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Risk Factors and Neurologic Outcomes in Patients With Traumatic Brain Injury and Coagulopathy Within 72 Hours Post-operatively

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Abstract

Background There are few studies on the development and effect of coagulopathy in patients with a traumatic brain injury (TBI) during the early post-operative period. We determined the risk factors and neurologic outcomes of in patients with a TBI and coagulopathy diagnosed by routine laboratory tests within 72 hours post-operatively.

Methods The baseline characteristics, intra-operative management, and follow-up results of 462 patients with TBIs were obtained and retrospectively analyzed by multivariate logistic regression from January 2015 to June 2019. Coagulopathy was defined as an activated partial thromboplastin time > 40 seconds, international normalized ratio >1.4, or a platelet count < 100×10^9 /L.

Results Multivariate logistic regression analysis revealed that the Glasgow Coma Scale (GCS) at the time of admission, Injury Severity Score (ISS) at the time of admission, pupil mydriasis, duration of surgery, intra-operative blood loss, and intra-operative crystalloid resuscitation were independent risk factors for patients who developed a coagulopathy post-operatively. 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 patients with and without coagulopathy 1 week after surgery. 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%). Furthermore, coagulopathy and contusion expansion in the early post-operative period were independent risk factors for TBI mortality after surgery. Intra-operative crystalloid resuscitation had a substantial diagnostic accuracy in predicting coagulopathy within 72 h post-operatively (area under the curve [AUC] = 0.972).

Conclusion Coagulopathy within 72 h post-operatively in patients with a TBI predicted worse disease progression and unfavorable neurologic outcomes. Hence, we should take practical and reasonable measures to manage these risk factors, which may protect patients with a TBI from post-operative coagulopathy.

Introduction

The prevalence of coagulopathy in patients with a traumatic brain injury (TBI) on arrival at the emergency room is 7%-63% and > 60% in patients with a severe TBI [1, 2]. The overall mortality of TBI-associated coagulopathy is 17%-86%, approximately 34% occurring within 24 h after injury [3–5]. The main challenge for management is to address the risk of hypo-coagulopathy, which is associated with prolonged bleeding or progression of hemorrhagic lesions, and this pathologic phenomenon may persist at least 48 h after injury [6].

The injury can induce a massive release of tissue factor into the systemic circulation, which leads to the activation of the extrinsic coagulation pathway. In addition, platelet dysfunction, endogenous anticoagulation, endothelial activation, fibrinogen modification, inflammation, and hyperfibrinolysis can elicit increased and potentially severe bleeding [7, 8], and play a critical role in coagulopathy after a TBI.

There are few studies focusing on the development and effect of coagulopathy in patients with a TBI during the early post-operative period. The present study investigated the risk factors and neurologic outcomes of coagulopathy in these patients within 72 h post-operatively.

Methods

Patient population and definitions

The clinical data and follow-up results of 462 patients with TBIs were obtained and retrospectively analyzed at the Trauma Center of the Second Affiliated Hospital of Air Force Medical University from January 2015 to June 2019. Among the 462 patients, 143 developed aa coagulopathy within 72 h after surgery. The inclusion criteria for this study were as follows: 18–70 years of age; GCS ≤ 8; Abbreviated Injury Scale (AIS) head ≥ 3; extracranial AIS < 3; and craniectomy without a pre-operative coagulopathy. The exclusion criteria were as follows: isolated penetrating head injury; multiple organ failure; time from injury to surgery > 12 h; pregnancy; > 2000 ml of intravenous fluids or blood before enrollment; pre-operative coagulopathy; and craniotomy before admission.

The indication for decompressive craniectomy was based on the 4th edition of TBI guidelines [9]. All surgeries were performed by an associate chief surgeon with 12 years of experience. A head CT scan was usually obtained immediately after surgery, and 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 an increase in the original size of abnormalities > 33% or 12.5 ml was considered to signify an expanding contusion [10, 11].

The patients underwent coagulation testing 2, 24, and 72 h after surgery. Coagulopathy was defined as an activated partial thromboplastin time (APTT) > 40 seconds or an international normalized ratio (INR) > 1.4 or a platelet count < 100×10^9 /L. Standard treatment of coagulopathy was generally based on the administration of tranexamic acid (10–20 mg/kg) within 3 h after 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].

The Ethics Committee of the Second Affiliated Hospital of Air Force Medical University approved this study (20194651).

Neurologic outcome assessment

The neurologic outcomes were evaluated 1 week, 3 months, and 6 months after surgery using mortality, the Glasgow Outcome Scale-Extended (GOS-E), and the 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 are represented as the mean ± SD and analyzed by Student's t-test for normal distributions. Continuous data that have a non-normal distribution are described as the 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 Fischer's exact test. Variables with a $p < 0.05$ were entered into multivariate regression analysis (F-to-enter set at zero) to identify risk factors for coagulopathy and mortality in patients with a post-operative TBI. To determine the area under the curve (AUC) for predicting coagulopathy in patients with a TBI in the early post-operative period, we performed receiver operating characteristic curve analysis. The difference between the AUC values was compared using a 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 TBIs, the incidence of coagulopathy within 72 h post-operatively was 30.9%. Patients who developed a coagulopathy had a lower GCS on admission ($p < 0.001$), a 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 post-operatively ($p = 0.014$), length of hospital stay ($p < 0.001$), intra-operative blood loss ($p < 0.001$), and intra-operative crystalloid resuscitation ($p < 0.001$) than patients without a coagulopathy after surgery (Tables 1 & 2).

Table 1
Baseline characteristics.

	Without coagulopathy (N= 319)	Coagulopathy (N = 143)	*P*
Male (n, %)	242 (75.9)	102 (71.3)	0.302
Age (years), mean ± SD	49.2 ± 15.3	52.0 ± 13.9	0.066
GCS on admission, mean ± SD	6.5 ± 1.4	5.9 ± 1.2	< 0.001
AIS on admission, mean ± SD	3.1 ± 0.6	3.2 ± 0.7	0.691
ISS on admission, mean ± SD	15.1 ± 4.8	16.5 ± 4.7	0.006
Time from injury to hospital admission (hours), mean ± SD	8.1 ± 3.7	7.7 ± 2.9	0.284
Time from injury to operation (hours), mean ± SD	11.0 ± 4.3	11.7 ± 3.7	0.083
PH on admission, mean ± SD	7.3 ± 0.2	7.3 ± 0.2	0.419
$HCO3^-$on admission, mean ± SD	26.8 ± 2.3	27.2 ± 2.7	0.057
Lactate on admission, mean ± SD	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, %)			
Violence	79 (24.8)	27 (18.9)	
Traffic accident	140 (43.9)	67 (46.9)	
Pedestrian	30 (9.4)	17 (11.9)	0.746
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, %)			
EDH	103/458 (22.5)	64/235 (27.2)	
SDH	151/458 (33.0)	72/235 (30.6)	0.318
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)	

GCS, Glasgow Coma Scale; AIS, Abbreviated Injury Scale; ISS, Injury Severity Score; EDH, extradural hematoma; SDH, subdural hematoma; DAI, diffuse axonal injury; SD, standard standard; IQR, interquartile range.

	Without coagulopathy (N= 319)	Coagulopathy (N = 143)	*P*
Type of trauma (n, %)			0.693
Isolated TBI	204 (63.9)	96 (67.1)	0.507
TBI + thoracic injury	69 (21.6)	32 (22.4)	0.857
TBI + maxillofacial injury	24 (7.5)	7 (4.9)	0.297
TBI + limb fracture	22 (6.9)	8 (5.6)	0.600
Type of surgery (n, %)			0.854
Unilateral craniectomy	296 (92.8)	132 (92.3)	
Bilateral craniectomy	23 (7.2)	11 (7.7)	
Duration of operation (hours), mean ± SD	3.1 ± 1.4	3.4 ± 1.3	0.046
Crystalloid resuscitation postoperation (ml), median (IQR)			
≤ 24 hours	1800 (1700–2050)	1758 (1800–2000)	0.274
≤ 72 hours	5560 (5000–6000)	5580 (5550–5900)	0.216
Contusion expansion postoperation (n, %)			0.007
≤ 24 hours	46 (14.4)	34 (23.8)	0.014
24–72 hours	11 (3.4)	9 (6.3)	0.165
> 72 hours	4 (1.3)	5 (2.8)	0.107
Deep venous thrombosis postoperation (n, %)	18 (5.6)	7 (4.9)	0.743
Hospital lengths of stay (days), median (IQR)	9 (5, 15)	12 (9, 16)	< 0.001

GCS, Glasgow Coma Scale; AIS, Abbreviated Injury Scale; ISS, Injury Severity Score; EDH, extradural hematoma; SDH, subdural hematoma; DAI, diffuse axonal injury; SD, standard standard; IQR, interquartile range.

Table 2
Intraoperative fluid manegement and transfusion of blood components

	Without coagulopathy (N= 319)	Coagulopathy (N = 143)	*P* value
Blood loss (ml), median (IQR)	600 (300–1000)	1200 (900–1500)	< 0.001
Urine loss (ml), median (IQR)	800 (500–1000)	700 (400–1000)	0.280
Crystalloid fluid (ml), median (IQR)	900 (1500–2000)	2500 (2000–3500)	< 0.001
Colloidal fluid (ml), median (IQR)	600 (500–1000)	500 (500–1000)	0.163
FFP transfusion (ml), median (IQR)	200 (0–400)	210 (0–400)	0.065
RBC transfusion (ml), median (IQR)	400 (0–800)	600 (0–1200)	0.103
FFP, fresh frozen plasma; RBC, red blood cell; IQR, interquartile range.			

Multivariate logistic regression analysis revealed an association between patients who developed a coagulopathy post-operatively 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% CI = 1.751–13.978; p = 0.003), duration of surgery (OR = 2.199; 95% CI = 1.853–2.610; p < 0.001), intra-operative blood loss (OR = 1.002; 95% CI = 1.001–1.002; p < 0.001), and intra-operative 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
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 who developed a coagulopathy and without a coagulopathy 1 week after surgery (Table 4). Coagulopathy within 72 h post-operatively revealed a trend for higher mortality at 1 week (66.7%), 3 months (71.4%), and 6 months (76.2%; Table 5). Furthermore, univariate and multivariate analyses showed that a coagulopathy within 72 h post-operatively (OR = 2.438; 95% CI = 1.190–4.994; p = 0.015), contusion expansion within 24 h (OR = 16.643; 95% CI = 7.528–36.795;

p < 0.001), between 24 and 72 h (OR = 8.365; 95% CI = 1.976–35.404; p = 0.004), and > 72 h (OR = 5.813; 95% CI = 2.025–16.684; p = 0.001) were independent risk factors for mortality in patients with a TBI post-operatively (Tables 6 & 7).

Table 4
The neurologic outcomes in different follow-up periods

	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, mean ± SD	3.9 ± 1.7	3.5 ± 1.5	0.024
mRS at one week, mean ± SD	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, mean ± SD	5.1 ± 1.9	5.1 ± 1.9	0.681
mRS at three months, mean ± SD	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, mean ± SD	6.2 ± 2.0	6.3 ± 1.8	0.593
mRS at six months, mean ± SD	1.9 ± 1.5	1.7 ± 1.6	0.455
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,%)	Coagulopathy at 2 h (N = 6)	Coagulopathy at 24 h (N = 64)	Coagulopathy at 72 h (N = 15)	Coagulopathy at 2 h + 24 h (N = 25)	Coagulopathy at 24 h + 72 h (N = 12)	Coagulopathy at 2 h + 24 h + 72 h (N = 21)
Mortality at 1 week	0	0	1 (6.7)	1 (4.0)	3 (25.0)[A]	14 (66.7)[BCDE]
Mortality at 3 months	1 (16.7)	2 (3.1)	3 (20.0)	4 (16.0)	4 (33.3)[F]	15 (71.4)[GHI]
Mortality at 6 months	2 (33.3)	4 (6.2)[KLM]	5 (33.3)	7 (28.0)	6 (50.0)	16 (76.2)[NO]
Statistical significance of paired comparison based on Bonferroni adjusted α level, α = 0.008.						
Mortality at one week: A. Coagulopathy at 24h + 72 h vs. Coagulopathy at 24 h (p = 0.003)						
B. Coagulopathy at 2h + 24h + 72 h vs. coagulopathy at 2 h (p = 0.006)						
C. Coagulopathy at 2h + 24h + 72 h vs. coagulopathy at 24 h (p < 0.001)						
D. Coagulopathy at 2h + 24h + 72 h vs. coagulopathy at 72 h (p < 0.001)						
E. Coagulopathy at 2h + 24h + 72 h vs. coagulopathy at 2h + 24 h (p < 0.001)						
Mortality at three months: F. coagulopathy at 24h + 72 h vs. coagulopathy at 24 h (p < 0.001)						
G. Coagulopathy at 2h + 24h + 72 h vs. coagulopathy at 24 h (p < 0.001)						
H. Coagulopathy at 2h + 24h + 72 h vs. coagulopathy at 72 h (p < 0.001)						
I. Coagulopathy at 2h + 24h + 72 h vs. coagulopathy at 2h + 24 h (p < 0.001)						
Mortality at six months: K. coagulopathy at 24 h vs. coagulopathy at 72 h (p < 0.001)						
L. Coagulopathy at 24 h vs.vs.coagulopathy at 2h + 24 h (p < 0.001)						
M. Coagulopathy at 24 h vs.vs.coagulopathy at 24h + 72 h (p < 0.001)						
N. Coagulopathy at 24 h vs.coagulopathy at 2h + 24h + 72 h (p < 0.001)						
O. Coagulopathy at 2h + 24h + 72 h vs.coagulopathy at 2h + 24 h (p < 0.001)						

Table 6
Univariate analysis of non-survived patients with postoperative TBI

	Non-survived (N = 43)	Survived (N = 419)	*P* value
Postoperative coagulopathy (n, %)	19 (44.2)	124 (29.6)	0.049
GCS on admission, mean ± SD	6.4 ± 1.3	6.6 ± 1.3	0.397
ISS on admission, mean ± SD	15.3 ± 4.8	15.6 ± 4.8	0.217
Pupil size (n, %)			
Normal	25 (58.1)	280 (66.8)	0.504
Unilateral mydriasis	16 (37.2)	121 (28.9)	
Bilateral mydriasis	2 (4.7)	18 (4.3)	
Surgery duration (hours), mean ± SD	3.1 ± 1.1	3.2 ± 1.4	0.592
Postoperative contusion expansion (n, %)			< 0.001
≤ 24 hours	21 (48.8)	37 (8.8)	< 0.001
24–72 hours	6 (14.0)	31 (7.4)	0.132
> 72 hours	3 (7.0)	9 (2.1)	0.058
Lengths of hospital stay (days), median (IQR)	4 (2–11)	11 (7–16)	< 0.001
Intraoperative blood loss (ml), median (IQR)	700 (300–1200)	800 (300–1200)	0.938
Intraoperative crystalloid resuscitation (ml), median (IQR)	1400 (800–2200)	1100 (800–2000)	0.338

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			
None	1.000		< 0.001
≤ 24 hours	16.643	7.528–36.795	< 0.001
24–72 hours	8.365	1.976–35.404	0.004
> 72 hours	5.813	2.025–16.684	0.001

The AUC value for predicting the incidence of post-operative 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) with a cut-off of 3.79 for duration of surgery, 0.972 (95% CI

= 0.957–0.986; p < 0.001) with a cut-off of 1650 ml for intra-operative crystalloid resuscitation, and 0.774 (95% CI = 0.732–0.817; p < 0.001) with a cut-off of 835 ml for intra-operative blood loss (Fig. 1). There were statistical differences in the AUC values between intra-operative 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 intra-operative blood loss (Z = 8.633, p < 0.001).

Discussion

Mounting evidence has identified the following independent risk factors for the development of acute coagulopathy after a TBI, including a GCS ≤ 8, pre-hospital intravenous fluid infusion ≥ 2000 ml, subarachnoid hemorrhage, and a midline shift on CT images [1, 2]. In addition, patients with a penetrating TBI had a higher incidence of coagulopathy and a higher mortality rate than patients with a closed craniocerebral injury [12]. Intravenous mannitol and hypertonic saline are routinely used to control intracranial hypertension in patients with a severe TBI; however, the impact of a single bolus infusion of hypertonic fluids could worsen 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, could be used to assess the severity of brain injury after trauma and predict the occurrence of coagulopathy for patients with a TBI within 72 h post-operatively. Notably, bilateral mydriasis pre-operatively was a strong predictor. Nevertheless, whether complicated by a coagulopathy or not, patients with a severe TBI, pupil mydriasis, and no light reflex, despite undergoing decompressive craniectomy, could have high mortality and disability rates.

In the setting of trauma or emergency surgery, intra-operative bleeding can be minimized with optimal pre-operative preparation, but cannot be prevented completely. In this study, duration of surgery (OR = 2.199) and intra-operative blood loss (OR = 1.002) were independent risk factors for post-operative coagulopathy. It has been confirmed that shortening the duration of surgery, avoiding unnecessary blood loss, and reducing blood transfusion may help save medical resources, reduce medical costs, and decrease the mortality rate [13, 14].

The main goals of fluid therapy for patients with a TBI are to optimize cerebral perfusion and maintain adequate cerebral oxygenation. The anesthesiologist may prefer rapid intra-operative 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 this study confirmed that a large volume of intra-operative crystalloid resuscitation is an independent risk factor for patients who developed a coagulopathy in the early post-operative period (OR = 1.004), there is still considerable controversy about fluid resuscitation for trauma patients. Shin et al. [15] reported that the volume of intra-operative fluid administration (900–1100 ml) is consistently associated with optimal 30-day mortality, respiratory complications, acute kidney injury, and post-operative length of stay in adults undergoing non-cardiac surgery. Hahn et al. [16] recommended the intra-operative 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 a TBI is associated with increased mortality; thus, limited resuscitation before and after surgery may be indicated [17, 18].

A coagulopathy in patients with a TBI has been strongly associated with progressive hemorrhagic injury. Approximately one-half of TBI patients with a coagulopathy could subsequently exhibit hemorrhagic progression of the initial brain contusions within 48 h [1]. There were statistical differences in contusion expansion within 24 h (p = 0.014) between patients who did and did not develop a coagulopathy 1 week after surgery based on our research. Furthermore, a coagulopathy within 72 h post-operatively and contusion expansion during the early post-operative period were independent risk factors for non-survival of patients with a 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, a coagulopathy could lead to different mortality rates, and a coagulopathy with early onset after injury and long duration is a marker of increased morbidity and poor outcomes [19]. Carrick et al. [6] implied that coagulopathy increased from 21–41% from the 1st day to the 3rd day in patients with a TBI. Finally, the mortality rate was 62%, and the length of stay was increased by 1 day ($p = 0.14$) in such patients. In agreement with Solla et al. [21] and Corbett [22], we reported that coagulopathy within 72 h post-operatively may portend a higher mortality rate, whether 1 week, 3 months, or 6 months. Therefore, it is essential to prevent coagulopathy and shorten the duration of the coagulopathy to improve clinical neurologic outcomes.

We have been cautious in interpreting these findings because of the limitations in our study. First, a 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 pre-hospital emergency care, were 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, did 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 patients with a TBI who develop coagulopathy within 72 h post-operatively, worse disease progression and unfavorable neurologic outcomes are likely during the early post-operative period. Moreover, a coagulopathy of long duration after surgery is associated with a high mortality rate at different post-operative periods. Hence, we should implement practical and reasonable measures to prevent and manage risk factors, which may protect patients with a TBI from developing a coagulopathy post-operatively.

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

Declarations

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Availability of data and materials

No

Authors' contributions

T C collected the data and drafted the manuscript. YL Y, GQ F, and QB G revised the language and grammar of the manuscript. T C, Z Q, XX T, and QB G provided the clinical data and searched the literature. LH L conceived and designed the experiments. All authors read and agreed to the final manuscript.

Ethics approval and consent to participate

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).

Consent for publication

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

Competing interests

The authors declare that they have no competing interests.

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Figures

Figure 1

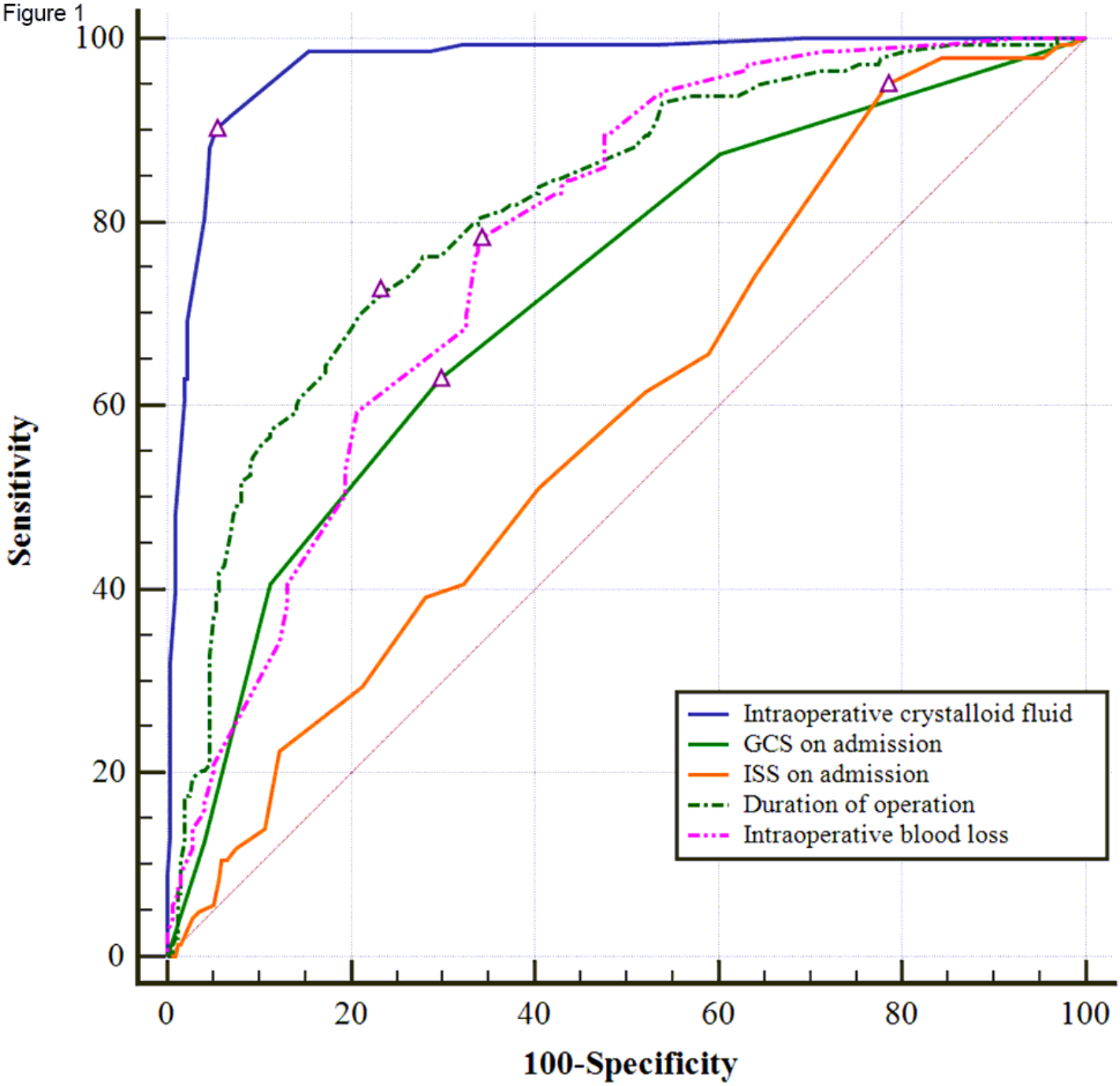

Figure 1

The AUC values for predicting incidence of coagulopathy postoperation were 0.719 for GCS on admission□0.589 for ISS on admission, 0.815 for duration of operation, 0.972 for intraoperative crystalloid fluid volume, and 0.774 for intraoperative blood loss. △, the cut-off value corresponding to Youden index.