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A PREDICTION MODEL FOR TRAUMA-INDUCED COAGULOPATHY BASED ON BLOOD GAS ANALYSIS AND COAGULATION FUNCTION: A RETROSPECTIVE STUDY

MODEL ZA PREDVIĐANJE TRAUMOM IZAZVANE KOAGULOPATIJE NA OSNOVU ANALIZE GASOVA U KRVI I PARAMETARA KOAGULACIJE: RETROSPEKTIVNA STUDIJA

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Summary

Background: There is little data regarding the method for predicting trauma-induced coagulopathy (TIC) in patients with severe trauma to facilitate early intervention. This study aimed to establish a prediction model for the early diagnosis of TIC in trauma patients.
Methods: This retrospective study included 214 patients with severe multiple injuries (injury severity score [ISS] ≥ 16) admitted to the Emergency Department of the First Affiliated Hospital of Zhejiang University School of Medicine between December 2020 and December 2022. Patients were divided into TIC (n=63) and non-TIC (n=151) groups based on an international normalised ratio (INR) >1.2 with active bleeding or need for blood products. Univariate analysis was performed, followed by multivariate logistic regression using SPSS 23.0 to identify independent risk factors and establish a prediction model. Model performance was evaluated by the receiver operating characteristic (ROC) curve, and a clinical application decision tree was constructed.
Results: Prothrombin time (PT) (OR=1.310, 95% CI: 1.043–1.645), fibrinogen (Fbg) (OR=0.254, 95% CI: 0.122–0.525), base excess (BE) (OR=0.752, 95% CI: 0.646–0.875), and LnD-dimer (OR=1.433, 95% CI:

Kratak sadržaj

Uvod: Postoji malo podataka o metodama za predviđanje traumom izazvane koagulopatije (TIC) kod pacijenata sa teškim traumama, koje bi omogućile ranu intervenciju. Cilj ove studije bio je da se uspostavi model predviđanja za ranu dijagnozu TIC-a kod pacijenata sa traumom.
Metode: Ova retrospektivna studija je obuhvatila 214 pacijenata sa teškim multiplim povredama (skor težine povrede [ISS]≥16), primljenih u Urgentni centar Prve pridružene bolnice Medicinskog fakulteta Univerziteta Zhejiang u periodu od decembra 2020. do decembra 2022. godine. Pacijenti su podeljeni u grupu sa TIC-om (n=63) i grupu bez TIC-a (n=151), na osnovu međunarodnog normalizovanog odnosa (INR) >1,2 uz aktivno krvarenje ili potrebu za primenom krvnih produkata. Sprovedena je univarijantna analiza, nakon čega je primenjena multivarijantna logistička regresija pomoću programa SPSS 23.0 radi identifikovanja nezavisnih faktora rizika i uspostavljanja modela predviđanja. Performanse modela procenjene su pomoću ROC krive, a konstruisano je i stablo odlučivanja za kliničku primenu.
Rezultati: Protrombinsko vreme (PT) (OR=1,310; 95% CI: 1,043–1,645), fibrinogen (Fbg) (OR=0,254; 95% CI: 0,122–0,525), bazni eksces (BE) (OR=0,752; 95%

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List of abbreviations: TIC, trauma-induced coagulopathy; CPM-TIC, clinical prediction model for TIC; Hb, haemoglobin; WBC, white blood cell; PLT, platelet count; PT, prothrombin time; APTT, activated partial thromboplastin time; INR, international normalised ratio; Fbg, fibrinogen; VHA, viscoelastic haemostasis analysis; BE, base excess; LA, lactic acid; ISS, injury severity score; ROC, receiver operating characteristic; AUC, area under the curve.

1.008–2.037) were identified as independent predictors of TIC. The clinical prediction model for TIC was formulated as: Model score = 0.27 × PT - 1.372 × Fbg + 0.36 × LnD-dimer - 0.285 × BE - 5.658. The model showed good discrimination, with an area under the curve (AUC) of 0.877 (95% CI: 0.827–0.928). At a cut-off value of -0.705, sensitivity and specificity were 76.2% and 88.3%, respectively. External validation in 99 patients confirmed a significantly higher TIC incidence in the high-risk group (score ≥-0.705) compared to the low-risk group (90.4% vs 25.6%, p<0.001).

Conclusion: Our study developed a prediction model to guide early effective interventions of TIC and improve the prognosis in severe trauma patients.

Keywords: prediction, model, coagulopathy, trauma

CI: 0,646–0,875) i LnD-dimer (OR=1,433; 95% CI: 1,008–2,037) su identifikovani kao nezavisni prediktori TIC-a. Klinički model za predviđanje TIC-a je formulisan na sledeći način: skor modela = 0,27 × PT - 1,372 × Fbg + 0,36 × LnD-dimer - 0,285 × BE - 5,658. Model je pokazao dobru diskriminativnu sposobnost, sa površinom ispod krive (AUC) od 0,877 (95% CI: 0,827–0,928). Pri graničnoj vrednosti od -0,705, senzitivnost je iznosila 76,2%, a specifičnost 88,3%. Eksterna validacija na 99 pacijenata potvrdila je značajno veću učestalost TIC-a u grupi visokog rizika (skor ≥-0,705) u poređenju sa grupom niskog rizika (90,4% prema 25,6%; p<0,001).

Zaključak: U našoj studiji razvijen je model predviđanja koji može usmeriti rane i efikasne intervencije kod TIC-a i poboljšati prognozu pacijenata sa teškim traumama.

Ključne reči: predviđanje, model, koagulopatija, trauma

Introduction

Trauma remains the fourth leading cause of death globally, with approximately 5 million deaths each year, accounting for 9% of global deaths (1). Uncontrollable bleeding after trauma is a major factor contributing to patient mortality. It poses significant challenges in treating patients with multiple injuries (2). Massive bleeding disrupts coagulation function, and early coagulation disorders caused by trauma are known as TIC. These patients require more rescue resources, such as blood transfusions, and stay longer in intensive care and hospitals (3, 4). TIC leads to a 3–4-fold increase in patient mortality (5). Currently, »damage control« strategies can significantly improve the survival of TIC patients, including damage control surgery, early whole blood or proportional blood product transfusion, and other measures (6, 7). Early and active intervention strategies targeting TIC can significantly improve patient prognoses (8). Therefore, the early identification of TIC is crucial for quickly and effectively initiating damage control interventions. However, rapid identification of TIC patients remains a challenge in clinical practice.

Currently, TIC is believed to be caused by various factors, including haemorrhagic shock, production of the thrombin-thrombomodulin complex related to tissue damage, and activation of anticoagulant and fibrinolytic pathways. Protein C activation may be the core pathophysiological mechanism underlying TIC and is closely related to increased fibrinolysis and Fbg loss (9–11). The severity of coagulation disorders is influenced by factors such as acidosis, hypothermia, blood dilution, inflammation, inadequate perfusion, and a coagulation factor-consuming environment (12, 13). When coagulation disorders occur after trauma, these influencing factors can interact with or promote each other, further accelerating disease deterioration.

To facilitate clinical treatment decisions for patients with TIC, we designed this study to identify the clinical and laboratory factors related to traumatic coagulation disease in patients with severe trauma. We aimed to analyse the factors influencing traumatic coagulation disease in these patients and construct a new model based on these factors to predict its occurrence in patients with multiple injuries. This will enable emergency physicians to identify high-risk patients with traumatic coagulation disease early and effectively initiate damage control interventions in patients with severe trauma.

Materials and Methods

Collected data

This study was based on the clinical data of 214 patients with multiple injuries admitted to the emergency department of the First Affiliated Hospital of Zhejiang University School of Medicine between December 2020 and December 2022. Patients were admitted within 4 h of injury.

Severe multiple injuries: Injuries to two or more anatomical parts caused by the same injury factor, with ISS ≥16 points. Based on the ISS scoring system, the human body is divided into 6 anatomical parts (14).

Inclusion criteria: 1. All patients diagnosed with severe multiple injuries were screened for TIC based on the diagnostic criteria at a later stage; 2. Ages ranged from 18 to 80 years.

Exclusion criteria: 1. Patients with trauma occurring more than 4 h before admission; 2. Patients who had been using antiplatelet or anticoagulant drugs long-term; 3. Patients with coagulation disorders or haemophilia due to chronic liver disease; 4. Pregnant, lactating, or potentially pregnant women; 5. Patients with incomplete information.

TIC diagnostic criteria: 1. INR > 1.2; 2. Presence of active or potential bleeding or need for blood product replacement treatments (15).

Based on the presence or absence of TIC, patients were divided into TIC and non-TIC groups.

Data were divided into general information and testing indicators. General information included sex, age, and ISS at admission. Laboratory testing indicators were assessed upon emergency admission (within 1 hour after arrival) and included plasma D-dimer level, complete blood count (including white blood cell count [WBC], haemoglobin [Hb], and platelet count [PLT]), arterial blood gas analysis (including pH, PaO_2, $PaCO_2$, BE and lactic acid [LA]), and coagulation function profile (including PT, activated partial thromboplastin time [APTT], and Fbg). All blood samples were collected in a non-fasting state, as emergency conditions precluded fasting requirements.

Ethics

This retrospective study was approved by the Ethics Committee of the First Affiliated Hospital of Zhejiang University School of Medicine; the ethical batch number is 2024-0987. We took measures to protect patients' privacy and personal information. Because retrospective studies are harmless, the local ethics committee waived the need for informed consent.

Statistics

SPSS 23.0 statistical software was used for analysis. Normally distributed measurement data were expressed as mean ± standard deviation ($\bar{x} \pm S$). A t-test or corrected t-test was used for comparison between groups. Count data were expressed as the number of cases (percentage) [n (%)] and compared between groups using the chi-square test. A nonparametric rank sum test was used to compare non-normally distributed measurement data between groups. Univariate analysis was used to analyse the influencing factors of TIC. The significant ($p<0.05$) variables were selected for further analysis. Multivariable logistic regression analysis was performed using the backward stepwise (likelihood ratio) method to identify independent risk factors for TIC, with all variables showing statistical significance in univariate analysis ($p<0.05$) entered simultaneously into the initial model, and non-significant variables removed stepwise based on the likelihood ratio test. Odds ratios (OR) with 95% confidence intervals (CI) were calculated to estimate the association between each independent predictor and TIC, with OR>1 indicating a positive association and OR<1 indicating a negative association. A prediction model was then constructed based on the regression coefficients (B) of the independent predictors retained in the final model. The model score was calculated as the linear combination of each predictor weighted by its corresponding B value plus the constant. The receiver ROC curve was used to assess the discrimination of the prediction model, and the Youden index was calculated as the maximum of sensitivity and specificity. $p<0.05$ was considered statistically significant.

Results

Comparison of general clinical data

A total of 214 patients with severe multiple injuries were enrolled in this study, including 164 males (76.64%) and 50 females (23.36%), with a mean age of 52.81±14.98 years and a mean ISS at admission of 26.28±8.06 points. Among these, 63 patients (29.44%) were diagnosed with TIC, and 151 comprised the non-TIC group. The levels of total WBC count ($P<0.05$), Hb ($P<0.001$), PLT ($P<0.05$), LnD-dimer levels ($P<0.001$), LA ($P<0.001$), BE ($P<0.001$), APTT ($P<0.05$), PT ($P<0.001$) and Fbg ($P<0.001$) differed significantly between the TIC and non-TIC groups. There were no differences between the two groups in terms of neutrophil and blood PH (*Table I*).

Predictive factors of TIC

The results of TIC-related univariate analysis are shown in *Table I*. Multivariable analysis using binary logistic regression showed that significant independent predictors of TIC were PT (odds ratio [OR]: 1.310, 95% confidence interval [CI]: 1.043–1.645), Fbg level (OR: 0.254, 95% CI: 0.122–0.525), LnD-dimer (OR: 1.433, 95% CI: 1.008–2.037), and BE (OR: 0.752, 95% CI: 0.646–0.875). The results are summarised in *Table II*.

Correlation analysis of TIC

To further evaluate the strength of association between each laboratory parameter and the occurrence of TIC, a correlation analysis was performed. Among all variables, PT (OR=1.310, P=0.020) and LnD-dimer (OR=1.433, P=0.045) showed positive correlations with TIC, while Fbg (OR: 0.254, P<0.001) and BE (OR: 0.752, P<0.001) demonstrated negative correlations. These findings suggest that PT, Fbg, BE, and LnD-dimer are the laboratory indicators most closely associated with TIC in patients with severe multiple injuries (*Table II*).

Table I Characteristics and laboratory examination in patients with severe trauma.

Characteristic	TIC	Non-TIC	P value
Male	48(63)	116 (151)	0.921
Age(mean ±SD)	50.81±17.11	53.64±13.97	0.248
ISS score	33.30±7.59	23.35±6.25	<0.001
WBC×10^9/L	16.54±6.42	13.95±5.06	0.002
Neutrophil×10^9 /L	10.86±6.38	19.09±8.13	0.548
Haemoglobin, g/L	119.33±25.68	134.09±16.78	<0.001
PLT×10^9 /L	196.03±167.35	220.80±65.85	0.014
lnD-dimer, ug/L	10.13±1.23	9.26±0.94	<0.001
LA, mmol/L	3.47±2.53	2.06±1.22	<0.001
PH	7.34±0.07	7.39±0.06	0.499
BE, mmol/L	-4.06±3.76	-0.74±2.36	<0.001
PT, S	14.05±3.07	12.15±1.46	<0.001
APTT, S	25.87±8.41	23.44±2.52	0.028
Fbg, g/L	1.59±0.64	2.41±0.71	<0.001

Significant differences were observed between the two groups for haemoglobin (Hb, P<0.001), prothrombin time (PT, P<0.001), fibrinogen (Fbg, P<0.001), base excess (BE, P<0.001), lnD-dimer (P<0.001), lactic acid (LA, P<0.001), white blood cell count (WBC, P<0.05), platelet count (PLT, P<0.05), and activated partial thromboplastin time (APTT, P<0.05). No intergroup differences were found for neutrophil count and blood pH.

ISS, injury severity score; Hb, haemoglobin; PT, prothrombin time; Fbg, fibrinogen; BE, base excess; LA, lactic acid; PH, pondus hydrogenii; WBC, white blood cell; PLT, platelet count; APTT, activated partial thromboplastin time.

Table II Multivariable analysis of factors associated with TIC.

Variable	P value	B	SE	Wald chi-square	OR	95% CI for OR
PT	0.020	0.270	0.116	5.407	1.310	1.043–1.645
Fbg	0.000	-1.372	0.372	13.631	0.254	0.122–0.525
lnD-dimer	0.045	0.360	0.179	4.021	1.433	1.008–2.037
BE	0.000	-0.285	0.077	13.568	0.752	0.646–0.875

Multivariable analysis using binary logistic regression showed that significant independent predictors of TIC were PT (OR: 1.310, 95% CI: 1.043–1.645), Fbg level (OR: 0.254, 95% CI: 0.122–0.525), LnD-dimer (OR: 1.433, 95% CI: 1.008–2.037), and BE (OR: 0.752, 95% CI: 0.646–0.875).

PT, prothrombin time; Fbg, fibrinogen; BE, base

Establishment of the prediction model

Through logistic multivariate analysis using the backward stepwise method, PT, Fbg, BE, and LnD-dimer levels were identified as significant independent risk factors for TIC. Based on the regression coefficients (B) obtained from the final logistic regression model, we established the clinical prediction model for TIC (CPMTIC) using the following formula: Model score = 0.27 × PT - 1.372 × Fbg + 0.36 × LnD-dimer - 0.285 × BE - 5.658. In this formula, the Score represents the linear predictor (logit) of TIC occurrence, which is calculated as the linear combination of each independent predictor weighted by its regression coefficient (B), plus the constant term. The AUC of the model was 0.877 (95% CI: 0.827-0.928), with a standard error of 0.026 (*Figure 1*). According to the ROC curve, the cut-off value of CPMTIC to predict TIC was -0.705. Based on these data, the sensitivity and specificity of the model were 76.2% and 88.3%, respectively.

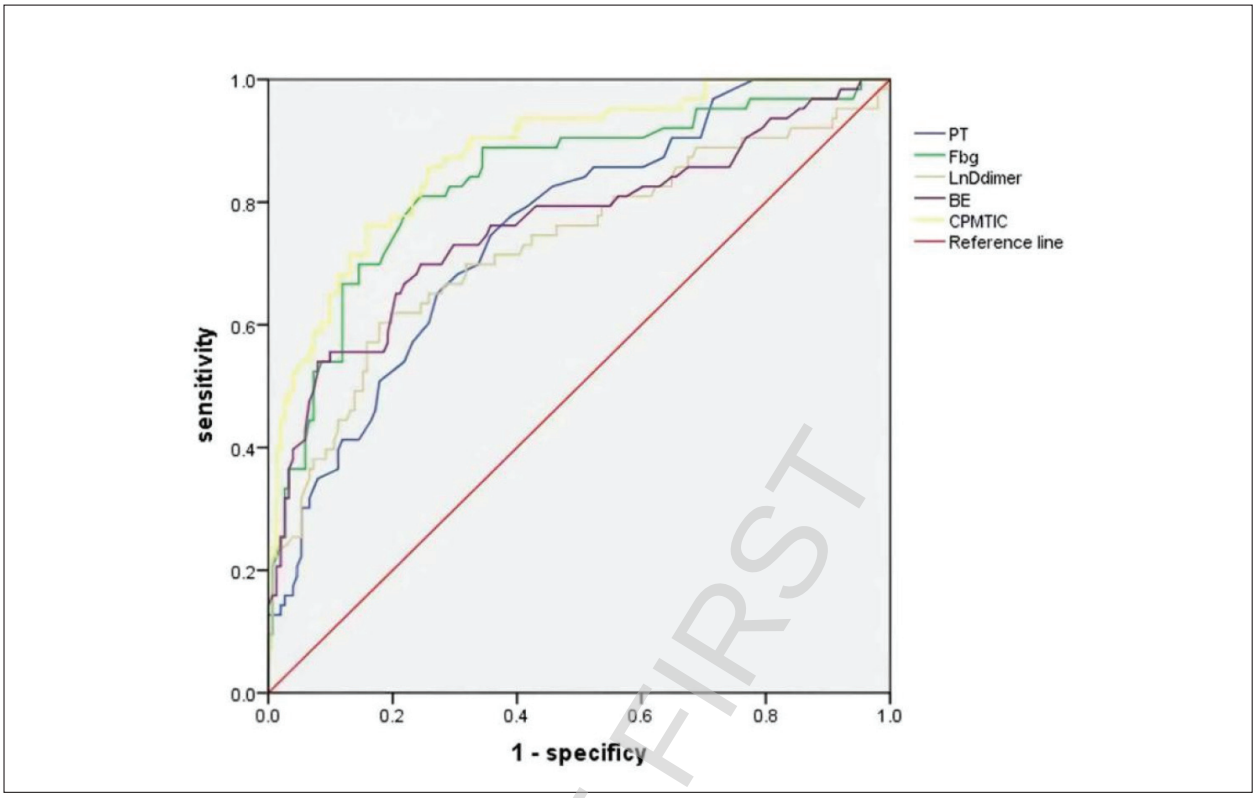

Figure 1 AUROC of the clinical prediction model for predicting trauma-induced coagulopathy(CPMTIC). The area under the ROC curve of the model was 0.877 (95% CI: 0.827–0.928), with a standard error of 0.026.

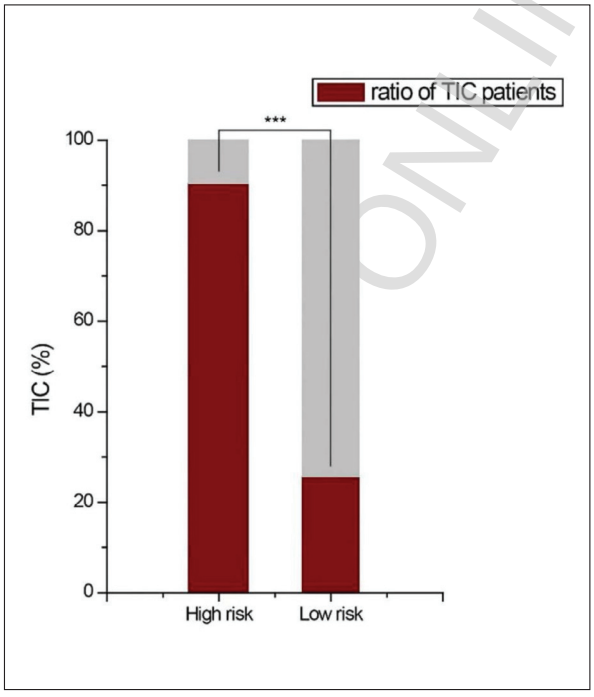

Figure 2 The proportion of trauma-induced coagulopathy (TIC) in the different groups. The proportion of TIC in the high-risk group (n=21)was much higher than that in the low-risk group (n=78) (90.4% vs 25.6%, p<0.001).

For model validation, data were collected again according to research standards. A total of 99 patients with multiple injuries admitted to the First Affiliated Hospital of Zhejiang University School of Medicine between March 2023 and October 2023 were included. Patient data were collected and divided into two groups based on their CPMTIC scores: a high-risk group (CPMTIC score ≥-0.705, n=21) and a low-risk group (CPMTIC score <-0.705, n=78). The analysis showed that the proportion of TIC patients in the high-risk group was significantly higher than that in the low-risk group (90.4% vs 25.6%; p<0.001) (*Figure 2*).

Discussion

In this retrospective study, we developed and validated a prediction model for early identification of TIC. Our main findings are as follows:

TIC occurred in approximately one-quarter of patients with severe multiple injuries.

Coagulation parameters (PT, APTT, Fbg, D-dimer), metabolic markers (BE, LA), and routine blood indicators (Hb, WBC, PLT) were associated with TIC on univariate analysis.

PT, Fbg, LnD-dimer, and BE were identified as independent predictors.

The model incorporating these four variables showed good predictive performance and was validated in an independent cohort.

These findings suggest that our model, based on four routinely available laboratory tests, may assist emergency clinicians in identifying high-risk TIC patients and facilitating timely intervention.

Despite continuous advancements in modern trauma treatment techniques, most early preventable deaths from trauma are due to uncontrolled bleeding (16–18). TIC is a complex condition involving coagulation, inflammation, and a multi-phenotypic disease process characterised by clot formation, decomposition, and overall disruption of vascular homeostasis. This results in haemostasis failure due to a combination of tissue damage and haemorrhagic shock. Approximately one-quarter to one-third of patients with multiple injuries develop TIC. This early coagulation change is secondary to the systemic activation of protein C and fibrinolysis pathways. It worsens with uncontrolled bleeding, fluid resuscitation, hypothermia, acidosis, severe injury, and systemic hypoperfusion.

Currently, the diagnosis of traumatic coagulation disease primarily relies on laboratory tests, such as routine blood tests and coagulation function tests. However, these tests have drawbacks, such as long waiting times. In recent years, the application of viscoelastic haemostasis analysis (VHA) has improved the identification of patients with early TIC. Nevertheless, the diagnosis of early TIC remains controversial (19, 20). Therefore, identifying patients with TIC as early as possible and implementing effective measures promptly are crucial to improving patient survival rates. For patients with TIC, early symptomatic treatments, such as blood transfusion, fluid replacement, and tranexamic acid, should be administered to improve prognosis and reduce mortality. Research has shown that early excessive treatment of traumatic coagulation disease can exacerbate the deterioration of coagulation function, leading to poor prognosis (4, 21). Accurate screening can prevent unnecessary early overtreatment (22, 23). Owing to current diagnostic limitations, the early identification of patients with TIC remains a significant challenge (24). Based on these factors, our study aimed to identify patients with TIC who require early intervention in response to existing clinical issues.

After severe trauma, significant blood loss and fluid replacement lead to the loss and consumption of coagulation factors (15, 25). Simultaneously, exposure to tissue factors (TF) after trauma drives the production of local thrombin and Fbg. Activated TF continuously recruits and activates factor VII (FVII) in the circulation to form a TF-FVII complex, completing the exogenous coagulation activation process. The TF-FVIIa complex can also activate platelets and induce the protein-activated receptor-mediated signalling pathway, thereby releasing more pro-inflammatory cytokines, further exacerbating the inflammatory response process, and creating a vicious cycle. During this process, coagulation factors involved in the exogenous coagulation pathway (such as FVII) are continuously consumed. Inflammatory responses and tissue perfusion disorders caused by microvascular thrombosis can also affect and damage the liver, the main site for synthesising most coagulation factors, including FVII (26–28). This decreases coagulation factor synthesis, reflected by prolonged PT. Prolonged PT is closely related to traumatic haemorrhage, coagulation dysfunction, and organ dysfunction, which is consistent with this study's conclusions.

Fbg is the most abundant coagulation factor in the blood (29), serving as a substrate for fibrin formation, clot formation, and platelet aggregation, playing a crucial role in haemostasis. Despite its high concentration, Fbg can rapidly decrease in the early stages of bleeding due to blood loss, dilution from fluid resuscitation, coagulation depletion, hypothermia, acidosis, and excessive breakdown (30–33). The incidence of Fbg depletion depends on injury severity. Recent studies have shown that initial Fbg levels below the normal range are independent risk factors for in-hospital mortality in patients with severe trauma (34). Low Fbg concentration is associated with increased blood transfusion volume, ventilator use days, and early and late mortality rates. Fbg supplementation can improve clot initiation and stability, potentially reducing mortality. Fbg levels can be increased with supplementation with fresh frozen plasma, cold precipitates, or plasma-derived virus-inactivated Fbg concentrates. However, the threshold level, optimal source, and timing of supplementation after trauma remain controversial (35, 36).

D-dimer is a specific degradation product of fibrin cross-linked by factor XIII and hydrolysed by plasmin, serving as a specific fibrinolytic marker reflecting the body's coagulation status (37). Severe trauma-induced shock and tissue hypoperfusion can damage the patient's vascular intima, cause subintimal tissue exposure, and activate the coagulation system. Damaged endothelial cells excessively release tissue plasminogen activator (tPA), and activation of the fibrinolytic system is mainly due to increased tPA levels. High tPA levels lead to hyperfibrinolysis. The absence of fibrinolytic inhibitors (including 2 anti-fibrinolytic enzymes) and platelet dysfunction exacerbate fibrinolysis (38, 39). Abnormal platelet function reduces plasminogen activator inhibitor-1 (PAI-1) levels. Studies show that tPA activation is accompanied by a significant

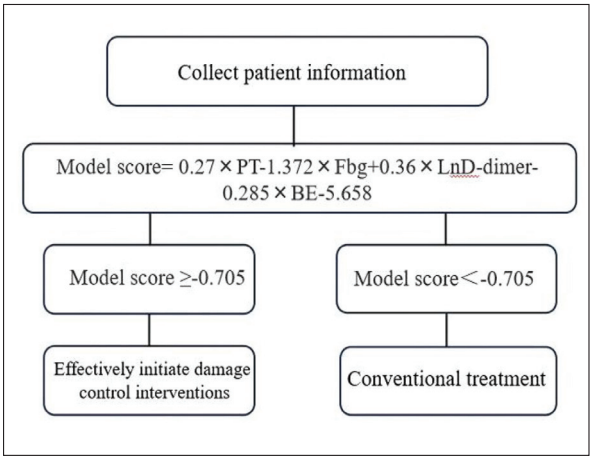

Figure 3 Example of management proposal using the clinical prediction model for predicting the trauma-induced coagulopathy (CPMTIC) assessment in treatment decision in TIC. The CPMTIC guides clinical decision-making. If the model score ≥-0.705, effectively initiating damage control interventions is recommended. If the model score is <-0.705, conventional treatment can be considered.

decrease in PAI-1 levels, which is also a significant characteristic of fibrinolysis in patients with trauma (40–42). Patients with severe trauma experience disturbances in their fibrinolysis and coagulation systems due to highly dynamic circulation and metabolism. The large number of inflammatory mediators released can activate the coagulation system, forming microthrombi and resulting in sustained abnormal elevation of D-dimer levels (43).

Patients with severe trauma often experience shock and tissue hypoxia, usually related to low blood volume and insufficient tissue perfusion from bleeding, leading to severe metabolic acidosis. The most obvious indicators are an increase in the negative value of BE and serum lactate concentration (44). Clinical studies have found BE to be an independent risk factor for haemorrhagic shock in patients with severe trauma (45, 46). BE reflects the acid-base balance of the internal environment, which is generally unaffected by respiratory factors. It mainly reflects metabolic factors and accurately indicates metabolic acid-base balance disorders in the body. The European guideline on management of major bleeding and coagulopathy following trauma (sixth edition) recommends using BE as a sensitive indicator for monitoring the degree of bleeding and shock (15). In vitro experiments have shown that acidosis can delay fibrin aggregation, weaken blood clot strength, and reduce V and IX factor activity and platelet aggregation (47–49). Fbg consumption increases, and the activity of each coagulation protease decreases by more than half when the pH drops from 7.4 to 7.2 (50, 51). These effects ultimately manifest as prolonged coagulation and increased bleeding times, providing strong evidence that acidosis is a major factor in coagulation diseases.

The TIC prediction model based on PT, D-dimer, BE, and Fbg levels showed good predictive performance. In retrospective studies, the area under the curve was 0.877. The diagnosis of TIC mainly relies on detecting coagulation function, and diagnostic accuracy is affected by many factors. Our model includes coagulation and metabolic indicators, and the detection indicators involved in the prediction model have been widely studied; the data are easily obtainable in clinical practice. With high accuracy and operability, the CPMTIC can help doctors quickly diagnose TIC and guide early effective treatment measures. For patients in the high-risk TIC group (CPMTIC score ≥-0.705), damage control interventions should be initiated more effectively. Meanwhile, those in the low-risk group should be given conventional treatment measures to avoid excessive treatment accelerating disease deterioration (*Figure 3*).

However, the sample size limitations may have introduced bias. Future studies should expand the sample size and involve multicentre research to validate the TIC prediction model. We noticed that many studies focused on applying VHA to the diagnosis of early TIC, and the results showed that VHA had good application value for the rapid diagnosis of TIC. Because the patients in this study were sent to the emergency department in critical condition, some patients did not undergo VHA testing, and the data could not be completely collected. In the follow-up study, we will include VHA testing further to improve the early and rapid diagnosis of TIC. Additionally, investigating the optimal timing and source of Fbg supplementation after trauma could provide further insights into improving patient outcomes and refining early intervention strategies.

Conclusion

Our study developed a simple and effective prediction model to identify high-risk patients with traumatic coagulation disease early. The TIC prediction model based on PT, D-dimer, BE, and Fbg levels could guide early effective interventions for TIC and improve the prognosis in severe trauma patients.

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Conflict of interest statement

All the authors declare that they have no conflict of interest in this work.

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