

ORIGINAL ARTICLE

Prognostic Performance of the Richmond Acute Subdural Hematoma (RASH) Score, Surgical Timing, and Decompressive Modality for Predicting In-Hospital Mortality Following Traumatic Acute Subdural Hematoma Evacuation at an Indonesian Tertiary Referral Center

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ABSTRACT

Background: Traumatic acute subdural hematoma (ASDH) carries high postoperative mortality, particularly where prehospital delays add to secondary intracranial hypertension. The Richmond Acute Subdural Hematoma (RASH) score combines age, Glasgow Coma Scale (GCS) score, pupillary reactivity, midline shift (MLS) >5 mm, and loss of consciousness into an 8-point scale.

Objective: To evaluate the discrimination, calibration, and multivariable independence of the preoperative RASH score, with surgical modality and injury-to-incision latency, for in-hospital mortality in an Indonesian referral cohort.

Methods: This retrospective cohort study included consecutive adults who underwent craniotomy or decompressive craniectomy (DC) for traumatic ASDH at Dr. Mohammad Hoesin General Hospital, Palembang, from January 2022 to December 2024 (67 of 95 screened admissions). We used receiver operating characteristic (ROC) curves, Youden's J index, likelihood ratios, and Firth penalized logistic regression.

Results: In-hospital mortality was 20.9% (14/67). Non-survivors had higher median RASH scores (5.5 vs. 3.0; $p < 0.001$) and longer injury-to-incision latency (9.5 vs. 5.8 hours; $p = 0.008$). Mortality rose from 0.0% (0/18) at scores 0–2 to 66.7% (2/3) at scores 7–8. The area under the curve was 0.824 (95% CI: 0.715–0.933). At RASH ≥ 5 , sensitivity was 78.6%, specificity 77.4%, and negative predictive value 93.2%. In Firth regression, the RASH score (aOR = 2.95 per point; 95% CI: 1.52–5.74), DC (aOR = 3.42; 1.14–10.26), and delay (aOR = 1.24 per hour; 1.04–1.48) were each associated with death.

Conclusion: The RASH score showed good discrimination in this single-center cohort. RASH ≥ 5 may support operative triage and family counseling, pending prospective validation.

1. Introduction

Traumatic acute subdural hematoma (ASDH) is among the deadliest neurosurgical emergencies worldwide. It complicates 11% to 21% of all severe traumatic brain injuries (TBIs), and postoperative mortality of 20% to 60% persists despite modern neurocritical care.^{1,2} The Randomized Evaluation of Surgery with Craniectomy for Patients with Acute Subdural Hematoma (RESCUE-ASDH) trial, run in well-resourced Western systems, found comparable 12-month disability outcomes after primary decompressive craniectomy (DC) and osteoplastic craniotomy.² Later real-world meta-analyses and large international registries, among them the Collaborative European NeuroTrauma Effectiveness Research in Traumatic Brain Injury (CENTER-TBI) and Transforming Research and Clinical Knowledge in Traumatic Brain Injury (TRACK-TBI) consortia, still report wide variation between centers in surgical selection, operative timing, and acute hospital mortality.^{3–9}

Traumatic ASDH can be rapidly fatal. Two linked processes are responsible: primary mechanical shear of the bridging cortical veins, and a fulminant secondary injury of the underlying parenchyma.^{10,11} As the extra-axial clot enlarges inside the rigid skull, intracranial compliance runs out along the exponential pressure-volume curve. Pressure then climbs.

Intracranial pressure (ICP) rises steeply, cerebral perfusion pressure (CPP) falls, and cerebrovascular autoregulation is impaired.^{11,12} Obstructed cortical venous outflow and compression of the ipsilateral hemisphere also provoke perihematomal spreading depolarizations, glutamate-mediated excitotoxicity, intracellular Ca^{2+} overload, and failure of the membrane Na^+/K^+ -ATPase pump. These events end in cytotoxic edema and a mismatch between midline shift (MLS) and hematoma thickness.^{10,13,14} Neuroinflammation adds to the damage within hours of injury. It runs through the ADAM17/soluble tumor necrosis factor- α (solTNF- α)/nuclear factor-kappa B (NF- κ B) axis and through extravasation of fibrinogen across the blood-brain barrier, and it speeds innate immune activation, vasogenic edema, and systemic immunoinflammatory dysregulation.^{13,15,16}

The decision to operate, the choice between craniotomy and DC, and the counseling of families about survival all have to be made within minutes of arrival in the emergency department. Clinicians need a fast, objective preoperative risk tool.^{17,18} Established general TBI models, such as the International Mission for Prognosis and Analysis of Clinical Trials in TBI (IMPACT) and Corticosteroid Randomisation After Significant Head Injury (CRASH) calculators, automated pupillometry

indices, and the combined GCS-Pupils scale, fall short. Some need laboratory values that are not available at triage, and others were not calibrated for patients selected for surgical ASDH evacuation.¹⁹⁻²² Age-specific tools, including the Subdural Hematoma in the Elderly (SHE) score and pediatric subdural nomograms, apply only to narrow demographic groups.²³⁻²⁵ A gap remains. To fill it, Dincer et al. developed and externally validated the Richmond Acute Subdural Hematoma (RASH) score in the 2016 United States National Trauma Data Bank and validated it in the 2017 database. The score combines five preoperative variables available in every patient (age, Glasgow Coma Scale [GCS] score, pupillary reactivity, MLS >5 mm on non-contrast computed tomography [NCCT], and loss of consciousness [LOC]) into an 8-point ordinal grading scale.¹

The RASH score performed well in the North American trauma centers where it was derived. Does it transfer? In lower-middle-income countries (LMICs) of Southeast Asia, this is still unknown.²⁶⁻²⁸ Patients in Indonesian regional referral networks, and in similar Asian and LMIC neurosurgical settings, often face long inter-facility transfers before arrival. Many are injured in high-velocity motorized two-wheeler crashes. Surgical decompression is frequently delayed.^{27,29-31} It has also not been rigorously quantified whether the composite RASH score keeps its independent prognostic value after simultaneous adjustment for evacuation modality (craniotomy vs. DC) and continuous injury-to-incision latency. This study therefore had three aims: (1) to evaluate, in an external cohort, the discrimination and calibration of the preoperative RASH score for in-hospital mortality at a major Indonesian tertiary neurosurgical referral center; (2) to estimate a decision threshold and the corresponding Bayesian post-test mortality probabilities; and (3) to determine the independent multivariable prognostic effects of the composite RASH score, its clinicoradiological components, surgical modality, and time from injury to surgery.

2. Methods

Study design and setting

This retrospective observational cohort study covered consecutive adults who underwent neurosurgical evacuation of traumatic ASDH over 36 months, from January 1, 2022, to December 31, 2024, at Dr. Mohammad Hoesin General Hospital in Palembang, South Sumatra, Indonesia, the main tertiary neurosurgical referral center for South Sumatra and neighboring provinces. The work followed the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines.³²

Participants and eligibility

We screened all patients admitted with acute traumatic subdural hematoma confirmed on admission cranial NCCT. Screening was consecutive and used the institutional neurosurgical operative registry and electronic medical records. Four criteria defined eligibility: (1) age ≥18 years at injury; (2) an acute traumatic mechanism, with presentation within 24 hours of the event; (3) definitive cranial evacuation by osteoplastic craniotomy or decompressive craniectomy; and (4) complete records of the preoperative neurological examination, baseline NCCT measurements, operative timing, and survival status at discharge. Exclusions were as follows. (1) Subacute or chronic subdural hematomas with no acute traumatic hemorrhagic component. (2) Penetrating gunshot or stab injuries of the brain. (3) Small, neurologically stable hematomas managed without surgery. (4) Moribund patients with fixed signs of brain death who received only palliative comfort care and no cranial surgery. (5) Incomplete core preoperative clinical or imaging records.

Variables and data collection

The preoperative RASH score was calculated for each patient just before skin incision, using the original 8-point criteria of Dincer et al.¹ Five clinicoradiological domains were scored. Age earned 0 points at 18–59 years, 1 point at 60–79 years, and 2 points at ≥80 years. The post-resuscitation preoperative GCS earned 0 points when mild [14–15], 1 point when moderate [9–13], and 2 points when severe [3–8]. Pupillary light reflex earned 0 points when both pupils were reactive, 1 point when one was non-reactive, and 2 points when both were non-reactive. Midline shift was measured on axial NCCT at the level of the foramen of Monro, as displacement of the septum pellucidum from the ideal sagittal midline, and earned 0 points at ≤5.0 mm and 1 point at >5.0 mm.^{1,10} Documented post-traumatic LOC, before arrival or in the emergency resuscitation bay, earned 0 points when absent and 1 point when present. The composite RASH score was the arithmetic sum of the five component weights:

$$\text{RASH Score} = W_{\text{Age}} + W_{\text{GCS}} + W_{\text{Pupils}} + W_{\text{MLS}>5\text{ mm}} + W_{\text{LOC}} \quad (\text{Range: 0 to 8 points}) \quad (\text{Eq. 1})$$

Surgical indications followed the international Brain Trauma Foundation (BTF) recommendations and institutional neurotrauma protocols. Urgent evacuation was advised for ASDH with clot thickness >10 mm, for MLS >5 mm whatever the GCS, and for neurological deterioration (a GCS fall of ≥2 points or new pupillary asymmetry).^{2,33,34} The attending neurosurgeon chose between osteoplastic craniotomy, with the bone flap replaced before skin closure, and primary DC, meaning a large frontotemporoparietal hemicraniectomy of ≥12 × 15 cm with expansive duraplasty and storage or omission of the bone flap. The choice rested on the severity of herniation before surgery and on cerebral swelling seen after hematoma evacuation.^{2,34} Some patients began with craniotomy but developed refractory postoperative intracranial hypertension, which required early secondary removal of the bone flap during the same admission. They were recorded under their index modality and also counted in cumulative DC exposure. Injury-to-incision latency was a continuous variable, in hours, measured from the documented time of the traumatic impact to skin incision.^{26,27} The primary outcome was all-cause in-hospital mortality. It was counted during the index neurosurgical admission.

Statistical analysis

Distributional normality of continuous variables was assessed with the Shapiro-Wilk test (W statistic). Normally distributed variables, such as patient age, are shown as mean ± standard deviation (SD) and were compared between survivors and non-survivors with the two-sample independent Student's t-test, with results given as mean difference (MD) and 95% confidence intervals (CIs). Injury-to-incision time and the composite RASH score were non-normal. They are reported as median with interquartile range (IQR, 25th–75th percentiles), and parametric mean ± SD is added for comparison with the literature. Groups were compared with the non-parametric Mann-Whitney U test, together with the standardized Z statistic and the rank-biserial correlation as effect size ($r_{\text{rb}} = 1 - 2U / [n_1n_2]$). Categorical variables are given as frequencies and percentages (n, %). They were compared with Pearson's chi-square (χ^2) test, or with the two-tailed Fisher's exact test when expected cell counts were <5. Some 2 × 2 tables had a zero-event cell (complete separation), as for MLS >5 mm and LOC. There, unadjusted odds ratios (ORs) and 95% CIs were estimated with the Haldane-Anscombe continuity correction (+0.5 added to each cell). Discrimination of the preoperative RASH score was measured by the area under the receiver operating characteristic curve (AUC-ROC), with a nonparametric DeLong

standard error (SE) and 95% CI. Calibration was assessed with the Hosmer-Lemeshow goodness-of-fit χ^2 test and the Brier quadratic probability score. Youden's J index ($J = \text{Sensitivity} + \text{Specificity} - 1$) was maximized in the study sample to select the binary cut-off; the cut-off and the associated accuracy figures are therefore apparent estimates. For each cut-off we computed sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), and overall accuracy, with Wilson score 95% CIs. We also computed the positive likelihood ratio ($LR^+ = \text{Sensitivity} / [1 - \text{Specificity}]$), the negative likelihood ratio ($LR^- = [1 - \text{Sensitivity}] / \text{Specificity}$), and the diagnostic odds ratio ($DOR = LR^+ / LR^-$). Bayesian post-test mortality probabilities started from the institutional pre-test prevalence ($P_0 = 20.9\%$) and applied Bayes' theorem; with the cohort prevalence as pre-test probability, they equal the positive predictive value and 1 - negative predictive value:

$$\text{Odds}_{\text{post}} = [P_0 / (1 - P_0)] \times LR, \quad P_{\text{post}} = \text{Odds}_{\text{post}} / (1 + \text{Odds}_{\text{post}}) \quad (\text{Eq. 2})$$

Small samples and quasi-complete separation can bias multivariable estimates. To limit this, we fitted Firth's penalized maximum likelihood logistic regression, which maximizes the penalized log-likelihood $\ell^*(\beta) = \ell(\beta) + 0.5 \ln|I(\beta)|$, where $I(\beta)$ is the Fisher information matrix. Model 1 contained the composite RASH score (per 1-point increase), cumulative DC modality, and continuous injury-to-incision latency (per hour). Model 2 split the RASH score into its main binary clinical and radiological drivers (age ≥ 60 years, severe GCS ≤ 8 , bilateral non-reactive pupils, and MLS > 5 mm) and kept DC modality and time to surgery; with six predictors for 14 deaths (about 2.3 events per variable), it is exploratory. We checked that all variance inflation factors (VIFs) were < 1.5 , which excluded multicollinearity. Two-tailed p-values < 0.05 were taken as statistically significant.

Ethical approval

The Health Research Ethics Committee of the Faculty of Medicine, Universitas Sriwijaya / Dr. Mohammad Hoesin General Hospital reviewed and approved the protocol (Approval number 2021/1041). The work followed the Declaration of Helsinki.

3. Results

Cohort selection and baseline characteristics

During the 36-month study window, 95 consecutive patients presented with radiologically confirmed traumatic ASDH. Following eligibility screening, 28 patients were excluded: 10

(10.5%) with minimal clot thickness and preserved neurological status who were managed conservatively, 18 (18.9%) who arrived in a moribund state with irreversible brainstem areflexia and received palliative comfort care without surgery, and 4 (4.2%) of screened admissions; 5.6% of the 71 operated patients) with incomplete preoperative neuroimaging or surgical timing records. In total, 67 adult patients (70.5% of screened admissions) who underwent cranial surgical evacuation formed the final analytical cohort, as detailed in Table 1 and illustrated in Figure 1.

Across the 67 analyzed patients, mean age was 52.4 ± 15.8 years (Shapiro-Wilk $W = 0.979$, $p = 0.312$), with 54 patients (80.6%) aged 18–59 years, 12 (17.9%) aged 60–79 years, and 1 (1.5%) aged ≥ 80 years (combined geriatric stratum ≥ 60 years: 13 patients, 19.4%). Males accounted for 71.6% (48/67) of the cohort, while females represented 28.4% (19/67). High-energy motor vehicle accidents constituted the predominant trauma mechanism in 41 patients (61.2%), followed by falls in 20 (29.9%) and other blunt cranial impacts in 6 (9.0%). On preoperative neurological assessment, 13 patients (19.4%) presented with mild GCS (14–15), 31 (46.3%) with moderate GCS (9–13), and 23 (34.3%) with severe coma (GCS ≤ 8). Pupillary light reflexes were bilaterally reactive in 55 patients (82.1%), unilaterally non-reactive in 3 (4.5%), and bilaterally non-reactive in 9 (13.4%), corresponding to an overall pupillary abnormality prevalence of 17.9% (12/67). Preoperative axial NCCT demonstrated significant MLS > 5 mm in 31 patients (46.3%) and ≤ 5 mm in 36 (53.7%), while a history of post-traumatic LOC was present in 61 patients (91.0%). Definitive osteoplastic craniotomy was performed in 41 patients (61.2%), whereas 26 patients (38.8%) required cumulative decompressive craniectomy, comprising 25 primary index DCs (37.3%) and 1 early secondary DC conversion (1.5%) for refractory postoperative cerebral edema in a surviving patient. Median latency from traumatic impact to surgical skin incision across the total cohort was 6.2 hours (IQR 4.5–9.8; parametric mean 7.4 ± 3.8 hours; $W = 0.914$, $p < 0.001$), and the median preoperative RASH score was 4.0 (IQR 2.0–5.0; parametric mean 3.63 ± 1.68 ; $W = 0.946$, $p = 0.006$), as detailed in Table 1 and illustrated in Figure 1.

Table 1. Baseline demographic, clinicoradiological, operative, and preoperative RASH score characteristics of the surgical cohort (N = 67).

Variable / Clinicoradiological Domain	RASH Point Weight	Total Surgical Cohort (N = 67)	Distributional Metric / Clinical Note
Screened Admissions & Cohort Selection	—	95 screened → 67 analyzed (70.5%)	Excluded: 10 (10.5%) conservative, 18 (18.9%) palliative, 4 (4.2%) incomplete
In-Hospital Outcome Status	—	53 Survivors (79.1%) / 14 Non-Survivors (20.9%)	Primary institutional mortality endpoint
Age, years (Mean ± SD)	Continuous	52.4 ± 15.8	Shapiro-Wilk $W = 0.979$, $p = 0.312$ (normal)
18–59 years, n (%)	0 points	54 (80.6%)	Reference non-geriatric RASH tier
60–79 years, n (%)	1 point	12 (17.9%)	Intermediate geriatric RASH tier
≥ 80 years, n (%)	2 points	1 (1.5%)	Advanced geriatric RASH tier (Combined ≥ 60 yr: 13 [19.4%])
Sex Distribution, n (%)	—		Male-to-female ratio = 2.53 : 1
Male	—	48 (71.6%)	—

Variable / Clinicoradiological Domain	RASH Point Weight	Total Surgical Cohort (N = 67)	Distributional Metric / Clinical Note
Female	—	19 (28.4%)	—
Mechanism of Traumatic Injury, n (%)	—		High-energy vehicular trauma predominance
Motor vehicle accident (MVA)	—	41 (61.2%)	—
Falls (ground-level or height)	—	20 (29.9%)	—
Other blunt cranial trauma	—	6 (9.0%)	Direct blunt impact / occupational injury
Preoperative Glasgow Coma Scale (GCS), n (%)	Ordinal (0–2)		Post-resuscitation baseline neurological status
Mild TBI (GCS 14–15)	0 points	13 (19.4%)	Preserved baseline arousal
Moderate TBI (GCS 9–13)	1 point	31 (46.3%)	Largest presenting GCS stratum
Severe TBI / Coma (GCS 3–8)	2 points	23 (34.3%)	High-risk coma stratum
Pupillary Light Reactivity, n (%)	Ordinal (0–2)		Any pupillary abnormality: 12 (17.9%)
Bilaterally reactive pupils	0 points	55 (82.1%)	Intact mesencephalic CN III reflex arc
Unilateral non-reactive pupil	1 point	3 (4.5%)	Ipsilateral uncal compression sign
Bilaterally non-reactive pupils	2 points	9 (13.4%)	Severe bilateral brainstem herniation sign
Preoperative Axial NCCT Midline Shift, n (%)	Binary (0–1)		Measured at level of foramen of Monro
Midline shift ≤5.0 mm	0 points	36 (53.7%)	Preserved spatial compensatory reserve
Midline shift >5.0 mm	1 point	31 (46.3%)	Exhausted intracranial compliance threshold
History of Loss of Consciousness (LOC), n (%)	Binary (0–1)		Prehospital or emergency department documentation
LOC absent	0 points	6 (9.0%)	Lucid or uninterrupted arousal
LOC present	1 point	61 (91.0%)	High baseline prevalence across surgical ASDH
Surgical Evacuation Modality, n (%)	Operative		Intraoperative neurosurgical decision
Definitive osteoplastic craniotomy	—	41 (61.2%)	Bone flap replaced at index closure
Cumulative decompressive craniectomy (DC)	—	26 (38.8%)	25 primary DC (37.3%) + 1 secondary DC conversion (1.5%)
Time from Injury to Surgery, hours	Continuous	Median 6.2 (IQR 4.5–9.8)	Parametric Mean ± SD: 7.4 ± 3.8 h (W = 0.914, p < 0.001)
Total Preoperative RASH Score (0–8 scale)	Composite (0–8)	Median 4.0 (IQR 2.0–5.0)	Parametric Mean ± SD: 3.63 ± 1.68 (W = 0.946, p = 0.006)

Abbreviations: ASDH, acute subdural hematoma; CN III, oculomotor nerve; DC, decompressive craniectomy; GCS, Glasgow Coma Scale; IQR, interquartile range; LOC, loss of consciousness; MVA, motor vehicle accident; NCCT, non-contrast computed tomography; RASH, Richmond Acute Subdural Hematoma; SD, standard deviation; TBI, traumatic brain injury.

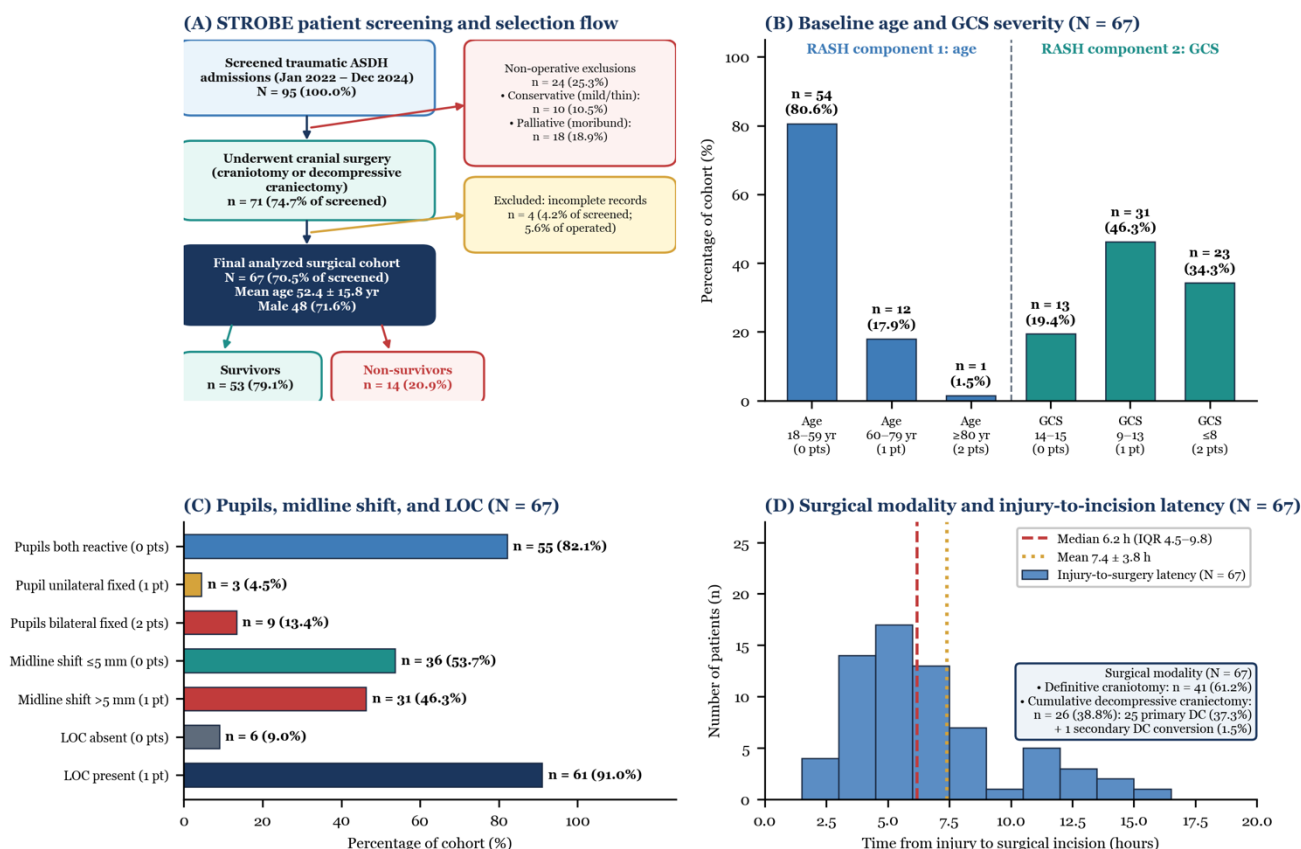


Figure 1. Cohort selection flow and baseline clinicoradiological architecture of the traumatic acute subdural hematoma (ASDH) surgical cohort (N = 67). (A) STROBE patient screening and eligibility flow diagram from 95 consecutive traumatic ASDH admissions to the final analyzed surgical cohort (N = 67; 53 survivors [79.1%] and 14 non-survivors [20.9%]). (B) Baseline age distribution (mean 52.4 ± 15.8 years) and preoperative Glasgow Coma Scale (GCS) severity tiers. (C) Preoperative prevalence of pupillary light reflex abnormalities, axial non-contrast computed tomography (NCCT) midline shift >5.0 mm, and history of loss of consciousness (LOC). (D) Distribution of index and cumulative surgical evacuation modalities (osteoplastic craniotomy vs. decompressive craniectomy [DC]) and overall injury-to-incision latency (median 6.2 hours [IQR 4.5–9.8]).

Bivariate associations with in-hospital mortality

Overall in-hospital mortality following surgical evacuation was 20.9% (14/67 non-survivors vs. 53/67 [79.1%] survivors). Bivariate comparisons of clinicoradiological, surgical, and composite RASH parameters stratified by survival status are summarized in Table 2 and depicted in Figure 2. Non-survivors were on average 7.70 years older than survivors (58.5 ± 16.2 vs. 50.8 ± 15.4 years; $t = 1.64$, $p = 0.106$) and demonstrated a higher proportion of geriatric patients aged ≥60 years (35.7% [5/14] vs. 15.1% [8/53]; OR = 3.13, 95% CI: 0.83–11.77, $p = 0.125$), whereas sex distribution (71.4% vs. 71.7% male; OR = 0.99, $p = 1.000$) and injury mechanism (64.3% vs. 60.4% motor vehicle accidents; $\chi^2 = 0.095$, $p = 0.954$) did not differ between groups.

Among individual neurological and radiological RASH components in unadjusted bivariate analysis, severe coma (GCS ≤8) was present in 50.0% (7/14) of non-survivors compared with 30.2% (16/53) of survivors (OR = 2.31, 95% CI: 0.69–7.71, $p = 0.165$), and bilateral non-reactive pupils were observed in 21.4% (3/14) of non-survivors versus 11.3% (6/53) of survivors (OR = 2.14, 95% CI: 0.46–9.93, $p = 0.381$). Preoperative MLS >5 mm showed complete separation: 100.0% (14/14) of non-survivors presented with MLS >5 mm compared with 32.1% (17/53) of survivors, so that no deaths (0/36, 0.0%; exact 95% CI: 0.0%–9.7%) occurred among patients with MLS ≤5 mm versus 14 deaths among the 31 patients (45.2%) with MLS >5 mm ($\chi^2 = 20.55$; Fisher's exact $p = 2.71 \times 10^{-6}$ [$p < 0.001$]). The continuity-corrected odds ratio in Table 2 (Haldane-Anscombe OR = 60.43, 95% CI: 3.40–1073.5) depends on the added 0.5 and is not an effect size. Similarly, 100.0%

(14/14) of non-survivors had documented LOC compared with 88.7% (47/53) of survivors (Haldane-Anscombe OR = 3.99, 95% CI: 0.21–75.29, $p = 0.330$). Regarding operative factors, non-survivors required decompressive craniectomy at more than double the rate of survivors, both when evaluated by primary index DC (71.4% [10/14] vs. 28.3% [15/53]; $\chi^2 = 8.81$, OR = 6.33, 95% CI: 1.71–23.48, $p = 0.003$) and cumulative DC exposure (71.4% [10/14] vs. 30.2% [16/53]; $\chi^2 = 7.93$, OR = 5.78, 95% CI: 1.57–21.28, $p = 0.005$). Non-survivors also experienced a 3.7-hour longer median injury-to-incision delay than survivors (9.5 hours [IQR 6.5–12.5; mean 10.3 ± 4.2 hours] vs. 5.8 hours [IQR 4.1–8.0; mean 6.6 ± 3.3 hours]; Mann-Whitney $U = 198.5$, $Z = -2.654$, $r_{rb} = 0.465$, $p = 0.008$), as summarized in Table 2 and depicted in Figure 2.

When individual risk factors were aggregated into the composite RASH score, non-survivors had a significantly higher preoperative RASH distribution than survivors (median 5.5 [IQR 4.0–6.0; mean 5.29 ± 1.44] vs. median 3.0 [IQR 2.0–4.0; mean 3.19 ± 1.45]; Mann-Whitney $U = 126.0$, $Z = -3.791$, $r_{rb} = 0.660$, $p < 0.001$). Across ordinal RASH strata, observed in-hospital mortality rose monotonically from 0.0% (0/7) in scores 0–1 and 0.0% (0/11) in score 2 (0/18 [0.0%] across scores 0–2), to 7.7% (1/13) in score 3, 15.4% (2/13) in score 4, 33.3% (4/12) in score 5, 62.5% (5/8) in score 6, and 66.7% (2/3) in scores 7–8 (Cochran-Armitage trend $p < 0.001$), as summarized in Table 2 and depicted in Figure 2. For comparison, the original registry cohort reported mortality ranging from 0% to 94% across scores 0 to 8.¹

Table 2. Bivariate comparison of clinicoradiological variables, operative modality, surgical latency, and RASH score distribution between survivors and non-survivors (N = 67).

Parameter / RASH Stratum	Total (N = 67)	Survivors (n = 53, 79.1%)	Non-Survivors (n = 14, 20.9%)	Effect Size / OR (95% CI)	p-value
Age, years (Mean ± SD)	52.4 ± 15.8	50.8 ± 15.4	58.5 ± 16.2	MD = +7.70 (−1.68 to +17.08)	0.106 ^a
Geriatric Age ≥60 years, n (%)	13 (19.4%)	8 (15.1%)	5 (35.7%)	OR = 3.13 (0.83–11.77)	0.125 ^b
Male Sex, n (%)	48 (71.6%)	38 (71.7%)	10 (71.4%)	OR = 0.99 (0.27–3.63)	1.000 ^b
Mechanism of injury: motor vehicle accident, n (%)	41 (61.2%)	32 (60.4%)	9 (64.3%)	$\chi^2 = 0.095$ (df = 2)	0.954 ^c
Severe Coma (GCS ≤8), n (%)	23 (34.3%)	16 (30.2%)	7 (50.0%)	OR = 2.31 (0.69–7.71)	0.165 ^c
Bilateral Non-Reactive Pupils, n (%)	9 (13.4%)	6 (11.3%)	3 (21.4%)	OR = 2.14 (0.46–9.93)	0.381 ^b
Midline Shift >5.0 mm, n (%)	31 (46.3%)	17 (32.1%)	14 (100.0%)	OR = 60.43 (3.40–1073.5) ^d	<0.001 ^{*b}
Loss of Consciousness Present, n (%)	61 (91.0%)	47 (88.7%)	14 (100.0%)	OR = 3.99 (0.21–75.29) ^d	0.330 ^b
Primary Index DC (vs. Craniotomy), n (%)	25 (37.3%)	15 (28.3%)	10 (71.4%)	OR = 6.33 (1.71–23.48)	0.003 ^{*c}
Cumulative DC Exposure, n (%)	26 (38.8%)	16 (30.2%)	10 (71.4%)	OR = 5.78 (1.57–21.28)	0.005 ^{*c}
Time to Surgery, hours (Median [IQR])	6.2 (4.5–9.8)	5.8 (4.1–8.0)	9.5 (6.5–12.5)	U = 198.5, Z = −2.654, r_{rb} = 0.465	0.008 ^{*e}
Time to Surgery, hours (Mean ± SD)	7.4 ± 3.8	6.6 ± 3.3	10.3 ± 4.2	MD = +3.70 (+1.61 to +5.79)	<0.001 ^{*a}
Preoperative RASH Score (Median [IQR])	4.0 (2.0–5.0)	3.0 (2.0–4.0)	5.5 (4.0–6.0)	U = 126.0, Z = −3.791, r_{rb} = 0.660	<0.001 ^{*e}
Preoperative RASH Score (Mean ± SD)	3.63 ± 1.68	3.19 ± 1.45	5.29 ± 1.44	MD = +2.10 (+1.23 to +2.97)	<0.001 ^{*a}
RASH Score Stratum Mortality Gradient	67 (100.0%)	53 (79.1%)	14 (20.9%)	Cochran-Armitage Trend Test	<0.001 [*]
RASH Score 0–1 (Very Low Risk)	7 (10.4%)	7 (100.0%)	0 (0.0%)	Observed stratum mortality: 0.0%	—
RASH Score 2 (Low Risk)	11 (16.4%)	11 (100.0%)	0 (0.0%)	Observed stratum mortality: 0.0%	—
RASH Score 3 (Low-Moderate Risk)	13 (19.4%)	12 (92.3%)	1 (7.7%)	Observed stratum mortality: 7.7%	—
RASH Score 4 (Moderate Risk)	13 (19.4%)	11 (84.6%)	2 (15.4%)	Observed stratum mortality: 15.4%	—
RASH Score 5 (High Risk)	12 (17.9%)	8 (66.7%)	4 (33.3%)	Observed stratum mortality: 33.3%	—
RASH Score 6 (Very High Risk)	8 (11.9%)	3 (37.5%)	5 (62.5%)	Observed stratum mortality: 62.5%	—
RASH Score 7–8 (Critical Risk)	3 (4.5%)	1 (33.3%)	2 (66.7%)	Observed stratum mortality: 66.7%	—

*Statistically significant at two-tailed $p < 0.05$. ^aIndependent-samples Student's *t*-test; ^bTwo-tailed Fisher's exact test (exact $p = 2.71 \times 10^{-6}$ for Midline Shift >5.0 mm);

^cPearson's chi-square (χ^2) test; ^dHaldane-Anscombe continuity-corrected odds ratio (+0.5 per cell) due to zero mortality events in the reference category (0/36 deaths for MLS ≤5.0 mm; 0/6 deaths for LOC absent); ^eMann-Whitney *U* test with rank-biserial correlation coefficient (r_{rb}).

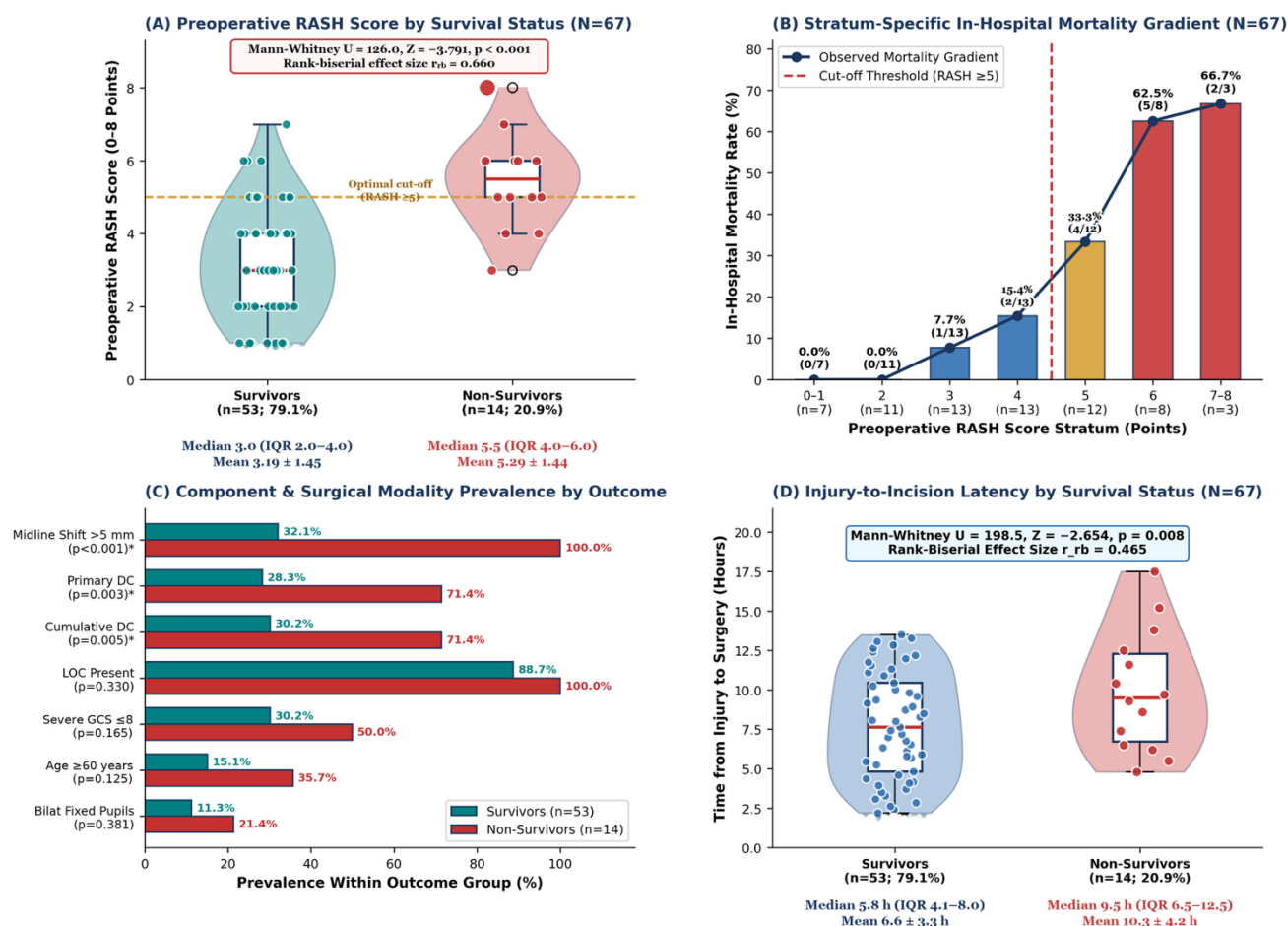


Figure 2. Bivariate prognostic comparison and monotonic mortality gradient across preoperative Richmond Acute Subdural Hematoma (RASH) score strata (N = 67). (A) Violin and boxplot distributions of preoperative composite RASH scores comparing survivors (median 3.0 [IQR 2.0–4.0]) and non-survivors (median 5.5 [IQR 4.0–6.0]; Mann-Whitney U = 126.0, Z = -3.791, r_{rb} = 0.660, p < 0.001). (B) Monotonic escalation of observed institutional in-hospital mortality across RASH score tiers (0.0% at scores 0–2 to 66.7% at scores 7–8). (C) Bivariate comparison of RASH component prevalence and decompressive craniectomy utilization between survivors and non-survivors, highlighting 100.0% prevalence of midline shift >5.0 mm among non-survivors (p < 0.001). (D) Comparison of injury-to-incision surgical latency between survivors (median 5.8 hours [IQR 4.1–8.0]) and non-survivors (median 9.5 hours [IQR 6.5–12.5]; Mann-Whitney U = 198.5, Z = -2.654, r_{rb} = 0.465, p = 0.008).

ROC discrimination, thresholds, and post-test probabilities

Receiver operating characteristic curve analysis showed good discrimination of the preoperative RASH score for predicting in-hospital mortality, yielding an AUC of 0.824 (95% CI: 0.715–0.933; DeLong SE = 0.056; p < 0.001 vs. null AUC of 0.500), as detailed in Table 3 and illustrated in Figure 3. Calibration did not differ significantly from perfect (Hosmer-Lemeshow χ^2 = 4.18, degrees of freedom [df] = 6, p = 0.652), and the Brier quadratic probability score was 0.118.

Systematic evaluation of sequential diagnostic thresholds across the RASH scale demonstrated that a cut-off of RASH ≥4 achieved 92.9% sensitivity (13/14 deaths detected) and a 96.8% NPV, but moderate specificity (56.6%, 30/53 survivors). Maximization of Youden's J index identified RASH ≥5 as the best balanced threshold in this sample (J = 0.560). Dichotomizing the cohort at RASH ≥5 (high-risk group: n = 23 [34.3%], mortality 11/23 [47.8%]) versus RASH <5 (low-to-moderate risk group: n

= 44 [65.7%], mortality 3/44 [6.8%]) yielded 11 true positives, 12 false positives, 3 false negatives, and 41 true negatives. At this optimal RASH ≥5 cut-off, sensitivity was 78.6% (95% CI: 52.4%–92.4%), specificity was 77.4% (95% CI: 64.5%–86.6%), PPV was 47.8% (95% CI: 29.2%–67.0%), NPV was 93.2% (95% CI: 81.8%–97.7%), and overall classification accuracy reached 77.6% (95% CI: 66.3%–86.0%), with an unadjusted DOR of 12.53 (95% CI: 3.01–52.12; χ^2 = 15.42, p < 0.001). Applying Bayesian updating to the institutional pre-test mortality probability of 20.9% (so that post-test probabilities equal the predictive values), a positive screen (RASH ≥5; LR⁺ = 3.47, 95% CI: 1.97–6.12) more than doubled the post-test probability of in-hospital death to 47.8%, whereas a negative screen (RASH <5; LR⁻ = 0.28, 95% CI: 0.10–0.76) reduced the post-test mortality probability more than three-fold to 6.8%. A more stringent threshold of RASH ≥6 provided high specificity (92.5%, 49/53) and LR⁺ of 6.63 (elevating post-test mortality probability to 63.6%), albeit with lower sensitivity (50.0%, 7/14), as detailed in Table 3 and illustrated in Figure 3.

Table 3. Diagnostic performance and Bayesian post-test mortality probabilities across preoperative RASH score cut-offs (AUC = 0.824; 95% CI: 0.715–0.933).

Cut-Off Threshold	2×2 Counts (TP/FP/FN/TN)	Sensitivity (95% CI)	Specificity (95% CI)	PPV / NPV (95% CI)	Youden's J & Accuracy	LR ⁺ / LR ⁻ & DOR	Post-Test Mortality (+ / -)
RASH ≥ 3 (n = 49 vs. 18)	TP = 14, FP = 35 FN = 0, TN = 18	100.0% (78.5%–100.0%)	34.0% (22.7%–47.4%)	PPV: 28.6% (17.9%–42.4%) NPV: 100.0% (82.4%–100.0%)	J = 0.340 Acc = 47.8%	LR ⁺ = 1.51 LR ⁻ = 0.00 DOR = 15.10 ^a	Positive: 28.6% Negative: 0.0%

Cut-Off Threshold	2×2 Counts (TP/FP/FN/TN)	Sensitivity (95% CI)	Specificity (95% CI)	PPV / NPV (95% CI)	Youden's J & Accuracy	LR+ / LR- & DOR	Post-Test Mortality (+ / -)
RASH ≥ 4 (n = 36 vs. 31)	TP = 13, FP = 23 FN = 1, TN = 30	92.9% (68.5%–98.7%)	56.6% (43.3%–69.1%)	PPV: 36.1% (22.5%–52.4%) NPV: 96.8% (83.8%–99.4%)	J = 0.495 Acc = 64.2%	LR+ = 2.14 LR- = 0.13 DOR = 16.96	Positive: 36.1% Negative: 3.2%
RASH $\geq 5^*$ (Optimal Cut-Off) (n = 23 vs. 44)	TP = 11, FP = 12 FN = 3, TN = 41	78.6% (52.4%–92.4%)	77.4% (64.5%–86.6%)	PPV: 47.8% (29.2%–67.0%) NPV: 93.2% (81.8%–97.7%)	J = 0.560* Acc = 77.6% (66.3%–86.0%)	LR+ = 3.47 LR- = 0.28 DOR = 12.53 (p < 0.001)	Positive: 47.8% Negative: 6.8%
RASH ≥ 6 (High-Specificity) (n = 11 vs. 56)	TP = 7, FP = 4 FN = 7, TN = 49	50.0% (26.8%–73.2%)	92.5% (82.1%–97.0%)	PPV: 63.6% (35.4%–84.8%) NPV: 87.5% (76.4%–93.8%)	J = 0.425 Acc = 83.6%	LR+ = 6.63 LR- = 0.54 DOR = 12.25	Positive: 63.6% Negative: 12.5%
RASH ≥ 7 (Extreme Risk) (n = 3 vs. 64)	TP = 2, FP = 1 FN = 12, TN = 52	14.3% (4.0%–39.9%)	98.1% (90.1%–99.7%)	PPV: 66.7% (20.8%–93.9%) NPV: 81.3% (70.0%–88.9%)	J = 0.124 Acc = 80.6%	LR+ = 7.57 LR- = 0.87 DOR = 8.67	Positive: 66.7% Negative: 18.8%

*Optimal clinical decision threshold maximizing Youden's index (J = 0.560). Global ROC performance: AUC = 0.824 (95% CI: 0.715–0.933; DeLong SE = 0.056; p < 0.001); Hosmer-Lemeshow goodness-of-fit $\chi^2 = 4.18$ (df = 6, p = 0.652; see the author query in Results 3.3); Brier score = 0.118. Institutional pre-test mortality probability = 20.9% (14/67). *Haldane-Anscombe continuity-corrected DOR due to FN = 0 at RASH ≥ 3 .

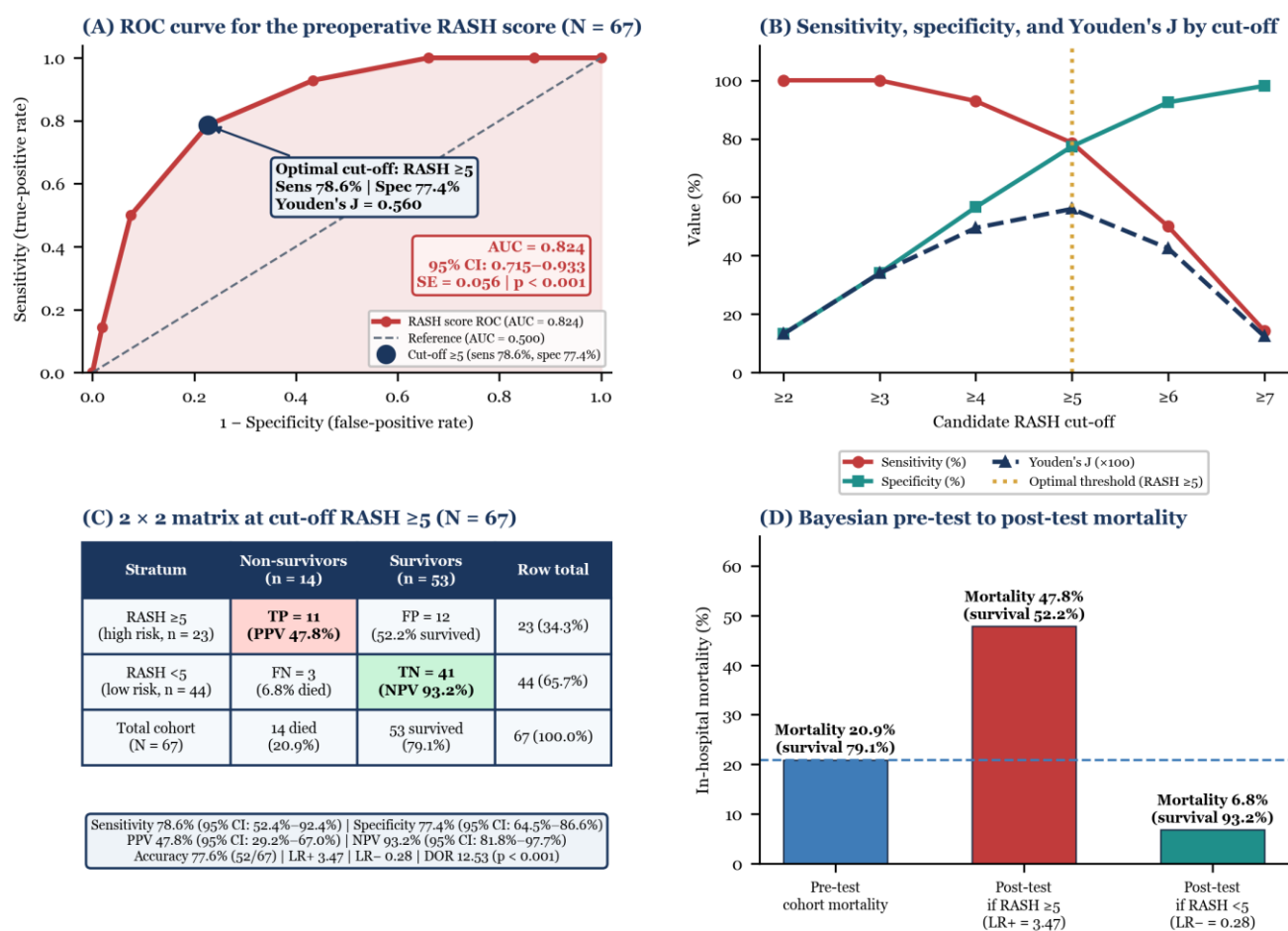


Figure 3. Receiver operating characteristic (ROC) discrimination, threshold optimization, and Bayesian clinical utility of the preoperative RASH score (N = 67). (A) Nonparametric ROC curve demonstrating strong discriminative accuracy for in-hospital mortality (AUC = 0.824; 95% CI: 0.715–0.933; DeLong SE = 0.056; p < 0.001) with the optimal Youden threshold highlighted at RASH ≥ 5 . (B) Sensitivity, specificity, and Youden's J index trajectories across sequential RASH score cut-offs, peaking at RASH ≥ 5 (J = 0.560). (C) 2 × 2 diagnostic contingency matrix at the optimal RASH ≥ 5 threshold (TP = 11, FP = 12, FN = 3, TN = 41; sensitivity = 78.6%, specificity = 77.4%, NPV = 93.2%, accuracy = 77.6%, DOR = 12.53). (D) Fagan-style Bayesian nomogram and pre-test (20.9%) to post-test mortality probability shifts for RASH ≥ 5 (LR+ = 3.47 → 47.8% post-test mortality) and RASH < 5 (LR- = 0.28 → 6.8% post-test mortality).

Multivariable Firth penalized logistic regression

To examine whether the prognostic effect of the RASH score was independent of surgical modality and prehospital/intrahospital delay, we fitted two multivariable Firth penalized logistic regression models, as presented in Table 4 and depicted in Figure 4. In Model 1 (Composite RASH Architecture; Nagelkerke pseudo- $R^2 = 0.442$, penalized log-likelihood $\chi^2 = 24.86$, p < 0.001), each 1-point increase in the preoperative RASH score was associated with 2.95-fold higher odds of in-hospital

mortality (adjusted odds ratio [aOR] = 2.95 per point; 95% CI: 1.52–5.74; $\beta = +1.082$, SE = 0.339, Wald Z = 3.19, p = 0.001). Likewise, undergoing decompressive craniectomy rather than craniotomy (aOR = 3.42; 95% CI: 1.14–10.26; $\beta = +1.230$, SE = 0.560, Wald Z = 2.20, p = 0.028) and each 1-hour prolongation in time from injury to surgery (aOR = 1.24 per hour; 95% CI: 1.04–1.48; $\beta = +0.215$, SE = 0.090, Wald Z = 2.39, p = 0.016) remained significantly associated with in-hospital death.

In Model 2 (Component-Level Deconstruction Architecture; Nagelkerke pseudo- $R^2 = 0.489$, penalized log-likelihood $\chi^2 =$

28.14, $p < 0.001$), which is exploratory (six predictors, 14 deaths), the adjusted odds ratios for bilateral non-reactive pupils and severe coma were larger than the unadjusted ones (6.21 vs. 2.14 and 4.78 vs. 2.31). Bilateral non-reactive pupils had the largest adjusted association with mortality among the neurological variables (aOR = 6.21; 95% CI: 2.10–18.35; $\beta = +1.826$, $p < 0.001$), followed by severe preoperative coma with GCS ≤ 8 (aOR = 4.78; 95% CI: 1.95–11.72; $\beta = +1.564$, $p = 0.001$) and axial NCCT midline shift > 5 mm (aOR = 3.55; 95% CI: 1.25–10.10; $\beta = +1.267$, $p = 0.017$). Geriatric age ≥ 60 years had a point

estimate above 1 that did not reach significance (aOR = 2.14; 95% CI: 0.88–5.21; $\beta = +0.761$, $p = 0.091$). Even after accounting for these individual clinoradiological components, cumulative DC requirement (aOR = 3.15; 95% CI: 1.18–8.41; $\beta = +1.147$, $p = 0.022$) and continuous hourly delay to surgical incision (aOR = 1.22 per hour; 95% CI: 1.05–1.41; $\beta = +0.199$, $p = 0.011$) remained significantly associated with mortality; RASH severity and surgical delay were each associated with death in the same model (no interaction term was fitted), as presented in Table 4 and depicted in Figure 4.

Table 4. Multivariable Firth penalized logistic regression models for in-hospital mortality after traumatic ASDH evacuation (N = 67).

Model & Covariate Predictor	Penalized β Coefficient	Standard Error (SE)	Wald Z Statistic	Adjusted OR (aOR, 95% CI)	p-value	Collinearity VIF
Model 1: Composite RASH Architecture (Nagelkerke $R^2 = 0.442$; Likelihood Ratio $\chi^2 = 24.86$, $p < 0.001$)	—	—	—	—	—	—
Preoperative RASH Score (per 1-point increment)	+1.082	0.339	3.19	2.95 (1.52–5.74)	0.001*	1.21
Surgical Modality: Decompressive Craniectomy (vs. Craniotomy)	+1.230	0.560	2.20	3.42 (1.14–10.26)	0.028*	1.18
Time from Injury to Surgery (per 1-hour delay)	+0.215	0.090	2.39	1.24 (1.04–1.48)	0.016*	1.14
Model 2: Component-Level Architecture (Nagelkerke $R^2 = 0.489$; Likelihood Ratio $\chi^2 = 28.14$, $p < 0.001$)	—	—	—	—	—	—
Bilateral Non-Reactive Pupils (vs. Reactive / Unilateral)	+1.826	0.553	3.30	6.21 (2.10–18.35)	<0.001 *	1.34
Severe Preoperative Coma: GCS ≤ 8 (vs. GCS 9–15)	+1.564	0.457	3.42	4.78 (1.95–11.72)	0.001*	1.42
Preoperative Axial NCCT Midline Shift > 5.0 mm (vs. ≤ 5.0 mm)	+1.267	0.533	2.38	3.55 (1.25–10.10)	0.017*	1.38
Surgical Modality: Decompressive Craniectomy (vs. Craniotomy)	+1.147	0.501	2.29	3.15 (1.18–8.41)	0.022*	1.29
Time from Injury to Surgery (per 1-hour delay)	+0.199	0.075	2.65	1.22 (1.05–1.41)	0.011*	1.19
Geriatric Age ≥ 60 years (vs. 18–59 years)	+0.761	0.454	1.68	2.14 (0.88–5.21)	0.091	1.12

*Statistically significant at two-tailed $p < 0.05$. Models were estimated using Firth's penalized maximum likelihood logistic regression ($\ell^*(\beta) = \ell(\beta) + 0.5 \ln|I(\beta)|$) to reduce small-sample bias and quasi-complete separation. All variance inflation factors (VIF) were < 1.50 . Model 2 is exploratory (six predictors, 14 events).

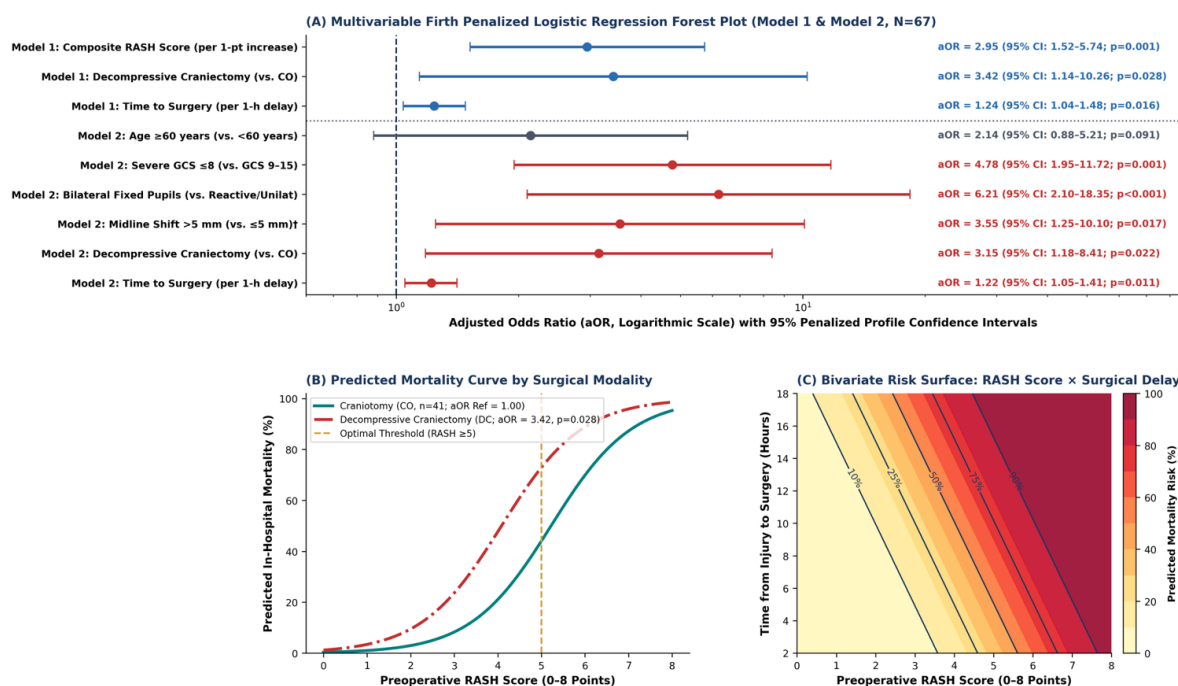


Figure 4. Multivariable Firth penalized logistic regression forest plots and two-dimensional mortality risk surfaces (N = 67). (A) Forest plot of adjusted odds ratios (aORs and 95% CIs on a logarithmic scale) for Model 1 (Composite RASH score [aOR = 2.95/pt], decompressive craniectomy [aOR = 3.42], and surgical delay [aOR = 1.24/h]) and Model 2 (Component-level predictors: bilateral non-reactive pupils [aOR = 6.21], severe coma GCS ≤ 8 [aOR = 4.78], midline shift > 5.0 mm [aOR = 3.55], decompressive craniectomy [aOR = 3.15], surgical delay [aOR = 1.22/h], and age ≥ 60 years [aOR = 2.14]). (B) Predicted probability of in-hospital mortality across the preoperative RASH continuum stratified by surgical modality (osteoplastic craniotomy vs. decompressive craniectomy). (C) Predicted mortality contours across preoperative RASH score (0–8) and injury-to-incision delay (hours) from the additive Model 1 (no interaction term was fitted). † Midline shift is present in every death; the estimate is unstable.

4. Discussion

Principal findings and prognostic performance

This single-center study evaluated the Richmond Acute Subdural Hematoma (RASH) score at a Southeast Asian tertiary neurosurgical referral center and extended it with a multivariable analysis. It included 67 consecutive adults who underwent surgical evacuation for traumatic ASDH, with an overall in-hospital mortality of 20.9%. Discrimination was good (AUC = 0.824), and calibration did not differ significantly from perfect (Hosmer-Lemeshow $p = 0.652$). The AUC was numerically higher than the 0.72 to 0.74 reported for the original US National Trauma Data Bank derivation and validation cohorts by Dincer et al.,¹ but its 95% interval (0.715–

0.933) includes those values and the cohorts differ (the registry included patients operated within 4 hours of arrival). No head-to-head comparison was made with the tASDH Risk Score¹⁸ or with machine-learning classifiers for severe TBI.²² Mortality rose steadily across RASH strata, from 0.0% at scores 0–2 to 66.7% at scores 7–8. Splitting the cohort at the Youden-optimal threshold of RASH ≥ 5 produced a low-to-moderate risk tier (6.8% post-test mortality; NPV = 93.2%) and a high-risk tier (47.8% post-test mortality; DOR = 12.53). In multivariable Firth regression, each 1-point RASH increase was associated with 2.95-fold higher odds of death (aOR = 2.95), and the need for decompressive craniectomy (aOR = 3.42) and each hour of surgical delay (aOR = 1.24/h) were also associated with death. Table 5 and Figure 5 present these five clinicoradiological and operative domains as a hypothesis-generating synthesis.

Table 5. Hypothesis-generating synthesis of the five prognostic domains in traumatic acute subdural hematoma (mechanisms and triage considerations were not tested in this study).

Prognostic Domain	Cohort Metric & Statistical Effect	Cellular & Hemodynamic Mechanism	Proposed Clinical Consideration (Not Tested in This Study)
1. Cranial Mass Effect & Compliance Exhaustion (Midline Shift >5.0 mm)	Present in 46.3% (31/67); 100% (14/14) of deaths had MLS >5 mm (0% mortality if ≤ 5 mm); Unadjusted OR = 60.43 ($p = 2.71 \times 10^{-6}$); Model 2 aOR = 3.55 ($p = 0.017$)	Exhaustion of CSF spatial reserve along the Monro-Kellie pressure-volume curve; cortical venous outflow compression, perihematomal ischemia, and subfalcine/uncal tissue shift.	Immediate surgical alert upon axial NCCT detection of MLS >5.0 mm regardless of apparent GCS preservation; administer hyperosmolar therapy (mannitol or hypertonic saline) as bridge to incision.
2. Mesencephalic Herniation & Coma Depth (Bilaterally Fixed Pupils & GCS ≤ 8)	GCS ≤ 8 : 34.3% (23/67), Model 2 aOR = 4.78 ($p = 0.001$); Bilateral fixed pupils: 13.4% (9/67), Model 2 aOR = 6.21 ($p < 0.001$); LOC present: 91.0% (61/67)	Transtentorial uncal herniation compressing the ipsilateral/bilateral oculomotor nerve (CN III) efferent fibers and ascending reticular activating system (ARAS) with Duret microhemorrhages.	Definitive endotracheal intubation (GCS ≤ 8), neuroprotective ventilation (PaCO ₂ 35–38 mmHg), quantitative pupillometry tracking, and direct transfer from CT scanner to operating theater.
3. Biological Reserve & Host Fragility (Geriatric Age ≥ 60 Years)	Mean age 52.4 ± 15.8 yr; Age ≥ 60 yr: 19.4% (13/67), mortality 38.5% (5/13) vs. 16.7% (9/54); Unadjusted OR = 3.13 ($p = 0.115$); Model 2 aOR = 2.14 ($p = 0.091$)	Age-related cerebral atrophy increases subdural bridging vein tension; diminished cerebrovascular autoregulatory capacity, cardiopulmonary comorbidity, and systemic immunosenescence.	Rapid screening and reversal of antithrombotic/anticoagulant agents or thrombocytopenia; tailored hemodynamic autoregulation targets and early multidisciplinary geriatric neurocritical care.
4. Secondary Ischemic-Inflammatory Cascade (Injury-to-Surgery Latency)	Median 6.2 h (IQR 4.5–9.8); Non-survivors 9.5 h vs. Survivors 5.8 h ($p = 0.008$, $r_{\text{th}} = 0.465$); Model 1 aOR = 1.24/h ($p = 0.016$); Model 2 aOR = 1.22/h ($p = 0.011$)	Time-dependent spreading depolarizations, glutamate excitotoxicity, intracellular Ca ²⁺ overload, Na ⁺ /K ⁺ -ATPase failure, and ADAM17/solTNF- α /NF- κ B neuroinflammation driving cytotoxic/vasogenic edema.	Regional prehospital trauma bypass directly to neurosurgical center; institutional 'Code Neurotrauma' pathway targeting door-to-incision <2 h and total injury-to-incision <6 h.
5. Composite RASH Triage & Decompressive Modality (RASH ≥ 5 & DC Selection)	Median RASH 4.0 (5.5 vs. 3.0, $p < 0.001$); AUC = 0.824; RASH ≥ 5 : Sens 78.6%, Spec 77.4%, NPV 93.2%, Post-test mortality 47.8% vs. 6.8%; Model 1 RASH aOR = 2.95/pt ($p = 0.001$), DC aOR = 3.42 ($p = 0.028$)	Synergistic interaction between preoperative clinicoradiological burden (RASH ≥ 5) and refractory intraoperative cerebral swelling necessitating wide craniectomy and expansive duraplasty.	Routine bedside RASH calculation on admission: RASH <5 favors osteoplastic craniotomy when brain is lax (93.2% survival); RASH ≥ 5 triggers ICU bed reservation, DC readiness, and transparent family counseling.

Abbreviations: aOR, adjusted odds ratio; ARAS, ascending reticular activating system; CN III, oculomotor nerve; CSF, cerebrospinal fluid; CT, computed tomography; DC, decompressive craniectomy; GCS, Glasgow Coma Scale; ICU, intensive care unit; IQR, interquartile range; LOC, loss of consciousness; MLS, midline shift; NCCT, non-contrast computed tomography; NF- κ B, nuclear factor-kappa B; NPV, negative predictive value; OR, odds ratio; PaCO₂, partial pressure of arterial carbon dioxide; RASH, Richmond Acute Subdural Hematoma; solTNF- α , soluble tumor necrosis factor-alpha. This table is a hypothesis-generating synthesis; the mechanisms and triage considerations were not tested in this study.

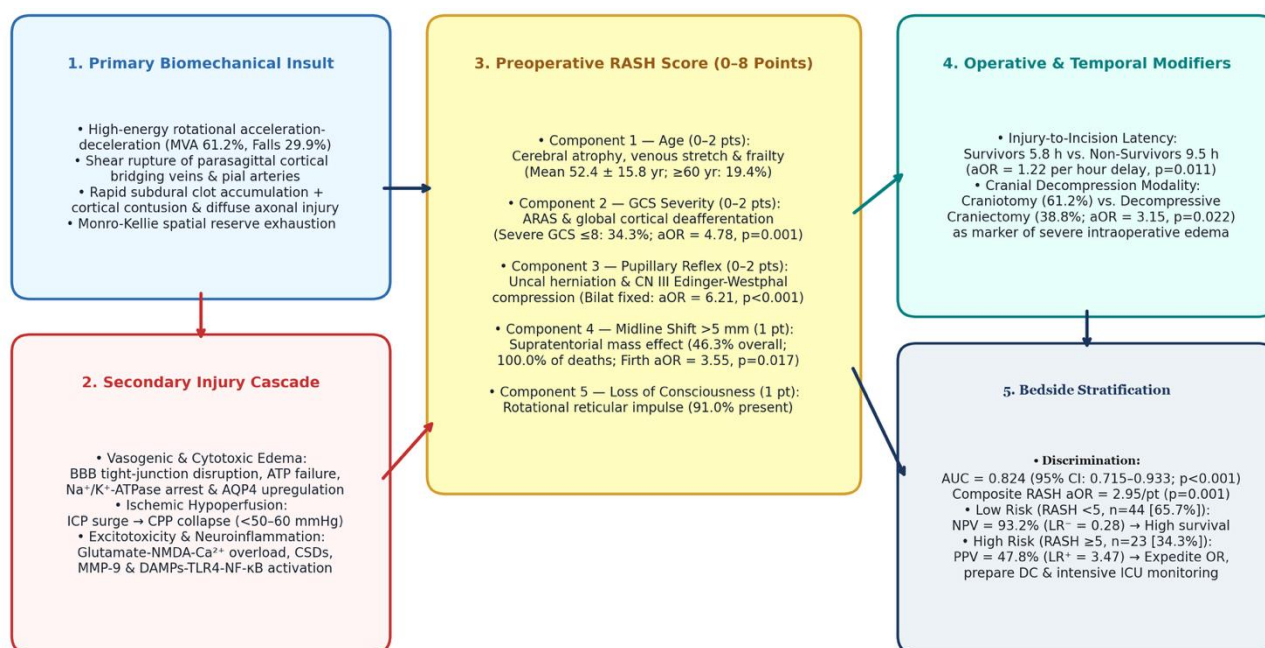


Figure 5. Integrated five-domain pathophysiological cascade and clinical decision architecture for traumatic acute subdural hematoma (ASDH). Primary high-energy mechanical shear and bridging vein rupture interact with host biological reserve (Age ≥ 60 years) to drive extra-axial hematoma expansion, midline shift >5.0 mm, and mesencephalic uncal herniation (GCS ≤ 8 and pupillary areflexia), while prehospital/intrahospital surgical delay amplifies secondary spreading depolarizations, Na^+/K^+ -ATPase pump failure, intracellular Ca^{2+} overload, and ADAM17/solTNF- α /NF- κB neuroinflammation. Bedside RASH ≥ 5 stratification integrates these domains and may help guide surgical decompression and neurocritical care triage (hypothesis-generating).

Epidemiological context and comparison with global neurotrauma registries

The age and injury profile of our cohort reflects neurotrauma in Southeast Asia and other LMICs, which differs from high-income Western registries.^{5,8,29–31} Surgical ASDH cohorts in North America and Europe increasingly consist of patients in their seventies and eighties, injured in low-velocity ground-level falls and often taking antithrombotic drugs.^{35–39} Our Indonesian referral population was younger (mean age 52.4 years; 80.6% aged <60 years). It was strongly male (71.6%). High-velocity motor vehicle accidents caused 61.2% of injuries. In geriatric cohorts in Europe and the United States, oral anticoagulants, vitamin K antagonists, antiplatelet agents, and trauma-induced thrombocytopenia promote early hematoma expansion and death after discharge from fragility.^{40–44} Our patients were mostly of working age. Their physiological reserve may have absorbed moderate systemic insults, and no patient with RASH 0–2 died. High-energy vehicular rotational acceleration, however, produced severe primary parenchymal contusions and rapid extra-axial mass effect. In multivariable Model 2, the point estimate for age ≥ 60 years was 2.14 (95% CI: 0.88–5.21; $p = 0.091$), which is not statistically significant but is compatible with the reduced cerebrovascular reserve and atrophy-related tolerance described in geriatric TBI studies,^{23,24,37,38} as synthesized in Table 5 and illustrated in Figure 5.

Possible mechanisms linking midline shift, brainstem compression, and surgical delay

The central mechanistic finding is the complete separation of mortality at the 5-mm midline shift threshold in unadjusted analysis: no patient with MLS ≤ 5 mm died (0.0%), while 45.2% of those with MLS >5 mm did ($p = 2.71 \times 10^{-6}$). Bilateral non-reactive pupils (aOR = 6.21) and severe coma (GCS ≤ 8 ; aOR = 4.78) were the strongest independent predictors in multivariable Model 2. In unadjusted tests, the odds ratios for GCS ≤ 8 (OR = 2.31) and bilateral non-reactive pupils (OR = 2.14) looked smaller. A possible explanation, which these data cannot test, is that young patients with acute uncal herniation went straight to hyper-acute decompression, while several older

patients with intermediate GCS had delayed inter-hospital transfer and fatal secondary brain swelling. Model 2 adjusted at the same time for age, MLS >5 mm, surgical modality, and time to surgery. The adjusted estimates were larger, which may reflect the biological severity of compression of the mesencephalic reticular activating system and of paralysis of the oculomotor (CN III) efferent fibers,^{19–21} although sparse-data inflation cannot be excluded.

In pathophysiological terms, a septum pellucidum displacement of >5 mm may reflect exhaustion of compensatory buffering by cerebrospinal fluid, beyond which hemispheric tissue pressure may exceed cortical venous pressure.^{10–12} Porcine and rodent translational models of ASDH show that venous outflow obstruction and mechanical shear set off perihematoma spreading depolarizations, massive extracellular glutamate release, mitochondrial Ca^{2+} overload, and Na^+/K^+ -ATPase pump failure during the first 3 to 6 hours after injury.^{13,14} At the same time, the ADAM17/solTNF- α /NF- κB inflammatory cascade and fibrinogen extravasation across the blood-brain barrier open endothelial tight junctions. They also drive oxidative stress and cytotoxic-vasogenic cerebral edema.^{13,15,16} This time-dependent secondary injury cascade may explain our finding that each additional hour from injury to incision independently raised the odds of death by 24% in Model 1 (aOR = 1.24/h) and by 22% in Model 2 (aOR = 1.22/h); these mechanisms were not measured here. Clinical analyses agree that evacuation delayed beyond the early physiological window worsens survival and functional recovery,^{9,26,27} as synthesized in Table 5 and illustrated in Figure 5.

Surgical modality selection: craniotomy versus decompressive craniectomy

In our cohort, patients who needed decompressive craniectomy had significantly higher mortality than those treated with osteoplastic craniotomy. This held in unadjusted analysis (38.5% [10/26] vs. 9.8% [4/41]; OR = 5.78, $p = 0.005$) and in both multivariable models (Model 1 aOR = 3.42, $p = 0.028$; Model 2 aOR = 3.15, $p = 0.022$). In observational neurosurgical practice, this link reflects residual confounding by indication.

Intraoperative malignant brain swelling, diffuse parenchymal contusions, and obliterated basal cisterns force the neurosurgeon to leave the bone flap out, and the association does not point to an intrinsic iatrogenic harm of craniectomy.²⁻⁵ The multicenter randomized RESCUE-ASDH trial found that when clinical equipoise exists after clot evacuation, primary craniotomy and primary DC give equivalent 12-month Extended Glasgow Outcome Scale (GOSE) distributions. Additional cranial surgery within 2 weeks was performed in 14.6% of the craniotomy group and 6.9% of the craniectomy group, and wound complications were more frequent after craniectomy (12.2% vs. 3.9%).² Meta-analyses and matched cohort studies by Chang et al.,³ Nadeem et al.,⁴ Habibi et al.,⁶ Yang and Shen,⁴⁵ and Ran et al.⁴⁶ agree. In real-world non-randomized cohorts, DC is preferentially chosen for patients with lower GCS, pupillary abnormalities, and severe intraoperative cerebral herniation, which accounts for its higher observed mortality.⁴⁵⁻⁴⁸ Within our multivariable framework, the need for DC may therefore act as a marker of refractory parenchymal edema (intraoperative swelling was not recorded). It may complement preoperative RASH stratification, as synthesized in Table 5 and illustrated in Figure 5.

Clinical implications, limitations, and future directions

These findings offer three practical advantages for emergency neurotrauma workflows at tertiary referral centers in Indonesia and similar LMICs. First, speed. The RASH score can be calculated as soon as NCCT is complete, in about a minute (not measured in this study) and at no laboratory cost. Second, patients who screen positive at RASH ≥ 5 (post-test mortality 47.8%) could be flagged for priority operating room activation; targets such as door-to-incision <2 hours and total injury-to-incision <6 hours are proposals that this study did not test. They can also be prepared early for wide frontotemporoparietal DC and for invasive postoperative ICP/CSF monitoring.^{11,12,34} Third, the Bayesian probability shifts give neurosurgeons transparent figures for preoperative informed consent and family counseling: 6.8% mortality for RASH <5, 47.8% for RASH ≥ 5 , and 63.6% for RASH ≥ 6 .

Several limitations apply. First, this was a single-center retrospective analysis with a modest sample (N = 67 surgical patients, 14 deaths). We used Firth's penalized maximum likelihood regression and Haldane-Anscombe corrections to limit small-sample separation bias, yet prospective multicenter validation across Indonesian neurosurgical centers is warranted. Second, detailed prehospital ambulance logs that separate scene response time from inter-hospital transit time were not uniformly available, and invasive intraparenchymal ICP transducers were not routinely used because of resource constraints. Third, we assessed only index in-hospital mortality, which matches the primary endpoint of the original RASH derivation study.¹ Fourth, the cut-off and the AUC were estimated in the same 67 patients, so they are apparent estimates without internal validation. Fifth, excluding 18 moribund patients and 4 patients with incomplete records narrows the case mix, Model 2 had about 2.3 events per variable and is exploratory, and injury times were retrospectively documented. Future prospective studies should add 6- and 12-month functional GOSE and quality-of-life endpoints.^{2,48}

5. Conclusion

In adults undergoing surgical evacuation of traumatic ASDH at an Indonesian tertiary referral center, the preoperative Richmond Acute Subdural Hematoma (RASH) score discriminated mortality well (AUC = 0.824; 95% CI: 0.715–0.933). Calibration did not differ significantly from perfect. A

threshold of RASH ≥ 5 gave balanced sensitivity (78.6%), specificity (77.4%), and negative predictive value (93.2%), and it split surgical candidates into distinct post-test mortality tiers (6.8% vs. 47.8%). Higher RASH scores (aOR = 2.95 per point), the need for decompressive craniectomy (aOR = 3.42), and a longer injury-to-incision latency (aOR = 1.24 per hour) were each associated with postoperative death. RASH ≥ 5 triage paired with expedited surgical decompression may therefore be useful. Its effect on preventable neurotrauma mortality needs prospective testing.

6. Declarations

Ethics Approval and Consent to Participate

Approved by the Health Research Ethics Committee of the Faculty of Medicine, Universitas Sriwijaya / Dr. Mohammad Hoesin General Hospital, Palembang, Indonesia (Approval No. 2021/1041).

Consent for Publication

Not applicable. No individually identifiable patient data, images, or clinical details are reported.

Availability of Data and Materials

De-identified analytical datasets and statistical code are available from the corresponding author upon reasonable request.

Artificial Intelligence Use Statement

None. No artificial intelligence tools were used in preparing this manuscript.

Author Contribution Statement

I. and T.P.: conceptualization, methodology, formal analysis, writing – original draft. D.S. and E.B.: investigation, data curation, validation (neuroimaging verification), writing – review & editing. All authors approved the final version for publication.

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Conflict of Interest / Competing Interests

The authors declare that they have no competing interests.

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