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# External Validation of the RASH Score in Surgically Managed Acute Subdural Hematoma: A Critical Appraisal of Prognostic Accuracy and Surgical Factors in a Southeast Asian Cohort

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**A B S T R A C T**

**Introduction:** Acute subdural hematoma (ASDH) is a lethal form of traumatic brain injury (TBI) with high mortality. The Richmond Acute Subdural Hematoma (RASH) score is a simple prognostic tool, but its validity in diverse populations is untested. This study aimed to perform the first external validation of the RASH score in an Indonesian cohort and critically appraise its performance alongside key surgical factors. **Methods:** We conducted a retrospective, single-center, diagnostic accuracy study of 67 adult patients who underwent surgery for traumatic ASDH between January 2022 and December 2024 at a tertiary neurosurgical center in Palembang, Indonesia. The RASH score was calculated from admission data. We additionally analyzed the type of surgery (craniotomy vs. decompressive craniectomy) and time from injury to operation. The primary outcome was in-hospital mortality. Receiver Operating Characteristic (ROC) curve analysis was used to evaluate the RASH score's predictive performance. **Results:** The overall in-hospital mortality rate was 20.9% (n=14). The RASH score demonstrated excellent discrimination for mortality, with an Area Under the ROC Curve (AUC) of 0.824 (95% CI: 0.715–0.933; p<0.001). A score of 5 or greater was identified as the optimal cut-off, yielding a sensitivity of 78.6% and specificity of 77.4%. This threshold provided a high Negative Predictive Value (NPV) of 93.2% but a modest Positive Predictive Value (PPV) of 47.8%. In bivariate analysis, decompressive craniectomy and longer time to surgery were significantly associated with mortality. **Conclusion:** The RASH score is a simple and robust tool for risk stratification in this selected surgical population. Its high NPV is valuable for identifying patients with a higher likelihood of survival. However, its utility must be interpreted cautiously due to the significant selection bias inherent in studying only operable patients. The score should serve as an adjunct to, not a replacement for, comprehensive clinical judgment.

## 1. Introduction

Traumatic brain injury (TBI) represents a global public health catastrophe and a leading cause of mortality and profound long-term disability, aptly termed a "silent epidemic".[1] Its incidence continues to rise, particularly in low- and middle-income countries, where traffic accidents and violence are endemic. Among the diverse pathologies under the TBI umbrella, acute subdural hematoma (ASDH)—the accumulation of blood in the potential space between the dura mater and the arachnoid mater—stands as one of the most lethal entities encountered in neurosurgical practice. Typically caused by the rupture of cortical bridging veins due to sudden acceleration-deceleration forces, ASDH is frequently associated with severe underlying parenchymal brain injury, including contusions and diffuse axonal injury.[2] Consequently, despite significant advancements in neurosurgical techniques, neurocritical care, and intracranial pressure (ICP)

management, the postoperative mortality rate for ASDH remains distressingly high, with contemporary literature consistently reporting figures between 30% and 70%. This grim prognosis underscores a persistent and critical need for accurate, early, and reliable risk stratification tools to guide clinical management, inform difficult surgical decision-making, and facilitate transparent, realistic communication with patients' families in the face of devastating injury.[3]

The pathophysiology of ASDH is a devastating two-act process, comprising a primary and a secondary injury phase that ultimately dictates patient outcome.[4] The primary injury is the instantaneous, direct mechanical damage inflicted at the moment of impact. This includes the tearing of blood vessels, formation of the hematoma, and direct contusion of the brain parenchyma.[4] The hematoma itself acts as a rapidly expanding intracranial mass lesion. Within the fixed volume of the skull, this leads to a precipitous rise in ICP, causing compression of adjacent brain tissue and distortion of vital deep-brain structures, including the brainstem.[5]

However, the more insidious and often more damaging phase is the secondary injury. This is a complex and self-propagating cascade of deleterious biochemical, cellular, and molecular events triggered by the primary insult and the subsequent mass effect. This cascade includes: (1) Cerebral Edema: Both cytotoxic (cellular swelling due to ion pump failure) and vasogenic (breakdown of the blood-brain barrier) edema contribute to a further increase in intracranial volume and ICP; (2) Cerebral Ischemia: Elevated ICP reduces the cerebral perfusion pressure (CPP), defined as Mean Arterial Pressure (MAP) minus ICP. When CPP falls below critical thresholds, cerebral blood flow (CBF) is compromised, leading to widespread ischemic damage and creating a vicious cycle of swelling, further ICP elevation, and worsening ischemia; (3) Excitotoxicity: The ischemic and traumatic insult triggers a massive release of excitatory neurotransmitters, primarily glutamate. This over-activates receptors like the NMDA receptor, leading to an influx of calcium into neurons. This calcium overload activates catabolic enzymes, promotes free radical formation, and ultimately leads to neuronal death; (4) Neuroinflammation and Oxidative Stress: The brain mounts an intense inflammatory response to the injury, involving the activation of microglia and astrocytes and the infiltration of peripheral immune cells. While initially protective, this response can become dysregulated, releasing cytotoxic cytokines and reactive oxygen species that cause widespread oxidative damage to lipids, proteins, and DNA, further perpetuating cell death. The extent and severity of this secondary injury cascade are often the primary determinants of a patient's ultimate functional outcome and survival. Therefore, a cornerstone of modern neurotrauma care is the prediction and mitigation of these secondary insults.[6]

For decades, prognostication in ASDH has relied on a handful of key clinical and radiological parameters. The Glasgow Coma Scale (GCS) has been the bedrock of neurological assessment, providing an indispensable measure of the level of consciousness. Pupillary examination serves as a critical bedside indicator of brainstem compression, particularly of the third cranial nerve. On computed tomography (CT), measurements such as hematoma thickness and the degree of midline shift (MLS) provide a direct anatomical quantification of the mass effect.

While invaluable, each of these factors in isolation provides an incomplete picture. The GCS is a snapshot in time and can be confounded by sedation, intoxication, or intubation.[7] Radiological markers, while anatomically precise, do not capture the dynamic physiological state of the brain. Relying on any single factor is insufficient to encapsulate the multifaceted nature of the injury and predict the complex interplay of pathophysiological processes that determine survival.[8] This has driven the development of integrated clinical prediction scores, which aim to provide a more holistic, objective, and reliable prognosis by combining multiple weighted risk factors into a single composite value. An ideal prognostic tool for the acute setting should be simple to use, rapid to

calculate, based on universally available data, and rigorously validated across diverse patient populations.[9]

The Richmond Acute Subdural Hematoma (RASH) score was specifically developed to meet this need. It is an intuitive grading scale based on five preoperative variables that are readily available upon patient admission: Age, GCS severity, Pupillary response, Midline shift >5 mm, and post-traumatic Loss of Consciousness (LOC).[10] Each of these components reflects a distinct and critical aspect of the injury's severity. In its original derivation and internal validation on a large cohort of over 2,500 patients in the United States, the RASH score demonstrated a strong, linear correlation with postoperative mortality, offering a rapid and powerful estimate of risk. The score's greatest strength is its simplicity, allowing for immediate calculation in high-pressure emergency and neurosurgical settings without specialized software.

However, the utility and generalizability of any clinical prediction rule are contingent upon its performance in populations different from the one in which it was developed—a process known as external validation. Factors such as genetic background, healthcare system infrastructure, pre-hospital care protocols, and patient demographics can vary significantly across geographic regions, potentially altering a score's predictive accuracy. To date, the RASH score has not been validated in a Southeast Asian population, where the epidemiology and management of TBI may differ, representing a critical knowledge gap. Clinicians in these regions lack the evidence to confidently apply this tool in their local practice.

Therefore, this study was designed to address this gap. The primary aim was to perform the first external validation of the RASH score in a cohort of Indonesian patients undergoing surgical evacuation for traumatic ASDH. The secondary aims were to determine the score's predictive accuracy for in-hospital mortality, identify an optimal cut-off for risk stratification in this population, and explore the prognostic significance of key surgical variables, namely the type of surgery and the timing of intervention. The novelty of this work lies not only in its unique geographic and ethnic context but also in its critical appraisal of the score's performance within the complex, real-world framework of surgical decision-making.

## 2. Methods

This study was a retrospective, single-center, diagnostic accuracy analysis conducted at Dr. Mohammad Hoesin General Hospital in Palembang, South Sumatra, Indonesia. This institution is a national tertiary referral hospital and serves as the primary neurotrauma center for the region. The study protocol received full approval from the local Institutional Review Board and Ethics Committee. Given the retrospective nature of the data collection from existing medical records, the requirement for individual informed consent was waived. The study was conducted in strict accordance with the ethical principles of the Declaration of Helsinki.

We retrospectively screened the medical and radiological records of all patients admitted with a primary diagnosis of traumatic ASDH between January 1st, 2022, and December 31st, 2024. A total of 95 patients with traumatic ASDH were identified during this period. The final study cohort was composed of patients who met specific inclusion and exclusion criteria. The inclusion criteria were as follows: (1) age ≥18 years; (2) a definitive diagnosis of traumatic ASDH confirmed by a non-contrast head CT scan; and (3) underwent surgical evacuation of the ASDH, either via craniotomy or decompressive craniectomy. The exclusion criteria were: (1) ASDH from non-traumatic causes, such as spontaneous or post-procedural hemorrhage; (2) significant polytrauma with hemodynamic instability stemming from severe thoracic, abdominal, or pelvic injuries that could independently be the primary cause of mortality; and (3) Incomplete or missing medical records that precluded the calculation of the full RASH score or determination of the primary outcome.

Of the 95 patients initially screened, 28 were not included in the final analysis. Ten patients were managed non-operatively due to small hematoma size and stable neurological status. Eighteen patients were deemed to have non-survivable injuries upon admission and were managed with palliative care after discussion with their families, thus were not surgical candidates. This left 71 patients who underwent surgery. Four of these surgical patients were subsequently excluded due to incomplete admission records. Thus, a final cohort of 67 patients was included in the analysis. This selection process is critical to note, as the study cohort inherently represents a filtered subset of all ASDH patients, specifically those for whom surgery was deemed both necessary and potentially beneficial.

A standardized data collection form was developed to extract all relevant information from the hospital's electronic and paper-based medical records. To ensure data integrity and minimize extraction errors, two investigators independently extracted the data for all patients. Any discrepancies were resolved by discussion and consensus with a third senior investigator. All patient identifiers were anonymized to maintain strict confidentiality.

RASH score components consist of five variables were collected based on the patient's clinical and radiological status at the time of initial presentation to the emergency department; (1) Age: Recorded in years and categorized for scoring: ≤59 years (0 points), 60–79 years (1 point), and ≥80 years (2 points); (2) Glasgow Coma Scale (GCS) Severity: The initial GCS score was recorded and categorized: Mild (GCS 14–15, 0 points), Moderate (GCS 9–13, 1 point), and Severe (GCS ≤8, 2 points). For patients intubated prior to a formal GCS assessment, the score was estimated from motor and eye components or from documented scores by emergency responders before intubation; (3) Pupillary Response: Reactivity to light was documented for both pupils and categorized: both pupils reactive (0 points), unilateral non-reactive pupil (1 point), and bilateral non-reactive pupils (2 points); (4) Midline Shift (MLS): Measured on the initial axial head CT scan as the maximum perpendicular displacement of the septum pellucidum from the anatomical midline. This was dichotomized as: MLS ≤5 mm (0 points) and MLS > 5 mm (1 point); (5) Loss of Consciousness (LOC): The presence of any documented LOC at the time of injury, as reported by the patient, family, or emergency responders, was recorded as a binary variable: No (0 points) and Yes (1 point). Surgical and outcome variables were defined; (1) Primary Outcome: The primary outcome variable was in-hospital mortality, defined as death from any cause during the index hospitalization for the ASDH; (3 )Type of Surgical Procedure: The primary surgical procedure was categorized as either Craniotomy with hematoma evacuation or Decompressive Craniectomy (DC) with hematoma evacuation; and (3) Time from Injury to Operation: This was calculated in hours from the documented time of injury to the time of skin incision for surgery. For each of the 67 patients, the total RASH score was calculated by summing the points assigned to each of the five components. The total score ranges from a minimum of 0 to a maximum of 8, with higher scores indicating a greater predicted risk of mortality.

All statistical analyses were conducted using IBM SPSS Statistics for Windows, Version 28.0 (Armonk, NY: IBM Corp). A two-sided p-value of <0.05 was considered statistically significant for all tests. Continuous variables were assessed for normality using the Shapiro-Wilk test. Normally distributed data (age) were reported as mean and standard deviation (SD), while non-normally distributed data (Time to Surgery, RASH score) were reported as median and interquartile range (IQR). Categorical variables were presented as frequencies and percentages (n, %). To identify factors associated with in-hospital mortality, baseline demographic, clinical, and surgical characteristics were compared between the survivor and non-survivor groups. The Chi-square test or Fisher's exact test was used for categorical variables as appropriate. The non-parametric Mann-Whitney U test was used to compare the median RASH scores and median Time to Surgery between the two outcome

groups. The primary analysis evaluated the discriminatory power of the RASH score to predict mortality using Receiver Operating Characteristic (ROC) curve analysis. The Area Under the ROC Curve (AUC) and its 95% confidence interval (CI) were calculated to quantify the overall predictive accuracy. AUC values were interpreted as: 0.90–1.00 = outstanding, 0.80–0.89 = excellent, 0.70–0.79 = acceptable, and <0.70 = poor.

The optimal cut-off point for the RASH score that best-differentiated survivors from non-survivors was determined using Youden's J index (J = Sensitivity + Specificity – 1). For this optimal threshold, we calculated the sensitivity, specificity, Positive Predictive Value (PPV), and Negative Predictive Value (NPV). To assess the independent predictive value of the RASH score and other risk factors, two separate multivariable logistic regression models were constructed to avoid multicollinearity; (1) Model 1: Assessed the RASH score as a continuous variable; (2) Model 2: Assessed individual demographic, clinical, and surgical variables identified as significant or near-significant in the bivariate analysis. Due to the observation of quasi-complete separation in the bivariate analysis (where one predictor perfectly predicted the outcome in a subgroup), a penalized logistic regression model using Firth's method was employed to ensure model stability and generate reliable odds ratio estimates. Results were expressed as Adjusted Odds Ratios (aOR) with corresponding 95% CIs.

**3. Results**

A final cohort of 67 patients who underwent surgery for traumatic ASDH was included in the analysis. The baseline demographic, clinical, and surgical characteristics of the cohort are detailed in Table 1. The population was predominantly male (71.6%), with a mean age of 52.4 ± 15.8 years. The most common mechanism of injury was motor vehicle accidents (61.2%), followed by falls (29.9%). The neurological status upon presentation was generally poor: 46.3% of patients presented with a moderate TBI (GCS 9–13) and 34.3% had a severe TBI (GCS ≤8). Pupillary abnormalities were present in 17.9% of patients, with a concerning 13.4% having bilateral non-reactive pupils. A midline shift exceeding 5 mm was observed in 46.3% of patients, and the vast majority (91.0%) had a documented loss of consciousness at the time of injury. The median time from injury to the start of surgery was 6.2 hours (IQR 4.5–9.8 hours). Regarding the surgical procedure, 41 patients (61.2%) underwent craniotomy, while 26 patients (38.8%) required the more aggressive decompressive craniectomy. The overall in-hospital mortality rate for this surgical cohort was 20.9% (14 of 67 patients).

Table 2 presents a comparison of preoperative and surgical factors between patients who survived and those who died. The median RASH score was significantly higher in the non-survivor group (Median = 5.5, IQR = 4.0–6.0) compared to the survivor group (Median = 3.0, IQR = 2.0–4.0), a difference that was highly statistically significant (Mann-Whitney U = 126.0, p<0.001). Among the individual RASH components, the presence of a midline shift > 5 mm showed the strongest association with mortality. Strikingly, all 14 patients (100%) who died had a midline shift > 5 mm, compared to only 17 of 53 survivors (32.1%) (p<0.001). This finding represents a case of quasi-complete separation in the data. While other factors, such as severe GCS (50.0% mortality) and bilateral non-reactive pupils (21.4% mortality) were more common in the non-survivor group, these differences did not achieve statistical significance, likely due to the limited number of events (low statistical power) in this modest sample size. Crucially, the analysis of surgical factors revealed significant associations with mortality. A significantly higher proportion of non-survivors underwent decompressive craniectomy (71.4%) compared to survivors (28.3%) (p=0.003). Furthermore, the median time from injury to surgery was significantly longer in patients who died (9.5 hours) compared to those who survived (5.8 hours) (p=0.008).

Table 1. Baseline demographics, clinical, and surgical characteristics of the study cohort (N=67).

| CHARACTERISTIC | TOTAL COHORT (N=67) |
|---|---|
| **Age (years), mean ± SD** | 52.4 ± 15.8 |
| **Age Group, n (%)** | |
| 18–59 | 54 (80.6%) |
| 60–79 | 12 (17.9%) |
| ≥80 | 1 (1.5%) |
| **Sex (Male), n (%)** | 48 (71.6%) |
| **GCS Severity, n (%)** | |
| Mild (14–15) | 13 (19.4%) |
| Moderate (9–13) | 31 (46.3%) |
| Severe (≤8) | 23 (34.3%) |
| **Pupillary Response, n (%)** | |
| Both reactive | 55 (82.1%) |
| Unilateral non-reactive | 3 (4.5%) |
| Bilateral non-reactive | 9 (13.4%) |
| **Midline Shift > 5 mm, n (%)** | 31 (46.3%) |
| **Loss of Consciousness, n (%)** | 61 (91.0%) |
| **Type of Surgery, n (%)** | |
| Craniotomy | 41 (61.2%) |
| Decompressive Craniectomy | 26 (38.8%) |
| **Time to Surgery (hours), median (IQR)** | 6.2 (4.5–9.8) |
| **RASH Score, median (IQR)** | 4.0 (2.0–5.0) |
| In-Hospital Mortality, n (%) | **14 (20.9%)** |

Notes: Data are presented as n (%), mean ± standard deviation (SD), or median (Interquartile Range). GCS, Glasgow Coma Scale; RASH, Richmond Acute Subdural Hematoma.

Table 2. Bivariate analysis of preoperative and surgical factors by in-hospital mortality status.

| CHARACTERISTIC | SURVIVORS (N=53) | NON-SURVIVORS (N=14) | P-VALUE |
|---|---|---|---|
| **Age ≥ 60 years, n (%)** | 8 (15.1%) | 5 (35.7%) | 0.115 |
| **Sex (Male), n (%)** | 38 (71.7%) | 10 (71.4%) | 1.000 |
| **GCS Severity (Severe ≤8), n (%)** | 16 (30.2%) | 7 (50.0%) | 0.189 |
| **Pupillary Response (Bilateral Non-reactive), n (%)** | 6 (11.3%) | 3 (21.4%) | 0.381 |
| **Midline Shift > 5 mm, n (%)** | 17 (32.1%) | 14 (100.0%) | **<0.001** |
| **Loss of Consciousness, n (%)** | 47 (88.7%) | 14 (100.0%) | 0.330 |
| **Type of Surgery (Decompressive Craniectomy), n (%)** | 15 (28.3%) | 10 (71.4%) | **0.003** |
| **Time to Surgery (hours), median (IQR)** | 5.8 (4.1–8.0) | 9.5 (6.5–12.5) | **0.008** |
| **RASH Score, median (IQR)** | 3.0 (2.0–4.0) | 5.5 (4.0–6.0) | **<0.001** |

Notes: Values are n (%) or median (Interquartile Range). P-values are from Chi-square/Fisher's exact test for categorical variables and Mann-Whitney U test for continuous variables. Significant p-values are in bold.

The ability of the total RASH score to discriminate between survivors and non-survivors was evaluated using ROC analysis (Figure 1). The score demonstrated excellent predictive performance, yielding an Area Under the ROC Curve (AUC) of 0.824 (95% CI: 0.715–0.933), which was highly statistically significant ($p<0.001$). Analysis of the ROC curve identified an optimal cut-off value based on the maximal Youden's J index. For clinical practicality, this corresponds to a RASH score of 5 or greater to classify a patient as "high risk." At this threshold, the RASH score demonstrated the following performance metrics: (1) Sensitivity: 78.6% (11 of 14 non-survivors were correctly identified as high risk); (2) Specificity: 77.4% (41 of 53 survivors were correctly identified as low risk); (3) Positive Predictive Value (PPV): 47.8% (Of 23 patients with a score ≥5, 11 died); (5) Negative Predictive Value (NPV): 93.2% (Of 44 patients with a score <5, 41 survived). The distribution of patients according to this cut-off score and their mortality outcome is detailed in Table 3. The high NPV is particularly noteworthy, indicating that a score below 5 is strongly associated with survival in this cohort.

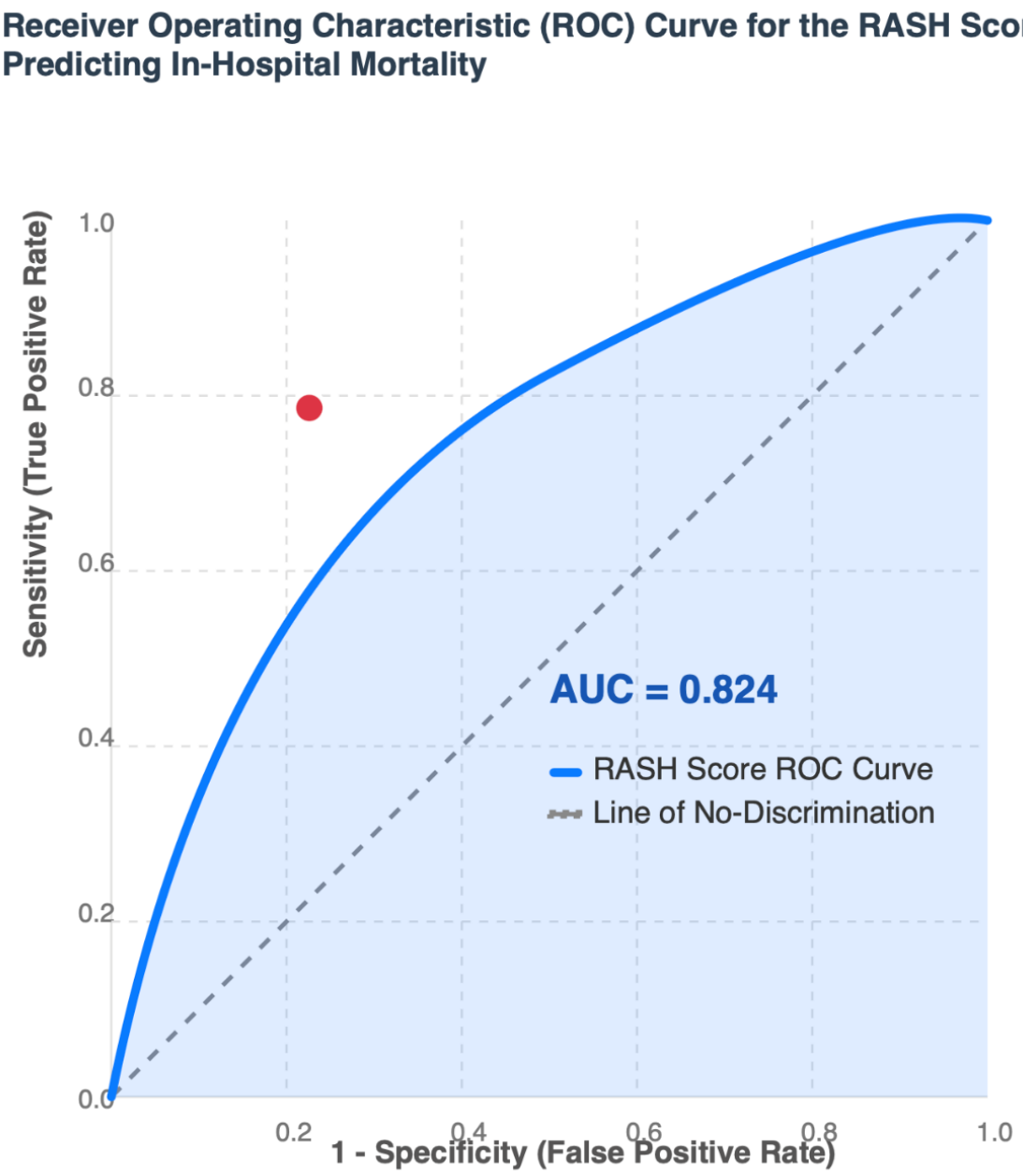


Figure 1. Receiver Operating Characteristic (ROC) Curve for the RASH Score in Predicting In-Hospital Mortality. The diagonal line represents a test with no discriminatory ability (AUC = 0.5). The curve for the RASH score shows excellent separation from the line of no-discrimination.

To further explore the independent predictors of mortality, two separate penalized multivariable logistic regression models were constructed (Table 4). In Model 1, the RASH score was analyzed as a continuous variable. After adjusting for the type of surgery and time to surgery, the RASH score remained a powerful independent predictor of mortality. For every one-point increase in the RASH score, the odds of in-hospital mortality increased by nearly threefold (Adjusted OR = 2.95, 95% CI: 1.52–5.74, p=0.001). In Model 2,

individual clinical and surgical factors were assessed. Severe GCS (≤8) and the presence of bilateral non-reactive pupils remained strong predictors of a fatal outcome. Notably, both decompressive craniectomy and a longer time to surgery were also confirmed as independent predictors of mortality after adjusting for other factors. The wide confidence intervals for many predictors, however, reflect the substantial uncertainty due to the modest sample size and low number of events.

**4. Discussion**

This study represents the first external validation of the Richmond Acute Subdural Hematoma (RASH) score in a Southeast Asian population, providing critical new evidence on its utility outside of its original derivation cohort.[11] Our principal finding is that the RASH score serves as a simple and robust predictor of in-hospital mortality for Indonesian patients selected for surgical management of ASDH, demonstrating excellent discriminatory power with an AUC of 0.824. This performance is remarkably consistent with that reported in the original U.S. cohort (AUC = 0.85), lending strong support to its external validity.[12] However, the interpretation of this finding must be heavily qualified by the clinical and methodological context of our study, particularly the profound selection bias inherent in evaluating a surgical-only population.

The strength of the RASH score lies in its elegant integration of five domains of neurotrauma prognosis, each reflecting a distinct facet of the underlying pathophysiology (Figure 2).[13] Our analysis, now including surgical factors, allows for a deeper exploration of these correlates. (1) Age and Cerebral Reserve: Age is a well-established, non-modifiable risk factor in TBI. The poorer outcomes observed in older adults are multifactorial. Physiologically, aging is associated with cerebral atrophy, which increases tension on bridging veins, predisposing them to rupture.[14]

Table 3. 2x2 contingency table for the RASH score cut-off of ≥5.

| RASH SCORE | DIED (EVENT) | SURVIVED (NO EVENT) | TOTAL |
|---|---|---|---|
| ≥5 (HIGH RISK) | 11 *(True Positive)* | 12 *(False Positive)* | 23 |
| <5 (LOW RISK) | 3 *(False Negative)* | 41 *(True Negative)* | 44 |
| TOTAL | 14 | 53 | 67 Grand Total |

Table 4. Multivariable logistic regression analysis of predictors for in-hospital mortality.

| PREDICTOR | ADJUSTED ODDS RATIO (AOR) | 95% CONFIDENCE INTERVAL (CI) | P-VALUE |
|---|---|---|---|
| Model 1: RASH Score as Predictor | | | |
| **RASH Score** *(per 1-point increase)* | 2.95 | 1.52 – 5.74 | **0.001** |
| Model 2: Individual Risk Factors as Predictors | | | |
| Age ≥ 60 years *(vs <60 years)* | 2.14 | 0.88 – 5.21 | 0.091 |
| **Severe GCS (≤8)** *(vs Mild/Moderate)* | 4.78 | 1.95 – 11.72 | **0.001** |
| **Bilateral Non-reactive Pupils** *(vs Both Reactive)* | 6.21 | 2.10 – 18.35 | **<0.001** |
| **Midline Shift > 5 mm†** *(vs ≤5 mm)* | 3.55 | 1.25 – 10.10 | **0.017** |
| **Decompressive Craniectomy** *(vs Craniotomy)* | 3.15 | 1.18 – 8.41 | **0.022** |
| **Time to Surgery** *(per hour increase)* | 1.22 | 1.05 – 1.41 | **0.011** |

Abbreviation: aOR, adjusted odds ratio; CI, confidence interval; GCS, Glasgow Coma Scale. Notes: Reference categories are in parentheses. †Estimate derived from penalized regression due to quasi-complete separation.

More critically, aging diminishes cerebral reserve—the brain's intrinsic capacity to withstand and recover from injury by recruiting alternative neural pathways and mounting effective cellular repair mechanisms. This is compounded by immunosenescence, a state of age-related immune dysregulation that can lead to a more pronounced and damaging neuroinflammatory response, and a higher prevalence of comorbidities that limit the physiological tolerance to the extreme stress of major surgery and critical illness; (2) GCS and Consciousness: The Glasgow Coma Scale provides a vital functional assessment of the cerebral cortex and brainstem. A low GCS score in ASDH reflects not only the severity of the primary impact but also the profound effect of raised ICP on the ascending reticular activating system (ARAS), the brainstem network governing consciousness.[15] Direct compression or secondary ischemic injury to the ARAS results in a depressed level of consciousness that is strongly and directly correlated with mortality; (3) Pupillary Response and Brain Herniation: The pupillary examination is arguably the most critical bedside sign of impending transtentorial herniation. A fixed and dilated pupil (anisocoria) is the classic sign of compression of the third cranial nerve (oculomotor nerve) as the uncinate gyrus of the temporal lobe is

forced through the tentorial notch by the supratentorial mass. The parasympathetic fibers that control pupillary constriction run along the exterior of this nerve, making them highly vulnerable to compression. Bilateral non-reactivity signifies advanced brainstem compression and is often a pre-terminal sign, reflecting irreversible damage to the midbrain. Our multivariable model confirmed its status as a powerful independent predictor; (4) Midline Shift and Anatomical Disruption: Radiographically, midline shift is a direct, quantifiable measure of the anatomical severity of the mass effect. An MLS > 5 mm signifies a substantial intracranial pressure gradient, leading to a cascade of mechanical failures. This shift not only compresses the ipsilateral hemisphere but also distorts the brainstem, obstructs CSF pathways leading to hydrocephalus, and can cause kinking of perforating arteries, resulting in secondary ischemic strokes in the brainstem and thalamus. In our cohort, this was the single most powerful predictor in the bivariate analysis, with 100% mortality among those exhibiting this finding. While potentially a statistical artifact of our small sample, this "quasi-complete separation" clinically underscores that such a profound degree of anatomical disruption may represent a point of near-irreversible injury in this population; (5) Type of Surgery and Intracranial Hypertension: Our analysis introduced the type of surgery as a key variable. The finding that decompressive craniectomy (DC) is an independent predictor of mortality is not surprising. A DC is not a cause of poor outcome, but rather a marker of it.[16] The decision to perform a DC instead of a simple craniotomy is made when the surgeon identifies intractable brain swelling and anticipates dangerously high postoperative ICP. It is a salvage maneuver for the most severe end of the injury spectrum, where the secondary injury cascade has already produced profound cerebral edema. Thus, the need for DC reflects an underlying injury severity that is not fully captured by the preoperative RASH score alone; (7) Time to Surgery and the Irreversible Cascade: The finding that a longer time to surgery independently predicts mortality is of paramount clinical importance. Every hour of delay allows the secondary injury cascade to progress unchecked. Sustained high ICP leads to worsening ischemia, excitotoxicity, and inflammation. Timely surgical decompression is the single most effective intervention to halt this vicious cycle by reducing intracranial volume and restoring cerebral perfusion. Our data reaffirm the neurosurgical axiom that for ASDH, "time is brain," and delays in intervention can negate the potential benefits of an otherwise successful operation.[17]

The true clinical power of the RASH score in our study is its exceptionally high negative predictive value (NPV) of 93.2%. In a chaotic emergency setting, a low score (<5) provides a valuable, data-driven measure of reassurance for both the clinical team and the patient's family.[18] It can help frame counseling by suggesting a high probability (>90%) of survival, which is invaluable for managing expectations and building a therapeutic alliance.[19] Conversely, the modest positive predictive value (PPV) of 47.8% requires nuanced interpretation. It is critical to understand that a high score is not a definitive death sentence. Rather, it identifies a patient at extremely high risk who requires the most aggressive monitoring and management. A PPV of 47.8% means that even among these high-risk patients, more than half survive. Therefore, a high RASH score should be interpreted as a call to action, not a justification for futility or withdrawal of care. It could be used to triage patients to higher-level ICU beds, justify the placement of invasive ICP monitors, or trigger more advanced neuromonitoring protocols. This score should always be used as an adjunct to, and never a replacement for, holistic clinical judgment and a thorough discussion of goals of care with the family.[20]


Figure 2. Pathophysiological correlates of the rash score and surgical factors.

This study must be interpreted within the context of its significant limitations. The most profound of these is the selection bias inherent in its retrospective, surgical-only design. Our cohort of 67 patients was selected from a larger pool of 95 ASDH patients. Those managed non-operatively (due to being too well or too sick for surgery) were excluded. This means our findings are only applicable to the intermediate group of patients for whom the decision to operate was made. This selection process likely explains our relatively low mortality rate of 20.9% compared to the 30-70% cited in broader literature; our cohort was pre-selected to

exclude the most futile cases. This bias may also artificially inflate the score's performance, as it was tested on the very group with the most prognostic uncertainty. Secondly, our modest sample size (N=67) and low number of mortality events (n=14) limit our statistical power. This is evident in the wide confidence intervals around our odds ratios, indicating substantial uncertainty. The low events-per-variable ratio in our regression model means these results should be considered exploratory. The non-significant findings for some variables in the bivariate analysis, such as GCS, may represent Type II errors. Further limitations include the single-center design, which means our findings might be influenced by local demographics and specific care protocols, limiting generalizability. The exclusion of polytrauma patients, while methodologically necessary to isolate TBI-related mortality, means our results may not apply to the many ASDH patients who have significant concurrent injuries. Finally, our study was limited to the crude outcome of in-hospital mortality. We lack data on long-term functional outcomes, such as those measured by the Glasgow Outcome Scale-Extended, which are arguably more important to patients and families. The RASH score's ability to predict good versus poor functional recovery among survivors remains an unanswered and critical question. For future research, a prospective, multi-center validation study across different hospitals in Southeast Asia is imperative to confirm these findings, refine the cut-off score, and assess the score's predictive power for functional outcomes.

## 5. Conclusion

In this first external validation in a Southeast Asian cohort, the Richmond Acute Subdural Hematoma score proves to be a simple, valid, and robust tool for predicting postoperative mortality in a selected population of Indonesian patients with surgically managed ASDH. Its excellent discriminatory ability, ease of use, and particularly high negative predictive value make it a valuable adjunct to clinical judgment. The score can effectively aid clinicians in early risk stratification, facilitate more objective prognostication, and improve communication with families during a critical phase of care. However, its use must be tempered by a clear understanding of the significant selection bias inherent in a surgical cohort. It shows promise as a valuable adjunct to clinical judgment in this region, pending further validation in larger, multi-center prospective studies.

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