HHS Public Access

Author manuscript

J Clin Neurosci. Author manuscript; available in PMC 2022 August 01.

Published in final edited form as:

J Clin Neurosci. 2021 August ; 90: 345–350. doi:10.1016/j.jocn.2021.06.023.

ABO blood groups do not predict progression of traumatic intracranial hemorrhage

Alyssa M. Tutunjian, MS, MPH[a], **Sandra S. Arabian, MBA**[b], **Jacqueline Paolino, MD**[c], **Elizabeth S. Wolfe, DHS, ATC**[b], **Eric J. Mahoney, MD**[b], **Horacio M. Hojman, MD**[b], **Benjamin P. Johnson, MD**[b], **Nikolay Bugaev, MD**[b]

[a]Tufts University School of Medicine, 145 Harrison Ave, Boston, MA 02111, United States of America

[b]Division of Trauma & Acute Care Surgery, Department of Surgery, Tufts Medical Center, 800 Washington St, #4488, Boston, MA 02111, United States of America

[c]Department of Surgery, Tufts Medical Center, 800 Washington St, Boston, MA 02111, United States of America

Abstract

ABO blood groups are associated with genetically predisposed variations in von Willebrand factor (VWF) resulting in higher risks of thrombotic events in non-O blood types and bleeding complications in blood type O. The role of ABO blood groups in progression of traumatic intracranial hemorrhage (TICH) is unknown. Given statistically lower VWF levels in blood type O in the general population, we hypothesized that blood type O patients have a higher risk of such progression. A retrospective review of adult trauma patients with isolated TICH admitted to a Level 1 trauma center over eight years was conducted. Patients were categorized with blood type O and non-O (types A, B, AB) delineation. The primary outcome was radiological progression of TICH during the first 24 hours. Secondary outcomes included surgical intervention after follow-up computed tomography (CT), complications, days on mechanical ventilation (DMV), intensive care unit (ICU) length of stay (LOS), hospital LOS, and mortality. Of 949 patients, 432 (45.5%) had blood type O. When comparing O and non-O groups, no significant differences were found in gender, age, race, admission vital signs, Glasgow Coma Scale, coagulation profile, TICH type, or Injury Severity Score. No difference in TICH progression was found between O and non-O groups: 73 (17%) vs 80 (15%), respectively, p=0.55. Blood type O mortality was 12 (3% vs. 23 (4%), p=0.174). Rate of TICH surgical intervention after follow-up CT, DMV, complications, and

Corresponding author: Nikolay Bugaev, MD, Division of Trauma & Acute Care Surgery, Department of Surgery, Tufts Medical Center, 800 Washington St, #4488, Boston, MA 02111, United States of America, Tel. 1-(617) 636-4488, Fax 1-(617) 636-8172, nbugaev@tuftsmedicalcenter.org.

CONFLICTS OF INTEREST

There are no conflicts of interest to disclose. There are no declarations of interest.

Declarations of interest: none

ICU and hospital LOS did not differ. No association between ABO blood types and radiological progression of TICH was identified.

Keywords

blood group antigens; von Willebrand factor; intracranial hemorrhage, traumatic; brain injuries, traumatic; radiologic progression

INTRODUCTION

Traumatic brain injury (TBI) is the most common cause of death and long-term disability in trauma patients [1]. According to the most recent data from the Centers for Disease Control and Prevention, approximately 2.87 million patients report to the Emergency Department (ED), are hospitalized, or die as a result of TBI yearly [2]. Prevention of secondary brain injury and traumatic intracranial hemorrhage (TICH) progression are the main goals of treatment for these patients [3]. Multiple factors have been associated with progression of TBI and worse clinical outcomes: systemic hypotension, hypoxia, presence of and larger size of midline shift on admission, elevated diastolic and mean arterial pressures on admission, bilateral location and higher initial volume of TICH, presence of multiple TICHs, presence of pre-existing conditions, older age, coagulopathy, lower initial Glasgow Coma Scale score (GCS), and higher Injury Severity Score (ISS) [4–6]. The most prevalent mechanism of injury in these trauma cases is a fall.

Von Willebrand Factor (VWF) is a known mediator of platelet aggregation and clot formation. Serum levels of VWF are highly genetically influenced, with ABO blood groups being one of the main determinants. It is well established that those with non-O type blood, on average, have 25% higher levels of VWF than those with blood type O [7–11]. Increased WVF levels in non-O type blood groups have been linked to a higher risk of thromboembolic events [8,10–13]. The association between ABO blood groups and bleeding tendency is not as clear, however. This has prompted further investigation into the relationship between lower levels of VWF in patients with blood type O and an increased tendency for bleeding in multiple pathological conditions. A meta-analysis by Dentali *et al.* [11] showed that blood type O was associated with a higher risk of bleeding complications in various non-trauma related conditions.

A literature search of manuscripts that addressed association between blood types, incidence and risk of progression of traumatic and non-traumatic brain bleeds, resulted in only one small cohort study evaluating an association between ABO groups and the clinical course of traumatic subdural hemorrhage [14]. Blood type A was found to be associated with higher risk of midline shift and postoperative seizures. The findings from this study, however, did not reflect a theoretical assumption in the higher risk of bleeding in the O blood group patients.

The aim of our study was to evaluate the association between ABO blood types and radiological progression of TICH in the first 24 hours after injury. We hypothesized that patients with blood type O would have a higher risk of such progression.

METHODS

This is a retrospective review of patients admitted to Tufts Medical Center, an American College of Surgeons verified Level 1 trauma center. The study included patients over a span of eight years, from February 22, 2007 to January 31, 2015. The Institutional Review Board granted the authors permission to perform this study.

Adult (age ≥15) trauma patients with isolated blunt TICH were included. TICHs were identified based on the International Classification of Diseases, Ninth Revision (ICD-9) within the following diagnosis ranges: 851, 852, and 853. Excluded patients consisted of those with multiple injuries, including traumatic hemorrhagic brain injuries with other substantial body region injury, identified by Abbreviated Injury Scale (AIS) score greater than two. The following variables were collected for all patients: demographics (age, gender, race), ABO blood type, preexisting comorbidities (per National Trauma Data Standard) [15], pre-existing anticoagulation therapy, admission vital signs, ISS, AIS, GCS, coagulation profiles, and blood components transfused. TICH types were classified as the following: subdural hemorrhage (SDH), subarachnoid hemorrhage (SAH), epidural hemorrhage (EDH), intraparenchymal hemorrhage (IPH), intraventricular hemorrhage (IVH). TICHs were also recorded as unilateral or bilateral in location. Only patients who underwent two computed tomography (CT) scans of the head during the first 24 hours were included (CT on admission and follow-up). The follow-up head CT scans were performed in all patients with a less then 24 hours history of trauma who were diagnosed with either subdural, epidural, or intraparenchymal hemorrhages, or isolated traumatic subarachnoid hemorrhages in patients on anticoagulation therapy. Patients who were taken to the operating room for surgical treatment of TICH after their admission CT scan were excluded from the study cohort.

Patients were categorized by their ABO blood group classification with an ultimate delineation between blood type O (study) and non-O (blood types A, AB, B) (control) study groups.

The primary outcome of the study was radiological progression of TICH, identified by radiologist interpretation of the admission and follow-up CT scans. The secondary outcomes included the need for surgical intervention for TICH after the follow-up CT, complications (per National Trauma Data Standard) [15], days on mechanical ventilation (DMV), intensive care unit (ICU) and hospital length of stay (LOS), and mortality.

Sample size power calculation.

Sample size power was computed using the normal approximation method. Results of the Dentali *et al.* [8] meta-analysis were used to hypothesize the difference between the study and control groups. Group sample sizes of 500 in the study group and 500 in control group achieved 93.674% power to detect a difference between the group proportions of 0.1100. The proportion in study group was assumed to be 0.5500 under the null hypothesis and 0.4400 under the alternative hypothesis. The proportion in the control group was 0.4400. The test statistic used was the two-sided Z-Test with pooled variance. The significance level of the test was 0.050.

Statistical Analysis

Univariate analyses were used to compare demographic and clinical characteristics between blood type O and non-O patients. Categorical variables were compared using chi-square tests, and continuous variables were compared using t-tests or Wilcoxon rank-sum tests, depending on their distribution. All statistical analysis was performed using Stata v16.1 (College Station, TX).

RESULTS

Cohort characteristics, blood type distribution

The study cohort included 949 patients, 432 (45.5%) of whom were identified as blood type O (Table 1). There were no significant differences in demographic characteristics between blood type O and non-O (Table 2). Overall, the study groups did not differ in the frequency of pre-existing conditions, except for a history of myocardial infarction, 1.06% vs 2.61%, p=0.04, and chronic respiratory conditions, 1.06% vs 0.19%, p=0.004.

The mechanisms of injury, admission vital signs, ISS, and head AIS did not differ significantly between O and non-O blood types (Table 2). On admission, the blood type O group had slightly higher partial thromboplastin times (PTT) (mean ± standard deviation: 28.1 ± 4.7 vs. 27.4 ± 5.2, p=0.03); however, other coagulation profile indicators were not significantly different between groups (Table 2).

The majority of patients in both the O and non-O group presented with SDH, followed by SAH and IPH (Table 3). The proportion of patients with bilateral TICH or presence of two or more TICH types did not differ between groups.

The proportion of patients with blood components administered was significantly lower in the O group (12.8% of Group O vs. 17.4% of Group non-O, p=0.015). The types of blood products were similar, with patients in both groups receiving platelets most often.

Outcomes

No difference in the risk of radiological progression of TICH was found between the O and non-O groups (17% vs. 15%, p=0.55). No differences between the study groups were found in any of the secondary outcomes: rate of surgical intervention for TICH after the follow-up CT, DMV, complications, ICU and hospital LOS, and mortality (Table 4).

DISCUSSION

This is the largest study to date that has evaluated the correlation between ABO blood groups and radiological progression of TICH. No difference in the radiological progression of TICH after the first 24 hours was found between patients with O and non-O blood types.

Our study evaluated the role of ABO blood groups and progression of TICH within the first 24 hours. Overall, 16% of patients had a documented radiological progression of TICH, and no differences in progression were found between type O and non-O blood groups. The study groups were comparable in terms of baseline demographics, rate of preexisting

conditions, and injury severity. The literature search resulted in only one study that evaluated a correlation between TICH and ABO blood groups. Through a retrospective review, the role of ABO blood groups in 100 patients with traumatic SDH was evaluated within a single institution [14]. Patients with blood type A had a significantly greater shift of midline brain structures on admission head CT and a higher risk of postoperative seizures. No differences in demographics or other clinical characteristics were reported between the patients with type A and patients with other ABO blood types that would explain the identified differences in midline shift and the risk of postoperative seizures.

The role of ABO blood types and non-traumatic brain bleeds has been evaluated in a limited number of studies, and with inconsistent results (Table 5). One report addressed a theoretical connection between ABO blood types and non-traumatic intracerebral hemorrhage (ICH). Dentali *et al.* [16] utilized multivariate analysis to identify risk factors for poor functional outcome through a multicenter retrospective review of 229 patients with spontaneous ICH. Blood type O was not found to be a predictor of post-discharge disability. Roh *et al.* [17] prospectively enrolled 272 patients with non-traumatic ICH and found that blood type B, in comparison to other ABO blood types, was associated with hemorrhagic expansion of ICH (OR 2.82; 95% CI 1.23–6.45). Based on these results, the authors suspected an association between ABO blood type and expansion of ICH. On the other hand, He *et al.* [18] retrospectively reviewed 210 patients with spontaneous ICH and found radiological progression of ICH within the first 48 hours in 41.7% of patients with blood type O in comparison to 18.7% in other ABO blood groups ($p<0.001$).

The tendency for bleeding complications in non-traumatic conditions have also been reported with contradicting results in patients with postpartum hemorrhage [21], LVAD patients [22], and patients undergoing abdominal surgeries [23].

Very few studies have investigated the association between ABO blood types and bleeding complications in the trauma population, and these studies demonstrate inconclusive results as well. The association of ABO blood groups with hemorrhage, thromboembolic events, and mortality was evaluated in a multicenter retrospective review of 1281 adults treated at three level 1 trauma centers in Germany [19]. The authors found no association between ABO blood types with either thromboembolic events, hemorrhage, or mortality. A retrospective observational study was conducted in two tertiary emergency critical care medical centers in Japan and included 901 patients with ISS>15 [20]. The overall and hemorrhage-related mortalities were significantly higher in patients with blood type O compared to other ABO blood groups. At the same time, packed red blood cell transfusions in the first 24 hours did not differ between blood groups. The authors concluded that further studies were needed to develop a therapeutic intervention to address the blood type O-related mortality.

The results of the present study did not support the hypothesis of a higher risk of TICH progression in those with blood type O. The theoretical assumption was that genetically predisposed lower levels of VWF in blood type O in the general population would present an increased risk for bleeding, but this did not lead to radiological expansion of TICH in our patients. One of the possible explanations to this finding is the recognition that ABO blood

types represent blood group phenotypes, not genotypes. For example, the individuals with blood type A can be either heterozygous AO or homozygous AA, with the latter genotype having higher level of VWF [24]. In the present study, we did not have information about genotypes and thus could not investigate the association between ABO genotypes and TICH progression. We also did not have information about serum VWF levels. It has been reported that 66% of variations of the level of VWF is genetically predisposed, with 30% of these variations explained by ABO blood groups [25].

We think that our study cohort is representative of typical patients with TICH. Analysis of the National Trauma Data Base of patients with TICH showed the similar cohort demographic characteristics and TICH distribution [26, 27]. This similarity allows us to suggest that the findings of our study can be generalized.

Our study has limitations inherent to its retrospective nature. One of the main limitations is related to the lack of volumetric measurements of TICH. We identified patients with radiological progression of TICH based on the subjective impression of a radiologist describing head CT changes, without explicit, accurate volumetric measurements. This subjectivity in CT interpretation could have been a source of a level of uncertainty. On the other hand, no statistically significant differences were found between the O and non-O patients in terms of clinical outcomes, such as a rate of complications, ICU LOS and hospital LOS, DMV, and mortality. These findings further confirmed the conclusion showing a lack of association between blood type O and progression of TICH.

CONCLUSION

In the population studied, mortality, complications, and hospital course were not significantly different. In TBI patients with subsequent hemorrhage, ABO blood type was not associated with TICH progression. Given the retrospective nature of our study, further investigations of the role of ABO blood type in TICH in long-term outcomes are warranted.

ACKNOWLEDGEMENTS

We would like to thank Leslie Richardson-Weber, Reuven Rabinovici, MD, and Janis L Breeze, MPH for their overall support in preparing this manuscript.

This study was supported by the National Center for Advancing Translational Sciences, National Institutes of Health, Award UL1TR002544.

REFERENCES

[1]. Shackford SR, Mackersie RC, Holbrook TL, Davis JW, Hollingsworth-Fridlund P, Hoyt DB, et al. The epidemiology of traumatic death: A population-based analysis. Arch Surg. 1993;128:571–5. [PubMed: 8489391]

[2]. Centers for Disease Control and Prevention, National Center for Injury Prevention and Control. TBI-related Emergency Department Visits, Hospitalizations, and Deaths (EDHDs) 2019 [Available from: https://www.cdc.gov/traumaticbraininjury/data/tbi-edhd.html].

[3]. Kurland D, Hong C, Aarabi B, Gerzanich V, Simard JM. Hemorrhagic progression of a contusion after traumatic brain injury: a review. J Neurotrauma. 2012;29(1):19–31. [PubMed: 21988198]

[4]. Bugaev N, Al-Hazmi M, Allcorn M, Arabian SS, Riesenburger R, Safain M, Burke S, Colangelo A, Rabinovici R. Blood pressure regulation to prevent progression of blunt traumatic intracranial hemorrhage in stable patients. Neurocrit Care. 2014 8;21(1):58–66. [PubMed: 24493080]

[5]. Powers AY, Pinto MB, Tang OY, Chen JS, Doberstein C, Asaad WF. Predicting mortality in traumatic intracranial hemorrhage. J Neurosurg. 2019 2 22:1–8.

[6]. Cepeda S, Gómez PA, Castaño-Leon AM, Martínez-Pérez R, Munarriz PM, Lagares A. Traumatic Intracerebral Hemorrhage: Risk Factors Associated with Progression. J Neurotrauma. 2015 8 15;32(16):1246–53. [PubMed: 25752340]

[7]. The ABO blood group. 2005. In: Dean L Blood Groups and Red Cell Antigens [Internet]. Bethesda (MD): National Center for Biotechnology Information (US)

[8]. Dentali F, Sironi AP, Ageno W, Crestani S, Franchini M. ABO blood group and vascular disease: an update. Semin Thromb Hemost. 2014;40(1):49–59. [PubMed: 24381150]

[9]. Franchini M, Capra F, Targher G, Montagnana M, Lippi G. Relationship between ABO blood group and von Willebrand factor levels: from biology to clinical implications. Thromb J. 2007;5:14. [PubMed: 17894864]

[10]. Franchini M, Lippi G. Relative Risks of Thrombosis and Bleeding in Different ABO Blood Groups. Semin Thromb Hemost. 2016;42(2):112–7. [PubMed: 26716497]

[11]. Dentali F, Sironi AP, Ageno W, Bonfanti C, Crestani S, Frattini F, et al. Relationship between ABO blood group and hemorrhage: a systematic literature review and meta-analysis. Semin Thromb Hemost. 2013;39(1):72–82. [PubMed: 23299820]

[12]. Muellner SK, Haut ER, Streiff MB, Holcomb JB, Cotton BA. ABO blood group as a potential risk factor for venous thromboembolism in acutely injured patients. Thromb Haemost. 2011;105(1):5–13. [PubMed: 21103665]

[13]. Iturbe T, Cornudella R, de Miguel R, Olave T, Moreno JA, Callen L, et al. Hypercoagulability state in hip and knee surgery: influence of ABO antigenic system and allogenic transfusion. Transfus Sci. 1999;20(1):17–20. [PubMed: 10621554]

[14]. Dubinski D, Won SY, Behmanesh B, Brawanski N, Geisen C, Seifert V, Senft C, Konczalla. The clinical relevance of ABO blood type in 100 patients with acute subdural hematoma. J. PLoS One. 2018 10 4;13(10):e0204331.

[15]. American College of Surgeons, Committee on Trauma. ACS NTDB National Trauma Data Standard: Data dictionary. 2014.

[16]. Dentali F, Pomero F, Annoni F, Giraudo AV, Maresca AM, Fenoglio L, et al. Role of ABO blood group as a prognostic factor in patients with spontaneous intracerebral hemorrhage. J Thromb Haemost. 2013;11(1):187–9. [PubMed: 23137307]

[17]. Roh D, Martin A, Sun CH, Eisenberger A, Boehme A, Elkind MSV, Pucci JU, Murthy S, Kamel H, Sansing L, Park S, Agarwal S, Connolly ES, Claassen J, Hod 2, Francis RO. ABO Blood Type and Hematoma Expansion After Intracerebral Hemorrhage: An Exploratory Analysis. Neurocrit Care. 2019 8;31(1):66–71. [PubMed: 30547310]

[18]. He Q, Zhou Y, Wang F1, Li B, Cheng Y, Xie ZY. Blood Type O Predicts Hematoma Expansion in Patients with Intracerebral Hemorrhage. J Stroke Cerebrovasc Dis. 2019 8;28(8):2311–2317. [PubMed: 31171459]

[19]. Hamsen Uwe, Nohl André, Baumann Andreas, Lefering Rolf, Boutakmant Laila, Waydhas Christian, Dudda Marcel, Armin Schildhauer Thomas, Jäger Marcus, and Wegner Alexander. The influence of ABO blood group on mortality in major trauma Orthop Rev (Pavia). 2019 12 2; 11(4): 8214. [PubMed: 31897279]

[20]. Takayama W, Endo A, Koguchi H, Sugimoto M, Murata K, Otomo Y. The impact of blood type O on mortalityofseveretraumapatients:aretrospectiveobservationalstudy.CritCare. 2018 5 2;22(1):100.

[21]. Kahr MK, Franke D, Brun R, Wisser J, Zimmermann R, Haslinger C. Blood group O: A novel risk factor for increased postpartum blood loss? Haemophilia. 2018 7;24(4):e207–e212. [PubMed: 29877601]

[22]. Sun GH, Bruguera C, Saadat S, et al. ABO Blood Group and Transfusions In the Intraoperative and Postoperative Period After LVAD Implantation. Journal of Cardiothoracic and Vascular Anesthesia. 2020 4;34(4):906–911. [PubMed: 31590941]

[23]. Schack A, Oreskov JO, Ekeloef S, Brodersen T, Ostrowski SR, Gögenur I, Burcharth J. The impact of blood type on transfusion after major emergency abdominal surgery. Transfusion. 2019 1;59(1):125–133. [PubMed: 30537035]

[24]. Jenkins PV, O'Donnell JS. ABO blood group determines plasma von Willebrand factor levels: a biologic function after all? Transfusion. 2006 10;46(10):1836–44. [PubMed: 17002642]

[25]. Orstavik KH, Magnus P, Reisner H, Berg K, Graham JB, Nance W. Factor VIII and factor IX in a twin population: evidence for a major effect of ABO locus on factor VIII level. Am J Hum Genet.1985, 37:89–101. [PubMed: 3919575]

[26]. Shpiner AC, Bugaev N, Riesenburger R, Ng I, Breeze JL, Arabian SS, Rabinovici R. The profile of blunt traumatic supratentorial cranial bleed types. J Clin Neurosci. 2019 1;59:79–83. [PubMed: 30420206]

[27]. Ng I, Bugaev N, Riesenburger R, Shpiner AC, Breeze JL, Arabian SS, Rabinovici R. The profile of blunt traumatic infratentorial cranial bleed types. J Clin Neurosci. 2019 2;60:58–62. [PubMed: 30342807]

HIGHLIGHTS

- The largest study investigating ABO blood groups and bleeding tendency in traumatic intracranial hemorrhage (TICH)
- Discovered no significant correlation between O and non-O blood groups and radiologic progression of TICH
- Hospital course, morbidity, and mortality were not different between O and non-O blood groups

Table 1.

Hematologic characteristics of included traumatic intracranial hemorrhage patients (n=949)

	A	B	AB	O
Blood type, n (%)	368 (38.8)	110 (12.6)	39 (4.1)	432 (45.5)

Table 2.

Demographics, admission and injury characteristics

	Blood Group		
	O (n=432)	**Non-O (n=517)**	**p-value**
Age, mean (SD)	64.5 (21.3)	63.6 (20.7)	0.52
Male, n (%)	237 (55)	314 (61)	0.07
White Race, n (%)	361 (84.5)	428 (83.6)	0.69
Comorbidities, median (IQR)	1 (2, 7)	1 (2, 7)	0.99
Mechanism of injury			
Fall, n (%)	357 (83)	418 (81)	0.48
Injury Severity Score, mean (SD)	17.0 (5.2)	16.9 (5.3)	0.74
Head Abbreviated Injury Scale *(missing 2)*, n (%)			
<3 or 9	3 (1)	2 (<1)	0.83
3	105 (24)	134 (26)	
4	262 (61)	305 (59)	
5	60 (14)	76 (15)	
Coagulation parameters			
Platelets (k/uL), mean (SD)	205.2 (92.6)	207.8 (97.3)	0.68
INR, mean (SD)	1.2 (0.5)	1.3 (0.7)	0.22
PTT (seconds), mean (SD)	28.1 (4.7)	27.4 (5.2)	0.03
Hg (g/dL), mean (SD)	13.0 (2.0)	12.8 (2.1)	0.08
Vital signs on ED arrival			
Hypotension, SBP <90 mmHg, n (%) *(missing 31)*	4 (1)	11 (2)	0.14
DBP mmHg, mean (SD)	81 (17)	81 (17)	0.67
MAP mmHg, mean (SD)	99.7 (21.3)	99.2 (22.6)	0.693
Heart Rate >100 bpm, n (%) *(missing 25)*	78 (18.5)	79 (15.7)	0.27
Admission GCS <15, n (%) *(missing 36)*	127 (31)	151 (30)	0.93
Patients transfused with blood products, n (%)			
FFP	48 (11)	72 (14)	0.19
PLT	84 (19)	126 (24)	0.07
PRBC	13 (3)	29 (6)	0.05
AFVII	3 (1)	10 (2)	0.10

SD standard deviation; IQR, interquartile range; INR, International Normalized Ratio; PTT, partial thromboplastin time; Hg, hemoglobin; ED, emergency department; SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; GCS, Glasgow Coma Scale; FFP, fresh frozen plasma; PLT, platelets; PRBC, packed red blood cells; AFVII, activated factor seven.

Table 3.

Traumatic intracranial hemorrhage characteristics

	Blood Group		
	O (n=432)	**Non-O (n=517)**	**p-value**
TICH bleed types			
Subdural hemorrhage, n (%)	262 (61)	307 (59)	0.69
Subarachnoid hemorrhage, n (%)	182 (42)	245 (47)	0.11
Intraparenchymal hemorrhage, n (%)	123 (28)	134 (26)	0.39
Intraventricular hemorrhage, n (%)	24 (6)	20 (4)	0.22
Epidural hemorrhage, n (%)	6 (1)	10 (2)	0.52
TICH characteristics			
Midline shift (mm), median(IQR)	0 (0, 0)	0 (0, 0)	0.604
Two or more TICH Bleed types, n (%)	131 (30)	169 (33)	0.44
Bilateral TICH, n (%)	112 (26)	145 (28)	0.46
Surgery after 2nd CT, n (%)	9 (2)	15 (3)	0.42

TICH, traumatic intracranial hemorrhage; CT, computed tomography.

Table 4.

Outcomes

	Blood Group				
	O	**Non-O**			**p-value**
Bleeding progression on 2nd CT, n (%)	73 (17)	80 (15.5)			0.55
		A	AB	B	0.28
		55(10.6)	3(0.6)	22 (4.3)	
Complications, median (IQR)	0 (0, 0)	0 (0, 0)			0.56
Discharged home *, n (%)	191 (45.5)	240 (49)			0.35
ICU LOS * (n=914)	1 (1, 3)	1 (1, 2)			0.91
Days on Mechanical Ventilation * median (IQR)	0 (0, 0)	0 (0, 0)			0.10
Hospital LOS, median (IQR)	3 (1.5, 5)	3 (2, 6)			0.87
Mortality, n (%)	12 (3)	23 (4)			0.17

* among those who survived to discharge

IQR, interquartile range; ICU, intensive care unit; LOS, length of stay; A, AB, B: blood types

Table 5.

Summary of literature

A. ABO and brain hemorrhage					
Study	**ICH Mechanism**	**Hemorrhage Type**	**Study Size (n)**	**ABO-Bleeding Relationship Identified?**	**Key Findings**
Dubinski[14]	Traumatic	SDH	100	Yes *	Patients with blood type O had experienced fewer cases of midline shift and postoperative seizure.
Dentali[16]	Non-traumatic	ICH	229	No	Blood type O was not found to be a predictor of post-discharge disability or clinical outcomes.
Roh[17]	Non-traumatic	ICH	272	Yes	Type B blood was associated with more hemorrhagic expansion of ICH, defined as > 33% expansion.
He[18]	Non-traumatic	ICH	210	Yes	Hematoma expansion was more common in patients with blood type O and was an independent predictor of radiological progression.
B. ABO and bleeding/coagulation tendency					
Study	**Study type**	**Trauma population**	**Relationship Investigated:**	**Relationship Identified?**	**Key Findings**
Dentali[8]	Meta-analysis 45 studies	No	Non-O blood type and vascular thromboembolism	Yes	Weak positive association between non-O blood group and vascular arterial thrombosis, particularly myocardial infarction
Franchini[9]	Literature review	No	ABO blood group and vWF levels	Yes	Genetic, including ABO type, play a role in the variation of plasma vWF levels. Consistent reporting of lower vWF in blood type O patients.
Franchini[10]	Systematic review	No	ABO blood group and bleeding and thromboembolism	Yes	Large evidence to conclude non-O blood type is associated with increased risk of venous thromboembolism, but less evidence in support of arterial thromboembolism. Inconclusive evidence regarding blood type O and hemorrhagic risk.
Dentali[11]	Systematic review, Meta-analysis, 22 studies	No	ABO blood group and bleeding risk	Yes	A significant association between blood group O and bleeding risk was observed.
Muellner[12]	Literature review	Yes	ABO blood group and venous thromboembolism risk in the acutely injured patient	Yes	Within the ABO blood groupings, A1 and B alleles have an increased risk for VTE A2, O1, O2 allele patients have a decreased risk. Trauma patients may benefit from increased screening and subsequent prophylaxis.
Iturbe[13]	Retrospective cohort, 79 patients	No	Markers of hypercoagulability, including ABO type and transfusion, in patients undergoing surgery of the hip and knee	Yes	Non-O blood group had a higher circulating level of a prothrombin fragment, a marker for hypercoagulability.
Hamsen[19]	Retrospective cohort, 1281 patients	Yes	ABO blood group and hemorrhagic and thromboembolic events in considerably injured patients	No	No significant influence was found between ABO blood groups on hemorrhage, thromboembolic events and mortality.

Takayama[20]	Retrospective cohort, 901 patients	Yes	ABO blood type on mortality in severe trauma	Yes	Blood Type O was a significant, independent risk factor for all-cause in-hospital mortality and death due to exsanguination.
Kahr[21]	Prospective cohort, 1487 patients	No	Women with blood group O and postpartum bleeding	Yes	There was a significant increase in blood loss after delivery in women with blood group O.
Sun[22]	Retrospective cohort, 116 patients	No	Blood group O patients and perioperative transfusion requirements, postoperative chest tube output, and postoperative changes in hematocrit in the setting of left ventricular assist device (LVAD) placement	No	There was no significant different between blood group O and non-O in transfusion rates, chest tube output, or hematocrit changes in the intra- and postoperative periods.
Schack[23]	Retrospective cohort, 869 patients	No	Blood type O and transfusion rates in patients after major abdominal surgery	No	There was no significant difference in need for transfusion between blood type 0 and non-O patients. No significant difference in hemoglobin levels pre- and postop between the groups were identified either.
Jenkins[24]	Review	No	Structure, proteolysis, and clearance of VWF, in the setting of ABO blood group antigen expression	Yes	Blood group genotype and phenotype are key determinants of plasma VWF levels: Blood type O, phenotype O^1O^1, had the lowest levels of VWF. Heterozygous genotypes, including A^1O^1, had lower levels of VWF than those not carrying an O^1 allele.
Orstavik[25]	Cross-sectional, 74 monozygotic and 84 like-sexed dizygotic twin pairs	No	ABO locus on Factor VIII and Factor IX levels in twin population	Yes	Type O patients had lowest levels of Factor VIII (Type A_1 and B had the highest). 30% of the genetic variance in Factor VIII is due to ABO blood type. No effect was found on Factor IX.

* Authors suggested this could be due to faster clot liquidation, bias of anticoagulation therapy, and hemostasis-related interventions.