# Risk Factors and Preventive Measures for Delayed Intracranial Hematoma Following Surgery for Severe Traumatic Brain Injury

*Ann. Ital. Chir.*, 2025 96, 7: 967–972
https://doi.org/10.62713/aic.4032

Cheng Kong[1], Zhijian Xu[2]

[1]Neurosurgery Department, Panan County People's Hospital, 322399 Jinhua, Zhejiang, China
[2]Neurosurgery Department, Affiliated Jinhua Hospital, Zhejiang University School of Medicine, 321000 Jinhua, Zhejiang, China

AIM: To investigate the risk factors and potential preventive measures of delayed intracranial hematoma (DIH) following severe traumatic brain injury (sTBI).

METHODS: Clinical data from 132 patients with sTBI who underwent decompressive craniectomy between January 2022 and December 2024 were retrospectively analyzed. The control group (102 cases) did not develop delayed intracranial hematoma postoperatively, while the study group (30 cases) experienced DIH. General clinical characteristics were compared between the two groups, and multivariate logistic regression was used to identify risk factors associated with DIH following sTBI.

RESULTS: No significant differences were observed in age, sex, pupil changes, prothrombin time, hematoma volume, subdural hematoma, or cerebral contusion between the two groups ($p > 0.05$). The study group exhibited longer thrombin time (TT) and activated partial thromboplastin time (APTT), higher systolic and diastolic blood pressures, and lower fibrinogen levels (all $p < 0.05$). A greater proportion of patients in the study group had a Rotterdam computed tomography (CT) score >3, Glasgow Coma Scale (GCS) ≤8, skull fractures, and epidural hematoma (all $p < 0.05$). Logistic regression analysis identified preoperative Rotterdam CT score >3, GCS ≤8, prolonged TT, elevated diastolic blood pressure (DBP), and systolic blood pressure (SBP) as independent risk factors for postoperative DIH ($p < 0.05$).

CONCLUSIONS: Independent risk factors for DIH following decompressive craniectomy in patients with sTBI include a preoperative Rotterdam CT score >3, skull fractures, prolonged TT, and elevated DBP and SBP. Early, targeted preventive strategies and timely interventions for high-risk patients may help reduce the incidence of DIH following sTBI. Larger, multicenter studies are warranted to validate these findings and identify additional contributing factors.

**Keywords:** severe craniocerebral injury; delayed intracranial hematoma; risk factors; preventive measures

## Introduction

Traumatic brain injury (TBI) is a common and frequently encountered condition in neurosurgical departments, typically resulting from high-energy external forces. Severe traumatic brain injury (sTBI), often defined by a Glasgow Coma Scale (GCS) score of 8 or less following resuscitation [1,2], accounts for approximately 15% to 20% of all TBI cases. Patients with sTBI frequently present in critical condition, with high rates of disability and mortality [3]. Acute intracranial hypertension and cerebral swelling are common complications in these patients [4], among which delayed intracranial hematoma (DIH) is one of the most frequent and severe, significantly increasing mortality risk and often resulting in poor outcomes despite treatment [5]. The reported incidence of DIH in sTBI patients ranges from 1% to 8% [6,7], although broader definitions may suggest higher rates [5,8]. DIH generally refers to the emergence of new hemorrhagic lesions, typically occurring within 48 hours to several weeks post-injury, in brain regions that appeared normal or near-normal on initial computed tomography (CT) scans [9].

Current clinical studies have identified several factors associated with the development of DIH in patients with sTBI. These include the presence of skull fractures, the time interval between injury and surgery, and plasma thrombin time (TT), all of which may contribute to increased incidence of DIH [10,11]. Failure to implement timely and effective interventions may lead to disease progression and poor treatment outcomes [12,13]. This study aimed to investigate the risk factors and preventive strategies for DIH following decompressive craniectomy in patients with sTBI. The findings of this study may help clinicians identify high-risk patients and develop timely interventions to reduce the incidence and improve the prognosis of delayed intracranial hematoma following decompressive craniectomy.

*Submitted: 7 March 2025 Revised: 10 June 2025 Accepted: 13 June 2025 Published: 10 July 2025*
*Correspondence to: Zhijian Xu, Neurosurgery department, Affiliated Jinhua Hospital, Zhejiang University School of Medicine, 321000 Jinhua, Zhejiang, China (e-mail: zjx15958964950@163.com).*

## Materials and Methods

*General Data*

A retrospective analysis was conducted on the clinical data of 132 patients with sTBI who underwent decompressive craniectomy at Panan County People's Hospital between January 2022 and December 2024. The control group consisted of 102 patients who did not develop delayed intracranial hematoma after surgery, while the study group comprised 30 patients who developed delayed intracranial hematoma. This study was approved by the Medical Ethics Committee of Panan County People's Hospital [Approval No.2025(05)]. Informed consent was obtained from all participants in accordance with the ethical guidelines. The study was conducted in accordance with the principles outlined in the Declaration of Helsinki.

Inclusion criteria: (1) patients with diagnoses of sTBI and delayed intracranial hematoma that met the corresponding diagnostic criteria; (2) patients with complete clinical data. Exclusion criteria: (1) patients with severe systemic diseases or failure of vital organs such as the heart, lungs, liver, or kidneys; (2) patients with malignant tumors.

*Definition and Diagnosis of DIH*

DIH was defined as the development of a new intracranial hematoma on a follow-up CT scan that was absent on the initial postoperative CT scan or as a significant increase in the size of a pre-existing, minor hematoma, occurring within 7 days after decompressive craniectomy [14,15]. The diagnosis was confirmed by at least two neurosurgeons based on CT imaging. Routine follow-up CT scans were performed within 24–48 hours post-surgery, with additional imaging conducted upon any neurological deterioration.

*Observation Indicators and Detection Methods*

The incidence and potential risk factors of DIH following decompressive craniectomy in patients with sTBI were evaluated. The development of DIH was statistically analyzed based on the following indicators:

(1) Demographic and clinical variables: Age, gender, pupil changes, time from injury to surgery, presence of intracerebral hematoma, skull fractures, subdural hematoma, epidural hematoma, hematoma volume, cerebral contusion, diastolic blood pressure (DBP), and systolic blood pressure (SBP) were compared between the two groups.

(2) Preoperative Rotterdam CT score [16]: The score was assessed based on four criteria: subarachnoid hemorrhage, intraventricular or basal cistern hemorrhage, degree of mass effect, and midline shift. The total score ranged from 0 to 6, with higher scores indicating greater injury severity.

(3) GCS [17]: The GCS score ranged from 3 to 15 and was determined by assessing verbal, eye, and motor responses in individuals with suspected brain injury.

(4) Preoperative coagulation parameters: Fibrinogen level, thrombin time (TT), prothrombin time (PT), and activated partial thromboplastin time (APTT) were measured. In the morning, 5 mL of fasting venous blood was drawn by the nursing staff and analyzed using an automated coagulation analyzer (CS-5100, Sysmex, Kobe, Japan, registration number: NME 2402061).

*Statistical Methods*

Statistical analyses were performed using IBM SPSS Statistics for Windows, version 26.0 (IBM Corp., Armonk, NY, USA). The Shapiro-Wilk test was used to assess the normality of continuous variables. Normally distributed continuous variables were compared using the independent samples *t*-test and expressed as mean ± standard deviation. Non-normally distributed continuous variables were analyzed using the Mann-Whitney U test and expressed as median (interquartile range). Categorical variables were compared using Pearson's chi-square ($\chi^2$) test and expressed as counts and percentages [n (%)]. Multivariate analysis to identify independent risk factors for DIH was conducted using binary logistic regression, including variables with $p < 0.05$ in univariate analysis or those considered clinically relevant. Odds ratios (OR) and 95% confidence intervals (CI) were calculated. A $p$-value $< 0.05$ was considered statistically significant. To evaluate multicollinearity, the variance inflation factor (VIF) was calculated via linear regression, and variables with VIF $\leq 5$ were retained to ensure model stability. Model fit was assessed using the Hosmer-Lemeshow test, with $p > 0.05$ indicating acceptable fit.

## Results

*Comparison of General Data Between the Two Groups*

There were no statistically significant differences between the two groups in gender, age, pupil changes, PT, hematoma volume, subdural hematoma or cerebral contusion (all $p > 0.05$). However, both TT and APTT were notably prolonged in the study group ($p < 0.05$). Additionally, the study group had a higher proportion of patients with Rotterdam CT scores >3, GCS scores ≤8, skull fractures, and epidural hematoma (all $p < 0.05$). SBP and DBP were significantly elevated in the study group compared to the control group ($p < 0.05$), while fibrinogen levels were significantly reduced ($p < 0.05$). Detailed results are presented in Table 1.

*Multivariate Analysis*

Collinearity analyses were performed using linear regression to calculate VIF for all predictor variables. All variables demonstrated VIF values ≤5 (range: 1.08–1.74), indicating no significant multicollinearity. Therefore, all selected variables (preoperative Rotterdam CT score >3, GCS ≤8, skull fractures, fibrinogen level, TT, APTT, intracerebral hematoma, epidural hematoma, DBP, and SBP) were included in the multivariate logistic regression model. The Hosmer-Lemeshow test indicated a good model fit ($p = 0.777$). Multivariate logistic regression analysis revealed that a preoperative Rotterdam CT score >3 points (OR 5.487, 95% CI 1.198–25.133, $p = 0.028$), GCS score ≤8

**Table 1. Comparison of baseline characteristics between the two groups.**

| Variable | Total (n = 132) | Control group (n = 102) | Study group (n = 30) | Statistic | *p*-value |
|---|---|---|---|---|---|
| Gender, n (%) | | | | $\chi^2 = 2.04$ | 0.153 |
| Male | 93 (70.45) | 75 (73.53) | 18 (60.00) | | |
| Female | 39 (29.55) | 27 (26.47) | 12 (40.00) | | |
| Age (years) | 47.10 ± 14.78 | 46.90 ± 15.45 | 47.77 ± 12.42 | t = –0.28 | 0.779 |
| Pupil changes, n (%) | | | | $\chi^2 = 0.16$ | 0.686 |
| No | 79 (59.85) | 62 (60.78) | 17 (56.67) | | |
| Yes | 53 (40.15) | 40 (39.22) | 13 (43.33) | | |
| Preoperative Rotterdam CT score, n (%) | | | | $\chi^2 = 7.92$ | 0.005 |
| >3 points | 67 (50.76) | 45 (44.12) | 22 (73.33) | | |
| ≤3 points | 65 (49.24) | 57 (55.88) | 8 (26.67) | | |
| GCS, n (%) | | | | $\chi^2 = 6.27$ | 0.012 |
| >8 points | 98 (74.24) | 81 (79.41) | 17 (56.67) | | |
| ≤8 points | 34 (25.76) | 21 (20.59) | 13 (43.33) | | |
| Skull fractures, n (%) | | | | $\chi^2 = 6.43$ | 0.011 |
| No | 75 (56.82) | 64 (62.75) | 11 (36.67) | | |
| Yes | 57 (43.18) | 38 (37.25) | 19 (63.33) | | |
| Fibrinogen (g/L) | 1.94 ± 0.62 | 2.04 ± 0.59 | 1.63 ± 0.61 | t = 3.30 | 0.001 |
| TT (s) | 19.70 ± 2.66 | 19.15 ± 2.48 | 21.54 ± 2.43 | t = –4.67 | <0.001 |
| PT (s) | 11.08 ± 1.44 | 11.02 ± 1.39 | 11.28 ± 1.59 | t = –0.89 | 0.376 |
| APTT (s) | 29.49 ± 3.46 | 28.89 ± 3.07 | 31.52 ± 3.96 | t = –3.85 | <0.001 |
| Intracerebral hematoma, n (%) | | | | $\chi^2 = 6.36$ | 0.012 |
| No | 79 (59.85) | 67 (65.69) | 12 (40.00) | | |
| Yes | 53 (40.15) | 35 (34.31) | 18 (60.00) | | |
| Subdural hematoma, n (%) | | | | $\chi^2 = 2.06$ | 0.151 |
| No | 68 (51.52) | 56 (54.90) | 12 (40.00) | | |
| Yes | 64 (48.48) | 46 (45.10) | 18 (60.00) | | |
| Epidural hematoma, n (%) | | | | $\chi^2 = 4.41$ | 0.036 |
| No | 79 (59.85) | 66 (64.71) | 13 (43.33) | | |
| Yes | 53 (40.15) | 36 (35.29) | 17 (56.67) | | |
| Hematoma volume (mL) | 50.36 ± 17.46 | 50.92 ± 16.83 | 48.43 ± 19.62 | t = 0.68 | 0.495 |
| Cerebral contusion, n (%) | | | | $\chi^2 = 0.11$ | 0.735 |
| No | 87 (65.91) | 68 (66.67) | 19 (63.33) | | |
| Yes | 45 (34.09) | 34 (33.33) | 11 (36.67) | | |
| DBP (mmHg) | 80.66 ± 12.52 | 78.09 ± 12.40 | 89.40 ± 8.36 | t = –5.77 | <0.001 |
| SBP (mmHg) | 131.13 ± 21.21 | 128.15 ± 20.37 | 141.27 ± 21.20 | t = –3.07 | 0.003 |

Note: t, *t*-test; $\chi^2$, Chi-square test; GCS, Glasgow Coma Scale; CT, computed tomography; TT, thrombin time; PT, prothrombin time; APTT, activated partial thromboplastin time; DBP, diastolic blood pressure; SBP, systolic blood pressure.

points (OR 7.297, 95% CI 1.385–38.438, $p = 0.019$), prolonged TT (OR 1.730, 95% CI 1.245–2.404, $p = 0.001$), higher SBP (OR 1.059, 95% CI 1.015–1.106, $p = 0.008$), and higher DBP (OR 1.100, 95% CI 1.026–1.180, $p = 0.007$) were independent risk factors for DIH. The results are summarized in Table 2.

## Discussion

With ongoing economic development, the incidence of sTBI in China has been rising annually. The clinical manifestations of sTBI primarily include severe limb motor deficits, impaired consciousness, aphasia, and headache, all of which significantly impact patient survival and quality of life [10,11].

Patients with sTBI often experience elevated intracranial pressure (ICP), which may compress ruptured blood vessels and the dura mater when ICP rises significantly. This compression affects cerebral perfusion and may contribute to ongoing hemorrhage and secondary brain injury, reflecting the complex pathophysiology of elevated ICP [12,13].

Decompressive craniectomy is commonly performed in sTBI patients to remove intracranial hematomas and lower ICP. However, once the "tamponade effect" is disrupted, pressure on the ruptured blood vessels is relieved, potentially leading to renewed bleeding and the onset of DIH, often resulting in poor prognosis [18,19]. The relatively high incidence of DIH following sTBI surgery underscores the importance of identifying associated risk factors and adopting individualized preventive measures [20,21].

In this study, a preoperative Rotterdam CT score >3 points, lower GCS score (≤8 points), prolonged TT, and elevated systolic and diastolic blood pressures (SBP and DBP) were

**Table 2. Multivariate analysis of risk factors for delayed intracranial hematoma following decompressive craniectomy.**

| Variable | $\beta$ | SE | Wald | $p$-value | OR (95% CI) |
|---|---|---|---|---|---|
| Preoperative Rotterdam CT score | | | | | |
| ≤3 points | | | | | Reference |
| >3 points | 1.702 | 0.776 | 4.807 | 0.028 | 5.487 (1.198–25.133) |
| GCS | | | | | |
| >8 points | | | | | Reference |
| ≤8 points | 1.987 | 0.848 | 5.496 | 0.019 | 7.297 (1.385–38.438) |
| Skull fractures | | | | | |
| No | | | | | Reference |
| Yes | 0.455 | 0.688 | 0.437 | 0.509 | 1.576 (0.409–6.073) |
| Fibrinogen (g/L) | –0.980 | 0.637 | 2.370 | 0.124 | 0.375 (0.108–1.307) |
| TT (s) | 0.548 | 0.168 | 10.673 | 0.001 | 1.730 (1.245–2.404) |
| APTT (s) | 0.183 | 0.097 | 3.531 | 0.060 | 1.201 (0.992–1.453) |
| Intracerebral hematoma | | | | | |
| No | | | | | Reference |
| Yes | 1.153 | 0.739 | 2.432 | 0.119 | 3.166 (0.744–13.479) |
| Epidural hematoma | | | | | |
| No | | | | | Reference |
| Yes | 0.598 | 0.744 | 0.646 | 0.422 | 1.818 (0.423–7.816) |
| DBP (mmHg) | 0.096 | 0.036 | 7.204 | 0.007 | 1.100 (1.026–1.180) |
| SBP (mmHg) | 0.058 | 0.022 | 6.968 | 0.008 | 1.059 (1.015–1.106) |

Note: OR, odds ratio; CI, confidence interval; SE, Standard Error.

identified as independent risk factors for DIH following decompressive craniectomy in sTBI patients. Coagulation function, evaluated through TT, PT, and APTT, indicates the activity of intrinsic and extrinsic coagulation pathways, as well as the intrinsic fibrinolytic system. Secondary fibrinolysis is commonly observed in sTBI patients, impairing coagulation and increasing the likelihood of postoperative bleeding, contributing to DIH formation [22,23]. Therefore, preoperative assessment of coagulation function is essential. In addition to transfusing packed red blood cells, plasma should be administered based on individual needs. Antifibrinolytic drugs may be used to manage secondary fibrinolysis and help reduce the risk of postoperative DIH [24].

A GCS score ≤8 indicates severe brain injury and serves as a significant clinical marker for DIH development [1]. The underlying mechanisms are multifactorial. First, severe primary brain injury, such as extensive cerebral contusions, compromises the integrity of brain parenchyma and local vasculature, increasing susceptibility to vascular rupture during subsequent pathophysiological changes [15,25]. Second, patients with severe brain tumor often exhibit impaired cerebrovascular autoregulation, making cerebral blood flow highly sensitive to fluctuations in systemic blood pressure, which increases the risk of vascular rupture [15,26]. Third, severe trauma can precipitate systemic coagulopathy, further elevating the risk of hemorrhage [27]. Additionally, surgical interventions frequently required for patients with low GCS scores, such as decompressive craniectomy, may contribute to the onset of DIH by imposing secondary stress on already compromised but initially non-hemorrhagic vessels. This occurs due to acute alterations in intracranial pressure dynamics and brain tissue shifts [28].

Postoperative hypertension, including elevations in SBP and DBP, has been identified as an independent risk factor for DIH. The core mechanisms are as follows: First, elevated blood pressure directly increases the wall stress on intracranial blood vessels. This is notably significant for structurally weakened vessels previously damaged by trauma, as excessive pressure may exceed their tolerance threshold, leading to rupture and subsequent hemorrhage [29]. Second, hypertension may impair cerebrovascular autoregulation, leading to localized cerebral hyperperfusion in areas with dysfunctional regulation. This altered hemodynamic state can increase vascular permeability and potentially contribute to vascular injury and rupture, exacerbating secondary brain damage in severe traumatic brain injury patients [26,30]. Third, persistent or sudden elevations in blood pressure can further compromise the integrity of the blood-brain barrier, which is often already compromised by the initial trauma [31]. The increased permeability allows the extravasation of blood components into the brain parenchyma, promoting hematoma formation or expansion. Concurrently, hypertension may enhance secondary brain injury mechanisms, including post-injury inflammatory responses and cerebral edema, which indirectly increase the risk of microvascular rupture and hemorrhage [26].

The Rotterdam CT score reflects the severity of brain injury and is based on four parameters: subarachnoid hemorrhage, intraventricular or basal cistern hemorrhage, mass effect, and midline shift. A preoperative Rotterdam CT score >3 indicates a severe brain injury. sTBI may result in adverse pathological changes in brain tissue, such

as swelling, edema, and bleeding. Since the cranial cavity has a fixed volume, an increase in intracranial content volume elevates ICP, complicating surgical decompression and increasing the risk of postoperative bleeding and delayed intracranial hematoma [32,33].

Cerebral contusion may directly damage vascular walls and surrounding connective tissue, leading to a delayed risk of intracranial hematoma formation. In sTBI patients with cerebral contusions, skull fractures, or a preoperative Rotterdam CT score >3, cranial CT and X-ray imaging should be promptly performed to identify critical tissue damage. Early detection enables clinicians to formulate targeted surgical procedures to reduce the incidence of delayed intracranial hematoma [34,35].

Our study has several limitations. First, it was a single-center retrospective analysis with a relatively small number of DIH cases, which may limit the statistical power and the generalizability of the findings. Although the number of predictors in the multivariate model exceeded the 10-events-per-variable rule for 30 events, collinearity analysis demonstrated low VIF values (all ≤1.74), supporting the inclusion of all clinically relevant variables. The Hosmer-Lemeshow test ($p$ = 0.777) further validated acceptable model fit. Larger, prospective, multicenter studies are needed to validate our findings and further investigate preventive strategies. Second, although we included all patients who underwent decompressive craniectomy for sTBI, some patients who died shortly after surgery may not have undergone follow-up imaging, potentially introducing survivorship bias. Third, we did not implement formal corrections for multiple comparisons, as this was an exploratory analysis. Thus, some associations may be prone to Type I errors. Fourth, the absence of matched controls or stratification between groups may result in residual confounding despite the use of multivariate adjustment. Finally, the study did not evaluate long-term functional outcomes or prognoses of patients with DIH, limiting our capacity to assess the broader clinical implications of the identified risk factors. These limitations highlight the need for future large-scale, prospective, multicenter studies to validate our findings and refine preventive strategies.

## Conclusions

In summary, our study identified several independent risk factors associated with the development of DIH following decompressive craniectomy in patients with sTBI. Notably, a preoperative Rotterdam CT score >3, a GCS score ≤8, prolonged TT, and elevated systolic and diastolic blood pressure were significant predictors of DIH occurrence. Patients presenting with these risk factors may benefit from early targeted preventive interventions and enhanced postoperative monitoring to mitigate the risk of delayed hemorrhage. Future multicenter studies involving larger patient cohorts are warranted to validate these findings, refine predictive models, and optimize clinical management strategies.

## Availability of Data and Materials

The data and materials in the current study are available from the corresponding author on reasonable request.

## Author Contributions

CK conducted the literature search. ZJX acquired the data. CK wrote the article and performed data analysis. ZJX contributed to important editorial changes in the manuscript. Both authors read and approved the final manuscript. Both authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

## Ethics Approval and Consent to Participate

This study was approved by the Medical Ethics Committee of Panan County People's Hospital [Approval No.2025(05)]. Informed consent was obtained from all participants in accordance with the ethical guidelines. The study was conducted in accordance with the principles outlined in the Declaration of Helsinki.

## Acknowledgment

Not applicable.

## Funding

This research received no external funding.

## Conflict of Interest

The authors declare no conflict of interest.

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