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ORIGINAL RESEARCH

Risk Factors and Neurologic Outcomes in Patients with Traumatic Brain Injury and Coagulopathy Within 72 h After Surgery

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Objective: The purpose of this study was to explore the effect of coagulopathy in patients with traumatic brain injury (TBI) during the early postoperative period.

Methods: The baseline characteristics, intraoperative management, and follow-up data of 462 patients with TBI between January 2015 and June 2019 were collected and retrospectively analyzed by multivariate logistic regression. Coagulopathy was defined as activated partial thromboplastin time > 40 s, international normalized ratio > 1.4, or platelet counts < 100×10^9/L.

Results: Multivariate logistic regression analysis revealed that the Glasgow Coma Scale (GCS) on admission, Injury Severity Score (ISS) on admission, pupil mydriasis, duration of surgery, intraoperative blood loss, and intraoperative crystalloid resuscitation were independent risk factors for patients who developed coagulopathy after surgery. There were statistical differences in mortality ($p = 0.049$), the Glasgow Outcome Scale-Extended (GCS-E; $p = 0.024$), and the modified Rankin Scale ($p = 0.043$) between the patients with and without coagulopathy 1 week after surgery. Coagulopathy within 72 h after surgery revealed the higher mortality at 1 week (66.7%), 3 months (71.4%), and 6 months (76.2%). Coagulopathy within 72 h after surgery in patients with a TBI predicted worse disease progression and unfavorable neurologic outcomes.

Conclusion: Taking practical and reasonable measures to manage these risk factors may protect patients with TBI from postoperative coagulopathy.

Keywords: traumatic brain injury, postoperative coagulopathy, surgery, risk factor, mortality

Introduction

The prevalence of coagulopathy is 7–63% in patients with traumatic brain injury (TBI) and >60% in patients with severe TBI on admission.[1,2] The overall mortality of TBI-associated coagulopathy is 17%–86%, and approximately 34% occur within 24 h after the injury.[3–5] The risks of hypocoagulopathy is associated with prolonged bleeding or progression of hemorrhagic lesions, and this pathologic phenomenon may persist at least 48 h after injury.[6]

In the early stage of trauma, multiple system functions, such as coagulation, anticoagulation and fibrinolysis are unbalanced due to tissue injury, hypoperfusion, systemic inflammatory reaction and other factors, which makes it difficult for the body to maintain normal hemostatic function, resulting in coagulopathy.[3,7] Coagulopathy is an important reason for the high incidence and progression of secondary injury caused by trauma, such as secondary intracranial hemorrhage,

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secondary cerebral infarction, deep vein thrombosis and discrete intravascular coagulation.[8,9]

It has been reported that the injury can induce a massive release of tissue factor into the systemic circulation, which leads to the activation of the extrinsic coagulation pathway. Moreover, platelet dysfunction, endogenous anticoagulation, endothelial activation, fibrinogen modification, inflammation, and hyperfibrinolysis can elicit increased and potentially severe bleeding,[10,11] which plays a critical role in coagulopathy after TBI.[8,12–14] There are few studies that have focused on the effect of coagulopathy on patients with TBI during the early postoperative period. Therefore, the purpose of the present study was to explore the risk factors and neurologic outcomes of coagulopathy in patients with TBI within 72 h after surgery.

Subjects and Methods

Patient Population

This study was approved by the Ethics Committee of the Second Affiliated Hospital of Air Force Medical University (20194651). All patients or their legal guardians signed the informed consent. A total of 462 patients with TBIs who were treated in the Trauma Center of the Second Affiliated Hospital of Air Force Medical University between January 2015 and June 2019 were enrolled in this retrospective study. The clinical data and follow-up data of the patients were collected and retrospectively analyzed. Among the 462 patients, 143 developed coagulopathy within 72 h after surgery. Inclusion criteria: 18–70 years of age; Glasgow Coma Scale (GCS) ≤ 8; Abbreviated Injury Scale (AIS) head ≥ 3; extracranial AIS < 3; and craniectomy without preoperative coagulopathy. Exclusion criteria: isolated penetrating head injury; multiple-organ failure; time from injury to surgery >12 h; pregnancy; intravenous fluids or blood > 2000 mL before enrollment; preoperative coagulopathy; and craniotomy before admission.

Definitions

The indication for decompressive craniectomy was based on the 4th edition of TBI guidelines.[15] All surgeries were performed by an associate chief surgeon with 12 years of experience. A head computed tomography (CT) scan was obtained at 0 h, 24 h, 72 h, and 5 days after surgery. An expanding contusion was diagnosed by comparison with the first head CT examination after surgery. Specifically, a follow-up CT scan that showed new lesions or increase in the original size of abnormalities > 33% or 12.5 mL was considered to signify an expanding contusion.[16,17]

The patients underwent coagulation testing at 2 h, 24 h, and 72 h after surgery. Coagulopathy was defined as an activated partial thromboplastin time (APTT) > 40 s, an international normalized ratio (INR) > 1.4, or platelet counts < 100×10^9/L. Standard treatment of coagulopathy was generally based on the administration of the following: tranexamic acid [10–20 mg/kg] within 3 h after the injury; red blood cells, plasma, and platelets in a 1:1:1 ratio; fresh frozen plasma [10–20 mL/kg]; platelets [5 mL/kg]; fibrinogen concentrate [30–50 mg/kg]; or cryoprecipitate [5–10 mL/kg].[3]

Neurologic Outcome Assessment

The neurologic outcomes were evaluated at 1 week, 3 months, and 6 months after surgery using mortality, the Glasgow Outcome Scale-Extended (GOS-E), and modified Rankin scale (mRS).

Statistical Analysis

SPSS 20.0 (SPSS, Inc., Chicago, IL, USA) and MedCalc (version 19.0.4; MedCalc, Inc., Mariakerke, Belgium) were used for statistical analysis. Continuous data with normal distribution are represented as the means ± standard deviation (SD) and analyzed by Student's *t*-test for normal distributions. Continuous data with non-normal distribution are described as median (IQR) and analyzed using the non-parametric rank-sum test (Wilcoxon-W test). Categorical data are presented as frequencies and analyzed by a Chi-square test or Fisher's exact test. Variables with $p < 0.05$ were entered into multivariate regression analysis (F-to-enter set at zero) to identify the risk factors for coagulopathy and mortality in patients with postoperative TBI. To determine the area under the curve (AUC) for predicting coagulopathy in patients with TBI in the early postoperative period, we performed receiver operating characteristic curve analysis. The difference between the AUC values was compared using parametric Z test. A p-value <0.05 was considered statistically significant, and p values for multiple comparisons were adjusted using the Holm–Bonferroni correction.

Results

In the cohort of 462 patients with TBI, the incidence of coagulopathy within 72 h after surgery was 30.9%. Patients with coagulopathy had significantly lower GCS on admission ($p < 0.001$), higher ISS on admission ($p = 0.006$), unilateral mydriasis ($p < 0.001$), bilateral mydriasis ($p = 0.005$), duration of surgery ($p = 0.046$), contusion expansion within 24 h after surgery ($p = 0.014$), length of hospital stay ($p < 0.001$), intraoperative blood loss ($p < 0.001$), and an increased

requirement for intraoperative crystalloid resuscitation ($p < 0.001$) than patients without coagulopathy after surgery (Tables 1 and 2).

Multivariate logistic regression analysis revealed the association between patients with coagulopathy after surgery and GCS on admission (OR = 0.748; 95% CI = 0.647–0.866; $p < 0.001$), ISS on admission (OR = 1.058; 95% CI = 1.016–1.102; $p = 0.007$), unilateral pupil mydriasis (OR = 3.405; 95% CI = 2.032–5.703; $p < 0.001$), bilateral pupil mydriasis (OR = 4.947; 95%

Table 1 Baseline Characteristics

Items	Without Coagulopathy (N=319)	Coagulopathy (N=143)	*P*
Male (n, %)	242 (75.9)	102 (71.3)	0.302
Age (years)	49.2 ± 15.3	52.0 ± 13.9	0.066
GCS on admission	6.5 ± 1.4	5.9 ± 1.2	< 0.001
AIS on admission	3.1 ± 0.6	3.2 ± 0.7	0.691
ISS on admission	15.1 ± 4.8	16.5 ± 4.7	0.006
Time from injury to hospital admission (h)	8.1 ± 3.7	7.7 ± 2.9	0.284
Time from injury to operation (h)	11.0 ± 4.3	11.7 ± 3.7	0.083
PH on admission	7.3 ± 0.2	7.3 ± 0.2	0.419
$HCO3^-$ on admission	26.8 ± 2.3	27.2 ± 2.7	0.057
Lactate on admission	2.5 ± 1.1	2.5 ± 1.4	0.870
Pupil size (n, %)			< 0.001
Normal	279 (87.5)	94 (65.7)	< 0.001
Unilateral mydriasis	34 (10.7)	39 (27.3)	< 0.001
Bilateral mydriasis	6 (1.9)	10 (7.0)	0.005
Trauma mechanism (n, %)			0.746
Violence	79 (24.8)	27 (18.9)	
Traffic accident	140 (43.9)	67 (46.9)	
Pedestrian	30 (9.4)	17 (11.9)	
Fall ≤ 3 m	35 (11.0)	16 (11.2)	
Fall>3 m	27 (8.5)	11 (7.7)	
Others	8 (2.5)	5 (3.5)	
Brain injury on initial CT (n, %)			0.318
EDH	103/458 (22.5)	64/235 (27.2)	
SDH	151/458 (33.0)	72/235 (30.6)	
DAI	27/458 (5.9)	10/235 (4.3)	
Brain contusion	147/458 (32.1)	80/235 (34.0)	
Diffuse brain swelling	30/458 (6.6)	9/235 (3.8)	
Type of trauma (n, %)			0.693
Isolated TBI	204 (63.9)	96 (67.1)	
TBI+ thoracic injury	69 (21.6)	32 (22.4)	
TBI+maxillofacial injury	24 (7.5)	7 (4.9)	
TBI+limb fracture	22 (6.9)	8 (5.6)	
Type of surgery (n, %)			0.85
Unilateral craniectomy	296 (92.8)	132 (92.3)	
Bilateral craniectomy	23 (7.2)	11 (7.7)	
Duration of operation (h)	3.1±1.4	3.4±1.3	0.046

(*Continued*)

Table 1 (Continued).

Items	Without Coagulopathy (N=319)	Coagulopathy (N=143)	*P*
Crystalloid resuscitation postoperation (mL)			
≤ 24 h	1800 (1700–2050)	1758 (1800–2000)	0.274
≤ 72 h	5560 (5000–6000)	5580 (5550–5900)	0.216
Contusion expansion postoperation (n, %)			0.007
< 24 h	46 (14.4)	34 (23.8)	
24 −72 h	11 (3.4)	9 (6.3)	
> 72 h	4 (1.3)	5 (2.8)	
Deep venous thrombosis postoperation (n, %)	18 (5.6)	7 (4.9)	0.743
Hospital lengths of stay (days)	9 (5, 15)	12 (9, 16)	< 0.001

Notes: Data are expressed as means ± SD, n(%) or median (IQR).
Abbreviations: GCS, Glasgow Coma Scale; AIS, Abbreviated Injury Scale; ISS, Injury Severity Score; EDH, extradural hematoma; SDH, subdural hematoma; DAI, diffuse axonal injury; SD, standard deviation; IQR, interquartile range.

Table 2 Intraoperative Fluid Management and Transfusion of Blood Components

Items	Without Coagulopathy (N=319)	Coagulopathy (N=143)	*P* value
Blood loss (mL)	600 (300–1000)	1200 (900–1500)	< 0.001
Urine loss (mL)	800 (500–1000)	700 (400–1000)	0.280
Crystalloid fluid (mL)	900 (1500–2000)	2500 (2000–3500)	< 0.001
Colloidal fluid (mL)	600 (500–1000)	500 (500–1000)	0.163
FFP transfusion (mL)	200 (0–400)	210 (0–400)	0.065
RBC transfusion (mL)	400 (0–800)	600 (0–1200)	0.103

Note: Data are expressed as median (IQR).
Abbreviations: FFP, fresh frozen plasma; RBC, red blood cell; IQR, interquartile range.

CI = 1.751–13.978; $p = 0.003$), duration of surgery (OR = 2.199; 95% CI = 1.853–2.610; $p < 0.001$), intraoperative blood loss (OR = 1.002; 95% CI = 1.001–1.002; $p < 0.001$), and intraoperative crystalloid resuscitation (OR = 1.004; 95% CI = 1.003–1.005; $p < 0.001$; Table 3).

Table 3 Multivariate Logistic Regression for Risk Factors of Coagulopathy in Patients with TBI After Surgery

Risk Factors	Odds Ratio	95% Confidence Interval	*P* value
GCS on admission	0.748	0.647–0.866	< 0.001
ISS on admission	1.058	1.016–1.102	0.007
Pupil size normal	1.000		< 0.001
Unilateral pupil mydriasis	3.405	2.032–5.703	< 0.001
Bilateral pupil mydriasis	4.947	1.751–13.978	0.003
Duration of operation	2.199	1.853–2.610	< 0.001
Intraoperative blood loss	1.002	1.001–1.002	< 0.001
Intraoperative crystalloid resuscitation	1.004	1.003–1.005	< 0.001

Abbreviations: GCS, Glasgow Coma Scale; ISS, Injury Severity Score.

There were statistical differences in mortality ($p = 0.049$), GOS-E ($p = 0.024$), and mRS ($p = 0.043$) between patients with coagulopathy and patients without coagulopathy 1 week after surgery (Table 4). Development of a coagulopathy within 72 h after surgery revealed a trend for higher mortality at 1 week (66.7%), 3 months (71.4%), and 6 months (76.2%), respectively (Table 5). Furthermore, univariate and multivariate analyses showed that coagulopathy within 72 h after surgery (OR = 2.438; 95% CI = 1.190–4.994; $p = 0.015$), contusion expansion within 24 h after surgery (OR = 16.643; 95% CI = 7.528–36.795; $p < 0.001$), contusion expansion between 24 and 72 h after surgery (OR = 8.365; 95% CI = 1.976–35.404; $p = 0.004$), and contusion expansion > 72 h after surgery (OR = 5.813; 95% CI = 2.025–16.684; $p = 0.001$) were the independent risk factors for mortality in patients with postoperative TBI (Tables 6 and 7).

The AUC value for predicting the incidence of postoperative coagulopathy was 0.719 (95% CI = 0.676–0.760; $p < 0.001$) with a cut-off of 6.5 for GCS on admission, 0.589 (95% CI = 0.535–0.644; $p = 0.002$) with a cut-off of 11.5 for ISS on admission, 0.815 (95% CI = 0.773–0.856; $p < 0.001$)

Table 4 The Neurologic Outcomes in Different Follow-Up Periods

Items	Without Coagulopathy (N=319)	With Coagulopathy (N=143)	*P* value
Mortality at one week (n, %)	24 (7.5)	19 (13.3)	0.049
GOS-E at one week	3.9 ± 1.7	3.5 ± 1.5	0.024
mRS at one week	3.7 ± 1.3	3.9 ± 1.2	0.043
Mortality at three months (n, %)	68 (21.3)	33 (23.1)	0.672
GOS-E at three months	5.1 ± 1.9	5.1 ± 1.9	0.681
mRS at three months	2.6 ± 1.5	2.7 ± 1.6	0.672
Mortality at six months (n, %)	82 (25.7)	40 (28.0)	0.609
GOS-E at six months	6.2 ± 2.0	6.3 ± 1.8	0.593
mRS at six months	1.9 ± 1.5	1.7 ± 1.6	0.455

Note: Data are expressed as means ± SD.
Abbreviations: GOS-E, Glasgow Outcome Scale-Extended; mRS, modified Rankin Scale.

Table 5 The Neurological Outcomes in Different Follow-Up Periods for Coagulopathy

Clinical Outcomes (n,%)	Mortality at 1 Week	Mortality at 3 Months	Mortality at 6 Months
Coagulopathy at 2 h (N=6)	0	1 (16.7)	2 (33.3)
Coagulopathy at 24 h (N=64)	0	2 (3.1)	4 (6.2)^{KLM}
Coagulopathy at 72 h (N=15)	1 (6.7)	3 (20.0)	5 (33.3)
Coagulopathy at 2+24 h (N=25)	1 (4.0)	4 (16.0)	7 (28.0)
Coagulopathy at 24+72 h (N=12)	3 (25.0)^{A}	4 (33.3)^{F}	6 (50.0)
Coagulopathy at 2+24+72 h (N=21)	14 (66.7)^{BCDE}	1571.4)^{GHI}	16 (76.2)^{NO}

Notes: Statistical significance of paired comparison based on Bonferroni adjusted α level, α=0.008. Mortality at one week: ^{A}Coagulopathy at 24 h+72 h vs Coagulopathy at 24 h (p = 0.003); ^{B}Coagulopathy at 2 h+24 h+72 h vs coagulopathy at 2 h (*p* = 0.006); ^{C}Coagulopathy at 2 h+24 h+72 h vs coagulopathy at 24 h (*p* < 0.001); ^{D}Coagulopathy at 2 h+24 h+72 h vs coagulopathy at 72 h (*p* < 0.001); ^{E}Coagulopathy at 2 h+24 h+72 h vs coagulopathy at 2h+24h (*p* < 0.001); Mortality at three months: ^{F}coagulopathy at 24 h +72 h vs coagulopathy at 24 h (*p* < 0.001); ^{G}Coagulopathy at 2 h+24 h+72 h vs coagulopathy at 24 h (*p* < 0.001); ^{H}Coagulopathy at 2 h+24 h+72 h vs coagulopathy at 72 h (*p* < 0.001); ^{I}Coagulopathy at 2 h+24 h+72 h vs coagulopathy at 2 h+24 h (*p* < 0.001); ^{J}Mortality at six months; ^{K}Coagulopathy at 24 h vs coagulopathy at 72 h (*p* < 0.001); ^{L}Coagulopathy at 24 h vs coagulopathy at 2 h+24 h (*p* < 0.001); ^{M}Coagulopathy at 24 h vs coagulopathy at 24 h+72 h (*p* < 0.001); ^{N}Coagulopathy at 24 h vs coagulopathy at 2 h+24 h+72 h (*p* < 0.001); ^{O}Coagulopathy at 2 h+24 h+72 h vs coagulopathy at 2 h+24 h (*p* < 0.001).

Table 6 Univariate Analysis of Non-Survived Patients with Postoperative TBI

Items	Non-Survived (N=43)	Survived (N=419)	*P* value
Postoperative coagulopathy (n, %)	19 (44.2)	124 (29.6)	0.049
GCS on admission	6.4 ± 1.3	6.6 ± 1.3	0.397
ISS on admission	15.3 ± 4.8	15.6 ± 4.8	0.217
Pupil size (n, %)			0.504
Normal	25 (58.1)	280 (66.8)	
Unilateral mydriasis	16 (37.2)	121 (28.9)	
Bilateral mydriasis	2 (4.7)	18 (4.3)	
Surgery duration (h)	3.1 ± 1.1	3.2 ± 1.4	0.592
Postoperative contusion expansion (n, %)			< 0.001
< 24 h	21 (48.8)	37 (8.8)	
24–72 h	6 (14.0)	31 (7.4)	
> 72 h	3 (7.0)	9 (2.1)	
Lengths of hospital stay (days)	4 (2–11)	11 (7–16)	
Intraoperative blood loss (mL)	700 (300–1200)	800 (300–1200)	0.938
Intraoperative crystalloid resuscitation (mL)	1400 (800–2200)	1100 (800–2000)	0.338

Notes: Data are expressed as means ± SD, n(%) or median (IQR).

Table 7 Multivariate Analysis of Non-Survived Patients with Postoperative TBI

Risk Factors	Odds Ratio	95% Confidence Interval	*P* value
Postoperative coagulopathy	2.438	1.190–4.994	0.015
Postoperative contusion expansion			< 0.001
None	1.000		
< 24 h	16.643	7.528–36.795	
24 −72 h	8.365	1.976–35.404	
> 72 h	5.813	2.025–16.684	

with a cut-off of 3.79 for the duration of surgery, 0.972 (95% CI = 0.957–0.986; $p < 0.001$) with a cut-off of 1650 mL for intraoperative crystalloid resuscitation, and 0.774 (95% CI = 0.732–0.817; $p < 0.001$) with a cut-off of 835 mL for intraoperative blood loss (Figure 1). There were statistical differences in the AUC values between intraoperative crystalloid resuscitation and GCS on admission (Z = 9.634, $p < 0.001$), ISS on admission (Z = 13.511, $p < 0.001$), duration of surgery (Z = 6.913, $p < 0.001$), and intraoperative blood loss (Z = 8.633, $p < 0.001$).

Discussion

Mounting evidence have identified the following independent risk factors for the development of acute coagulopathy after TBI: a GCS ≤ 8; pre-hospital intravenous fluid infusion ≥ 2000 mL; subarachnoid hemorrhage; and midline shift on CT imaging.[1,2] In addition, patients with penetrating TBI have higher incidence of coagulopathy and higher mortality rate than patients with closed craniocerebral injury.[18] Intravenous mannitol and hypertonic saline are routinely used to control intracranial hypertension in patients with severe TBI. However, a single bolus infusion of hypertonic fluids worsens hypocoagulability and hyperfibrinolysis in patients with hemorrhagic shock trauma.[4] The present study demonstrated that some risk factors, such as GCS on admission, ISS on admission, and abnormal pupil size, can be used to assess the severity of brain injury after trauma and predict the occurrence of coagulopathy for patients with TBI within 72 h after surgery. Notably, preoperative bilateral mydriasis was a strong predictor of coagulopathy. Nevertheless, for severe TBI patients with or without coagulopathy, despite undergoing decompressive craniectomy, there are high mortality and disability rates.

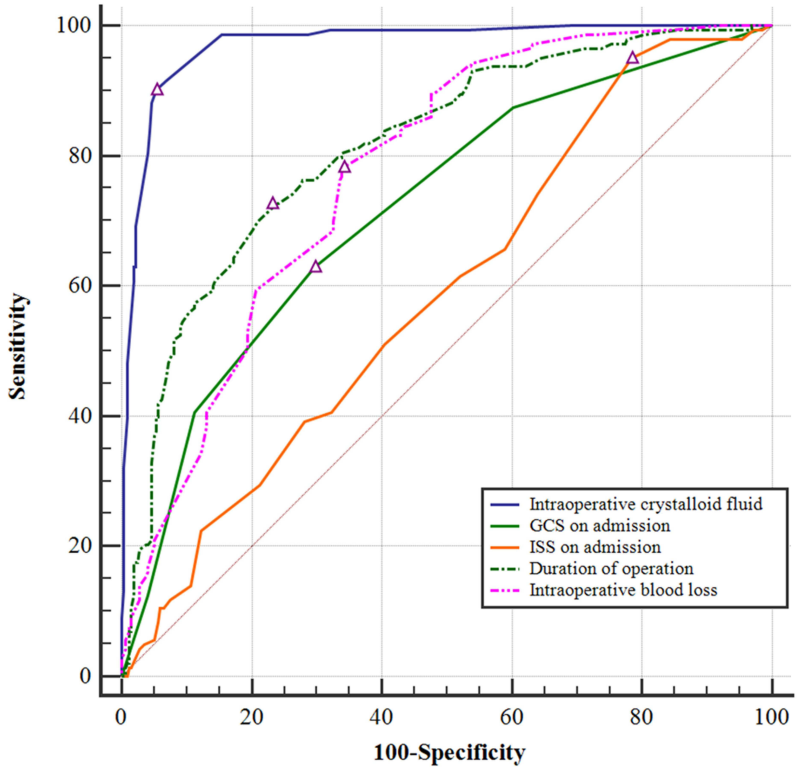

Figure 1 The AUC values for predicting the incidence of postoperative coagulopathy were 0.719 for GCS on admission, 0.589 for ISS on admission, 0.815 for duration of surgery, 0.972 for intraoperative crystalloid fluid volume, and 0.774 for intraoperative blood loss. [Δ]The cut-off value corresponding to Youden index.

In the setting of trauma or emergency surgery, intraoperative bleeding can be minimized with optimal preoperative preparation, but cannot be prevented completely. In this study, the duration of surgery (OR = 2.199) and intraoperative blood loss (OR = 1.002) were the independent risk factors for postoperative coagulopathy. It has been confirmed that shortening the duration of surgery, avoiding unnecessary blood loss, and reducing blood transfusion may help save the medical resources, reduce the medical costs, and decrease the mortality rate.[19,20]

The main goals of fluid therapy for patients with TBI are to optimize cerebral perfusion and maintain adequate cerebral oxygenation. The anesthesiologist may prefer rapid intraoperative fluid infusion to maintain blood pressure and cerebral blood flow stability when blood pressure decreases markedly after the induction of general anesthesia or relief of intracranial hypertension. Although we have confirmed that a large volume of intraoperative crystalloid resuscitation is an independent risk factor for patients with coagulopathy in the early postoperative period (OR = 1.004), there is still considerable controversy about fluid resuscitation for trauma patients. Shin et al[21] have reported that the volume of intraoperative fluid administration (900–1100 mL) is consistently associated with optimal 30-day mortality, respiratory complications, acute kidney injury, and postoperative length of stay in adults undergoing non-cardiac surgery. Hahn et al[22] have recommended the intraoperative administration of 3–5 mL/kg/h of crystalloids. However, additional fluid should be administered to patients who have more bleeding during surgery. Crystalloid resuscitation (>2000 mL) for patients with TBI is associated with increased mortality. Therefore, limited resuscitation before and after surgery may be indicated.[23,24]

A coagulopathy in TBI patients is strongly associated with progressive hemorrhagic injury. Approximately one-half of TBI patients with coagulopathy subsequently exhibit hemorrhagic progression of the initial brain contusions within 48 h.[1] There were statistical differences in contusion expansion within 24 h between patients who did and did not develop coagulopathy 1 week after surgery in our research. Furthermore, coagulopathy within 72 h after surgery and contusion expansion during the early postoperative period were the independent risk factors for non-survival of patients with TBI. The research results conclusively prove that coagulopathy alone and contusion expansion secondary to a coagulopathy may be associated with increased mortality.

At different onset times, coagulopathy could lead to different mortality rates, and coagulopathy with early onset after injury and long duration is a marker for increased morbidity and poor outcomes.[25] Carrick et al have reported that the incidence of coagulopathy is increased from 21% to 41% from the 1st to 3rd days in patients with TBI.[6] Finally, the mortality rate is 62%, and the length of stay is increased by 1 day in such patients. In agreement with Solla et al[26] and Yuan et al,[27] we reported that coagulopathy within 72 h after surgery portended a higher mortality rate. Therefore, it is essential to prevent coagulopathy and shorten the duration of the coagulopathy to improve clinical neurologic outcomes.

There are also some limitations in our study. First, retrospective clinical study has a significant selection bias that might influence the results. Second, these data, including osmotic/diuretic drugs, fluid resuscitation, severe hypoxia or asphyxia, and blood loss in the prehospital emergency care, are not uniformly available for us to incorporate into the subgroup analysis. Third, traditional blood coagulation tests, as the most commonly used method to detect coagulation abnormalities, do not provide the status of platelets, fibrinogen function, and fibrinolysis in coagulation cascades. Indeed, thromboelastography could play an essential role in accurately diagnosing platelet dysfunction, fibrinogen deficiency, and hyperfibrinolysis.

Conclusion

Among TBI patients who develop coagulopathy within 72 h after surgery, there may be worse disease progression and unfavorable neurologic outcomes in the early postoperative period. Moreover, coagulopathy in long duration after surgery is associated with high mortality rate at different postoperative periods. Hence, we should implement practical and reasonable measures to prevent and manage those risk factors, which may protect TBI patients from developing coagulopathy after surgery.

Abbreviations

TBI, traumatic brain injury; GCS, Glasgow Coma Scale; AIS, Abbreviated Injury Scale; APTT, activated partial thromboplastin time; INR, International Normalized Ratio; GOS-E, Glasgow Outcome Scale-Extended; mRS, modified Rankin Scale; IQR, interquartile range; ISS, Injury Severity Score.

Ethics Approval and Informed Consent

All procedures performed in the study were in accordance with the ethical standards and approved by the Medical Ethics Committee of the Second Affiliated Hospital of Air Force Medical University (Grant number: 20194651). This study was conducted in accordance with the Declaration of Helsinki.

Consent for Publication

We have obtained the informed consent from all patients or their legal guardians.

Author Contributions

All authors made substantial contributions to conception and design, acquisition of data, or analysis and interpretation of data; took part in drafting the article or revising it critically for important intellectual content; agreed to submit to the current journal; gave final approval of the version to be published; and agree to be accountable for all aspects of the work.

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Disclosure

Tao Chang, Xigang Yan, Chao Zhao are co-first authors for this study. The authors declare that they have no competing interests.

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