platelet sub-endothelial adhesion, platelet-to-platelet, and platelet aggregation, by acting as a specific carrier of FVIII and protecting it from proteolytic degradation [11]. Seventy percent of plasma variation in vWF/FVIII is genetically determined with nearly one-third of this variation accounted for by ABO type [12]. Type-O blood has been shown to have 25% lower plasma levels of vWF and Factor VIII (FVIII) than the non Type-O [12]. Several studies have been performed with varying results of either increased propensity to bleeding in Type-O patients or no significantly increased bleeding [13–16] . The published literature is therefore inconclusive regarding the impact of Type-O blood on hemorrhage. A large meta-analysis of 22 studies was performed in which 4,919 of 9,468 patients with bleeding complications were Type-O (52%) and 202,319 of 457,284 patients without bleeding complications were Type-O (44.2%), thus showing Type-O blood patients are at higher risk of bleeding compared to non Type-O patients (95% CI=1.25 to 1.42, $p<0.001$) [17]. This leads to the premise that patients with Type-O blood may indeed be at increased risk of hemorrhage.

Physiologic changes of pregnancy significantly increase levels of vWF and FVIII in women without an underlying hematologic disorder, reaching levels by far >100 U/dL at the time of delivery. For women with vWD who have baseline levels of vWF and FVIII>30 u/dL, the corrective adaptation induced by pregnancy gives these individuals the ability to achieve

normal levels by late gestation [18]. Therefore, pregnancy itself may compensate for lower than normal vWF/FVIII in Type-O individuals, providing a protective effect for increased risk of bleeding during and after delivery. In our study, we aimed to evaluate the association between Type-O blood and risk of bleeding among patients undergoing vaginal and cesarean delivery and identifying independent risk factors for hemorrhage.

Methods

This was a retrospective cohort analysis of pregnant women who delivered from January 2015 to June 2018 by vaginal or cesarean delivery at nineteen hospitals belonging to a large national hospital management company in the United States. Data on demographics, maternal characteristics, preexisting conditions, and pregnancy-related conditions were extracted from the national database. This dataset offered a large sample population encompassing various hospital sizes across the country. Women with a reported blood type were included. PPH was defined as blood loss greater than or equal 1000 mL regardless of delivery mode. Using at least 40,000 per group , a power >0.95 was obtained to detect a difference in the incidence of transfusion of 0.5% vs 0.7%, which was defined as the minimum clinically relevant difference.

The primary outcome was hemorrhage in Type-O and non Type-O patients reported as hematocrit absolute drop and stratified into 4 categories (0 to <5%, 5% to <10%, 10% to <15%, and ≥15%). Hematocrit drop was missing for a total of 14,240 patients. Estimated blood loss (EBL) was compared among patients with blood Type-O and non Type-O among all modes of deliveries and subdivided by mode of delivery (vaginal and cesarean). EBL was divided into four categories: <500 mL, >500 mL to <1000 mL>1000 mL to <2000 mL, ≥ 2000 mL and each category was compared across the dependent variable.

We compared birth outcomes in propensity matched patients using generalized estimating equations with patients nested within matched pairs, and adjusting for the propensity to have Type-O. Propensity for Type-O was matched using the Greedy Matching algorithm with caliper of 2%, 1-to-1 for Type-O vs non-O patients. Propensity for type O was the probability of Type O, based on the results of a logistic regression model predicting blood type based on the predictors: delivery type, race, marital status, age, body mass index, parity, number of previous cesarean sections, predelivery hematocrit and white blood cell count, Rhesus status, diabetes mellitus, gestational diabetes, interpregnancy interval, placenta previa, use of vacuum or forceps, and chorioamnionitis. Probability of Type O was calculated for each patient, from the regression equation y = intercept + b1v1 + b2v2 + … + bnvn, where the b's were the regression parameter estimates and v's were the values of each variable in the model, for each patient, with the equation probability = exp(y)/ (1 + exp(y)). Balance was checked for pre-delivery variables after matching using chi-square or Fishers exact test for categorical variables, and t-test for continuous variables. Any background variables that remained significantly different between blood types after matching, if any, would have been used as covariates in the final generalized estimating equations (GEE) models. The study was approved by the George Washington University Institutional Review Board.

Results

The original data included 50,052 Type-O mothers and 46,325 non Type-O. The matched sample included 40,964 Type-O and the same number of non Type-O. Among the non Type-O patients, 66.5% were Type-A, 26.2% were Type-B, and 7.1% were Type-AB. In addition, 62.8% of the patients had a vaginal delivery, whereas 37.2% had a cesarean delivery. When we compared the maternal characteristics between Type-O and non Type-O (Table 1.), there was no significant difference among the demographical and patient-related variables between the matched patients. Postpartum hemorrhage, defined as >1000 ml regardless of type of delivery, in Type-O patients was 6.37% and that in non Type-O was 6.38% (p=0.96).

There was no significant difference in absolute hematocrit drop between patients with Type-O and non Type-O in all deliveries (p=0.2), Table 2. When stratified by mode of delivery, there was no significant difference in the absolute hematocrit drop in any category between the Type-O and non Type-O in vaginal delivery (p=0.75). However, there was a trend-level association in cesarean delivery, in which the lowest level of HCT drop (0% - <5%) was slightly more likely for mothers with non Type-O (53% vs 52%).

EBL categories were not significantly different among the two groups in the vaginal delivery (p=0.84). However, in cesarean delivery, there was a significant association between blood type and EBL, in which EBL <500 ml was slightly more likely for mothers with Type-O, compared with non Type-O (25.6% vs 24.3%; p=.03). When taking all deliveries into account, there was no significant difference in estimated blood loss across all four categories between Type-O and non Type-O patients (p=0.45) Table 3. The prevalence of PPH among cesarean deliveries (defined as EBL >1000 mL) was 10.1% (3091/30496). In addition, the prevalence of PPH among vaginal deliveries (defined as EBL >500 mL) was 4.9% (2540/51,432).

There were 203 Type-O patients with transfusion (0.50%) and 186 non-Type-O patients (0.45%) (p=0.42). In the GEE model, after nesting cases within matched pairs and adjusting for the propensity to have Type-O, the adjusted OR for transfusion was 1.09 (95% CI 0.90 – 1.34; p=.38) for those with Type) vs non Type-O patients.

Discussion

In this large multi-center analysis of the relationship between blood type and bleeding/PPH, there was an association between blood loss and Type-O among cesarean delivery. These important findings were in accordance to an expected relationship showing PPH with Type-O blood as described by Drukker et. al [14]. In that study, Type-O patients were at 1.14 fold risk of PPH (2.3% vs 2.0%, p<0.001); a mild risk that persisted after multivariate analysis but there was no difference in blood product transfusion among patients by blood type. Another study evaluating postpartum blood loss showed that Type-O patients had higher blood loss (529.2 mL ± 380.4 mL vs. 490.5 mL ± 276.4mL, p=0.002) [13]. Contrary to our findings, Ali-Saleh et al. found no association of blood type with increased risk of early PPH [16]. Interestingly, the association between bleeding and blood group has not been restricted

to the obstetrics population; a study comparing the risk of bleeding of gastroduodenal ulcers and Type-O blood patients showed a positive correlation as well [15].

One potential hypothesis to explain these findings is that patients with Type-O blood have a different coagulation profile compared to non Type-O patients. In particular, the concentration levels and activity of vWF are decreased in Type-O patients [19–21]. A recent meta-analysis by Dentali showed Type-O blood poses a heritable risk factor for bleeding [17]. Moreover, patients with non Type-O are at 2-folds higher risk of developing venous thromboembolism [22]. Another study showed that the risk for cerebral hemorrhage and thromboembolism were not significantly different between patients with blood Type-O and non Type-O, although patients with blood Type-O were at lower risk of nonvalvular atrial fibrillation [23]. In one study assessing the relation between Type-O and risk of bleeding in cardiac surgery, Welsby et al. showed that there is no significant difference in postoperative primary hemostasis despite laboratory evidence for abnormal primary hemostasis among patients with Type-O prior to surgery [24].

vWF can rise up 60-100% higher of baseline levels in the third trimester of pregnancy compared to the non-pregnant state. [25]. There is a higher risk of PPH in patients with low third trimester vWF levels [26]. Given the large database nature of the current study, vWF assays were not available for analysis. However, the authors propose that the clinically significant data on PPH and transfusion requirement can be an indirect surrogate. In this study, there is no statistical significance in the need for transfusion among Type-O and non Type-O patients; therefore, despite potentially low vWF levels, transfusion requirement was not impacted. An explanation is that the phenomenon of hemorrhage is multidimensional involving imbalance between coagulation and anticoagulation cascades including a myriad list of proteins and factors. Other known risk factors of postpartum hemorrhage may also play a role although in our cohort there was no significant difference among them including predelivery anemia, assisted vaginal delivery and chorioamnionitis. In a recent study, it has been shown that pre-delivery anemia is a risk factor for increased bleeding after delivery [27].

It has been shown in the literature that cesarean delivery, operative vaginal delivery (forceps), and placenta previa are independent risk factors [28,29]. There is controversial evidence on how to choose the hemostasis outcome when studying PPH. Despite clear definitions of PPH, diagnosing PPH by the amount of blood loss remains daunting because of lack of an accurate measurement tool. Finally, deciding on the primary outcome, whether it is the need for transfusion or maternal comorbidity composite score, can be challenging and not uniform across both descriptive and analytical studies.

Major strengths of this study are the heterogeneous population from over 19 different hospitals across the United States, the large number of subjects included (>80,000), which gave us the power to detect small differences between blood type groups, and the availability of extensive demographic, medical and obstetrical data. However, the retrospective study design is of course a limitation—especially in potential lack of accurate diagnosis of PPH and the lack of knowledge regarding the manner from which the blood loss measurements were made.

In conclusion, we highlight our results showing no significant association between non Type-O and risk of blood loss among all deliveries, despite a slight increase in postpartum hemorrhage among cesarean deliveries in the Type-O group.. The clinical benefit of determining whether the ABO system possesses an inheritable bleeding risk is important, particularly in the obstetrics setting, as several task forces ,including the Association of Women's Health, Obstetrics and Neonatal Nurses (AWHONN) and the California Maternal Quality Care Collaborative (CMQCC) [30,31], are working on delineating the risk factors associated with postpartum hemorrhage and constructing protocols for effective management. Further prospective studies to identify differences between non Type-O and Type-O patients are warranted. These should include analysis of various coagulation studies including vWF, fibrinogen, factor VIII, and platelet studies. In the event that Type-O is indeed a heritable risk factor for PPH, we believe targeted hemodynamic observations and possible pharmacologic prophylaxis for select pregnant patients during labor and delivery has the potential to reduce adverse bleeding events and maternal mortality.

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References

1. James AH, Jamison MG (2007) Bleeding events and other complications during pregnancy and childbirth in women with von Willebrand disease. J Thromb Haemost 5 (6):1165–1169. doi:10.1111/j.1538-7836.2007.02563.x [PubMed: 17403089]
2. Khan KS, Wojdyla D, Say L, Gulmezoglu AM, Van Look PF (2006) WHO analysis of causes of maternal death: a systematic review. Lancet 367 (9516):1066–1074. doi:10.1016/S0140-6736(06)68397-9 [PubMed: 16581405]
3. Knight M, Callaghan WM, Berg C, Alexander S, Bouvier-Colle MH, Ford JB, Joseph KS, Lewis G, Liston RM, Roberts CL, Oats J, Walker J (2009) Trends in postpartum hemorrhage in high resource countries: a review and recommendations from the International Postpartum Hemorrhage Collaborative Group. BMC Pregnancy Childbirth 9:55. doi:10.1186/1471-2393-9-55 [PubMed: 19943928]
4. Say L, Chou D, Gemmill A, Tuncalp O, Moller AB, Daniels J, Gulmezoglu AM, Temmerman M, Alkema L (2014) Global causes of maternal death: a WHO systematic analysis. Lancet Glob Health 2 (6):e323–333. doi:10.1016/S2214-109X(14)70227-X [PubMed: 25103301]
5. Zhang WH, Alexander S, Bouvier-Colle MH, Macfarlane A, Group M-B (2005) Incidence of severe pre-eclampsia, postpartum haemorrhage and sepsis as a surrogate marker for severe maternal morbidity in a European population-based study: the MOMS-B survey. BJOG 112 (1):89–96. doi:10.1111/j.1471-0528.2004.00303.x
6. Zwart JJ, Dupuis JR, Richters A, Ory F, van Roosmalen J (2010) Obstetric intensive care unit admission: a 2-year nationwide population-based cohort study. Intensive Care Med 36 (2):256–263. doi:10.1007/s00134-009-1707-x [PubMed: 19902177]
7. Zwart JJ, Richters JM, Ory F, de Vries JI, Bloemenkamp KW, van Roosmalen J (2008) Severe maternal morbidity during pregnancy, delivery and puerperium in the Netherlands: a nationwide population-based study of 371,000 pregnancies. BJOG 115 (7):842–850. doi:10.1111/j.1471-0528.2008.01713.x [PubMed: 18485162]
8. Committee on Practice B-O (2017) Practice Bulletin No. 183: Postpartum Hemorrhage. Obstet Gynecol 130 (4):e168–e186. doi:10.1097/AOG.0000000000002351 [PubMed: 28937571]
9. Marshall AL, Durani U, Bartley A, Hagen CE, Ashrani A, Rose C, Go RS, Pruthi RK (2017) The impact of postpartum hemorrhage on hospital length of stay and inpatient mortality: a National

Inpatient Sample-based analysis. Am J Obstet Gynecol 217 (3):344 e341–344 e346. doi:10.1016/j.ajog.2017.05.004 [PubMed: 28502758]

10. Bannow BS, Konkle BA (2018) Inherited Bleeding Disorders in the Obstetric Patient. Transfus Med Rev 32 (4):237–243. doi:10.1016/j.tmrv.2018.06.003 [PubMed: 30097224]
11. Franchini M, Capra F, Targher G, Montagnana M, Lippi G (2007) Relationship between ABO blood group and von Willebrand factor levels: from biology to clinical implications. Thromb J 5:14. doi:10.1186/1477-9560-5-14 [PubMed: 17894864]
12. Jenkins PV, O'Donnell JS (2006) ABO blood group determines plasma von Willebrand factor levels: a biologic function after all? Transfusion 46 (10):1836–1844. doi:10.1111/j.1537-2995.2006.00975.x [PubMed: 17002642]
13. Kahr MK, Franke D, Brun R, Wisser J, Zimmermann R, Haslinger C (2018) Blood group O: A novel risk factor for increased postpartum blood loss? Haemophilia 24 (4):e207–e212. doi:10.1111/hae.13537 [PubMed: 29877601]
14. Drukker L, Srebnik N, Elstein D, Levitt L, Samueloff A, Farkash R, Grisaru-Granovsky S, Sela HY (2016) The association between ABO blood group and obstetric hemorrhage. J Thromb Thrombolysis 42 (3):340–345. doi:10.1007/s11239-016-1360-5 [PubMed: 27071803]
15. Bayan K, Tuzun Y, Yilmaz S, Dursun M, Canoruc F (2009) Clarifying the Relationship Between ABO/Rhesus Blood Group Antigens and Upper Gastrointestinal Bleeding. Digestive Diseases and Sciences 54 (5):1029–1034. doi:10.1007/s10620-008-0446-0 [PubMed: 18716867]
16. Ali-Saleh M, Lavie O, Abramov Y (2019) Evaluation of blood type as a potential risk factor for early postpartum hemorrhage. Plos One 14 (4). doi:ARTN e0214840 10.1371/journal.pone.0214840
17. Dentali F, Sironi AP, Ageno W, Bonfanti C, Crestani S, Frattini F, Steidl L, Franchini M (2013) Relationship between ABO Blood Group and Hemorrhage: A Systematic Literature Review and Meta-Analysis. Seminars in Thrombosis and Hemostasis 39 (1):72–82. doi:10.1055/s-0032-1329550 [PubMed: 23299820]
18. Castaman G (2013) Changes of von Willebrand Factor during Pregnancy in Women with and without von Willebrand Disease. Mediterr J Hematol Infect Dis 5 (1):e2013052. doi:10.4084/MJHID.2013.052 [PubMed: 23936623]
19. Moeller A, Weippert-Kretschmer M, Prinz H, Kretschmer V (2001) Influence of ABO blood groups on primary hemostasis. Transfusion 41 (1):56–60. doi:10.1046/j.1537-2995.2001.41010056.x [PubMed: 11161246]
20. Leebeek FW, Eikenboom JC (2016) Von Willebrand's Disease. N Engl J Med 375 (21):2067–2080. doi:10.1056/NEJMra1601561 [PubMed: 27959741]
21. Gill JC, Endres-Brooks J, Bauer PJ, Marks WJ Jr, Montgomery RR, (1987) The effect of ABO blood group on the diagnosis of von Willebrand disease. Blood 69 (6):1691–1695 [PubMed: 3495304]
22. Franchini M, Mannucci PM (2014) ABO blood group and thrombotic vascular disease. Thromb Haemost 112 (6):1103–1109. doi:10.1160/TH14-05-0457 [PubMed: 25187297]
23. Blustin JM, McBane RD, Mazur M, Ammash N, Sochor O, Grill DE, Wysokinski WE (2015) The Association Between Thromboembolic Complications and Blood Group in Patients With Atrial Fibrillation. Mayo Clinic Proceedings 90 (2):216–223. doi:10.1016/j.mayocp.2014.11.013 [PubMed: 25659240]
24. Welsby IJ, Jones R, Pylman J, Mark JB, Brudney CS, Phillips-Bute B, Mathew JP, Campbell ML, Stafford-Smith M, Cardiothoracic Anesthesiology Research Endeavours DoADUMC (2007) ABO blood group and bleeding after coronary artery bypass graft surgery. Blood Coagul Fibrinolysis 18 (8):781–785. doi:10.1097/MBC.0b013e3282f1029c [PubMed: 17982320]
25. James AH, Konkle BA, Kouides P, Ragni MV, Thames B, Gupta S, Sood S, Fletcher SK, Philipp CS (2015) Postpartum von Willebrand factor levels in women with and without von Willebrand disease and implications for prophylaxis. Haemophilia 21 (1):81–87. doi:10.1111/hae.12568 [PubMed: 25333737]
26. Stoof SC, van Steenbergen HW, Zwagemaker A, Sanders YV, Cannegieter SC, Duvekot JJ, Leebeek FW, Peters M, Kruip MJ, Eikenboom J (2015) Primary postpartum haemorrhage in

women with von Willebrand disease or carriership of haemophilia despite specialised care: a retrospective survey. Haemophilia 21 (4):505–512. doi:10.1111/hae.12635 [PubMed: 25688733]

27. Ahmadzia HK, Phillips JM, James AH, Rice MM, Amdur RL (2018) Predicting peripartum blood transfusion in women undergoing cesarean delivery: A risk prediction model. PLoS One 13 (12):e0208417. doi:10.1371/journal.pone.0208417 [PubMed: 30551126]
28. Helman S, Drukker L, Fruchtman H, Ioscovich A, Farkash R, Avitan T, Samueloff A, Grisaru-Granovsky S (2015) Revisit of risk factors for major obstetric hemorrhage: insights from a large medical center. Arch Gynecol Obstet 292 (4):819–828. doi:10.1007/s00404-015-3725-y [PubMed: 25903520]
29. Reyal F, Sibony O, Oury JF, Luton D, Bang J, Blot P (2004) Criteria for transfusion in severe postpartum hemorrhage: analysis of practice and risk factors. Eur J Obstet Gynecol Reprod Biol 112 (1):61–64. doi:10.1016/j.ejogrb.2003.04.001 [PubMed: 14687741]
30. Main EK, Cape V, Abreo A, Vasher J, Woods A, Carpenter A, Gould JB (2017) Reduction of severe maternal morbidity from hemorrhage using a state perinatal quality collaborative. Am J Obstet Gynecol 216 (3):298 e291–298 e211. doi:10.1016/j.ajog.2017.01.017 [PubMed: 28153661]
31. Kawakita T, Mokhtari N, Huang JC, Landy HJ (2019) Evaluation of Risk-Assessment Tools for Severe Postpartum Hemorrhage in Women Undergoing Cesarean Delivery. Obstet Gynecol 134 (6):1308–1316. doi:10.1097/AOG.0000000000003574 [PubMed: 31764744]

Highlights

- The overall prevalence of PPH in our study was 6.4%.
- Non Type-O is significantly related with drop in hematocrit in cesarean delivery, but this does not translate to increased transfusion requirement.
- No difference in Patients with Type-O and non Type-O for estimated blood loss more than 1000 mL after any delivery.
- More robust studies are needed to account for the mechanism in which blood type is associated with increased risk of bleeding.

Table 1.

Maternal, gestational and intrapartum characteristics of our population

	Type-O (n= 40,964)	non Type-O (n=40,964)	*P*
Maternal Characteristics			
Maternal age (years)	27.9 ± 6.5	27.9 ± 6.5	.81
Rhesus positive	37,032 (90.4%)	37,009 (90.4%)	.78
Advanced maternal age (>35 years old)	6,564 (16.0%)	6,540 (16.0%)	.82
Married	20,027 (48.9%)	20,053 (49.0%)	.86
Race			
African-American	6,440 (15.7%)	6,361 (15.5%)	.45
White	22,493 (54.9%)	22,455 (54.8%)	.79
Prior cesarean delivery			.83
0	37,802 (92.3%)	37,832 (92.3%)	
1	1,910 (4.6%)	1,860 (4.5%)	
2	871 (2.1%)	881 (2.2%)	
3 or more	381 (1.0%)	391 (1.0%)	
Parity			.34
0	7,949 (19.4%)	7,925 (19.4%)	
1	21,407 (52.2%)	21,410 (52.2%)	
2	5,655 (13.8%)	5,804 (14.2%)	
3 or more	5,953 (14.6%)	5,825 (14.2%)	
Diabetes mellitus	1,200 (2.9%)	1,211 (3.0%)	.82
Body mass index (kg/m2)	31.8 ± 6.4	31.7 ± 6.5	.87
Gestational Characteristics			
Interpregnancy interval <1 year	682 (1.7%)	681 (1.7%)	.99
Multifetal gestation	490 (1.2%)	489 (1.2%)	.97
Preoperative hematocrit	35.1 ± 3.5	35.1 ± 3.5	.93
Predelivery Anemia (hematocrit<32%)	7,420 (18.1%)	7,398 (18.1%)	.84
Predelivery Thrombocytopenia (platelet < $150x10^3$)	4,260 (10.4%)	4,256 (10.4%)	.96
Predelivery white blood cell count ($x10^3/mm^3$)	10.5 ± 3.0	10.5 ± 3.1	.63
Placenta previa	274 (0.7%)	267 (0.7%)	.76
Placental abruption	317 (0.8%)	314 (0.8%)	.90
Placenta accreta/increta/percreta	31 (0.08%)	27 (0.07%)	.60
Diabetes mellitus (gestational)	2,527 (6.2%)	2,540 (6.2%)	.85
Gestational hypertension/preeclampsia	1,183 (2.9%)	1,150 (2.8%)	.49
History of Postpartum Hemorrhage	814 (2.0%)	767 (1.9%)	.23
Intrapartum Characteristics			
Gestational age (weeks)	38.4 ± 2.9	38.4 ± 2.8	.89
Preterm (<37 weeks)	3,946 (9.6%)	4,033 (9.9%)	.31

	Type-O (n= 40,964)	**non Type-O (n=40,964)**	***P***
Spontaneous vaginal delivery	24,567 (60.0%)	24,594 (60.0%)	.85
Assisted or operative vaginal delivery			
Vacuum	2,474 (6.0%)	2,480 (6.1%)	.93
Forceps	367 (0.9%)	365 (0.9%)	.94
Chorioamnionitis	367 (0.9%)	376 (0.9%)	.74
Any prior cesarean delivery	3,162 (7.7%)	3,132 (7.7%)	.69
1st cesarean delivery	12,366 (30.2%)	12,362 (30.1%)	.80
Repeat cesarean (N=30,496)	2,895 (7.1%)	2,873 (7.0%)	.80

Table 2.

Comparison Hematocrit drop among patients by mode of delivery.

	Vaginal			CS			All*		
Hematocrit absolute drop (%)‡	**Type-O (N=20,752)**	**Non Type-O (N=20,692)**	***P***	**Type-O (N=13,083)**	**Non Type-O(N=13,161)**	***P***	**Type-O (N=40,964)**	**Non Type-O (N=40,964)**	***P***
0% to <5%	13,311 (64.2%)	13,243 (64.0%)	.76	6786 (51.9%)	6994 (53.1%)	.08	20,097 (59.4%)	20,237 (59.8%)	.24
5% to <10%	6,663 (32.2%)	6,658 (32.2%)		5531 (42.3%)	5,379 (40.9%)		12,194 (36.1%)	12,037 (35.6%)	
10% to <15%	721 (3.5%)	743 (3.6%)		710 (5.4%)	742 (5.6%)		1,431 (4.2%)	1,485 (4.3%)	
≥15%	57 (0.1%)	48 (0.2%)		56 (0.4%)	46 (0.4%)		113 (0.3%)	94 (0.3%)	

‡Hematocrit drop was missing for 8130 Type-O and 7340 non Type-O patients.

*missing 14,240 entries.

All data are reported as n (%)

Table 3.

Maternal Estimate blood loss (EBL) during delivery.

	EBL	Type-O	Non Type-O	*P*
Vaginal	≤ 500mL	24,436 (95.1%)	24,456 (95.05%)	.84
	>500mL to ≤1000mL	977 (3.8%)	967 (3.8%)	
	>1000mL to ≤2000mL	226 (0.9%)	232 (0.9%)	
	>2000mL	64 (0.3%)	74 (0.3%)	
Cesarean Section	≤ 500mL	3,913 (25.6%)	3695 (24.2%)	.03
	>500mL to ≤1000mL	9,791 (64.2%)	10,006 (65.7%)	
	>1000mL to ≤2000mL	1,392 (9.1%)	1379 (9.0%)	
	>2000mL	165 (1.1%)	165 (1.1%)	
All	≤ 500mL	28,349 (69.2%)	28,151 (68.7%)	.45
	>500mL to ≤1000mL	10,768 (26.3%)	10,973 (26.8%)	
	>1000mL to ≤2000mL	1,618 (3.9%)	1,611 (3.9%)	
	>2000mL	229 (0.6%)	229 (0.6)	

All data are reported as n (%)