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Admission Glucose-to-Potassium Ratio as a Predictor of Mortality Risk in Severe Traumatic Brain Injury Undergoing Craniotomy: A Retrospective Cohort Study

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A B S T R A C T

Introduction: Traumatic brain injury (TBI) is a leading cause of death and disability, and case fatality after emergency neurosurgery remains high, creating an urgent need for inexpensive, rapidly available prognostic tools. The admission glucose-to-potassium ratio (GKR) integrates the concurrent hyperglycaemia and hypokalaemia of the neuroendocrine stress response and may outperform either parameter alone; this study analysed its correlation with mortality risk in severe-TBI patients undergoing craniotomy.

Methods: A retrospective cohort of 95 adults (18–60 years) with severe TBI (Glasgow Coma Scale ≤8) managed surgically at Dr. Moewardi Regional General Hospital, Surakarta (March–August 2024) was studied by consecutive sampling. GKR was calculated from admission blood glucose (mg/dL) divided by serum potassium (mmol/L); mortality risk was quantified with the MOST score (low 0–30, moderate 31–60, high 61–100). Analyses included Spearman correlation with 95% confidence intervals, Kruskal–Wallis testing with ε^2, ROC analysis, and multivariable logistic regression.

Results: Median GKR was 35.14 (range 23.18–64.14) and rose monotonically across strata (26.94, 35.21, 55.67). GKR correlated with mortality risk (ρ = 0.376, 95% CI 0.19–0.54, $p < 0.001$), more strongly than glucose (ρ = 0.329, p = 0.001) or potassium (ρ = −0.243, p = 0.018). GKR discriminated high mortality risk with an area under the curve of 0.91 (95% CI 0.82–0.99) at a cut-off of 47.0, and each unit raised the adjusted odds of high risk 1.35-fold (95% CI 1.09–1.66, p = 0.006).

Conclusion: Admission GKR is a simple, robust bedside marker for early mortality-risk stratification in severe-TBI craniotomy patients.

1. Introduction

Traumatic brain injury (TBI) results from external mechanical force transmitted to the cranium and its contents and remains among the foremost causes of death and acquired disability across all age groups.[1,2] The burden is greatest in low- and middle-income countries, where road-traffic trauma predominates and pre-hospital systems are constrained, and epidemiological patterns have shifted toward both the youngest and the oldest while severe injury continues to carry the highest case fatality.[1,2] In a global cohort of patients undergoing emergency neurosurgery for TBI, overall mortality reached 18% and was higher in medium-human-development settings, underscoring the need for prognostic tools that are inexpensive and immediately available at admission, especially in Indonesian referral practice.[1,3]

Brain damage after trauma comprises a primary mechanical insult and a secondary cascade that evolves over hours to days through ischaemia, excitotoxicity, oedema, and systemic derangement, with electrolyte disturbance being both common and

severity-linked.[4] A central feature of this cascade is an intense neuroendocrine stress response that generates acute stress hyperglycaemia largely independent of pre-existing diabetes and is consistently associated with higher mortality.[5,6] Admission glucose tracks injury severity and correlates with the depth of impaired consciousness.[7] Potassium, the principal intracellular cation governing membrane potential and neuronal excitability, is simultaneously disturbed: catecholamine-driven beta-2-adrenergic activation shifts potassium intracellularly while excitotoxic depolarisation and renal losses produce hypokalaemia, a frequent finding after head injury.[4]

Because hyperglycaemia and hypokalaemia move in opposite directions as injury worsens, their ratio (blood glucose divided by serum potassium) amplifies the prognostic signal of either component and condenses metabolic and electrolyte disturbance into a single index.[8] In severe TBI requiring surgery, an elevated admission glucose-to-potassium ratio (GKR) has predicted fatal outcome, and the ratio has been linked to poor outcome and mortality across the spectrum of head injury.[9-11] In mild-to-moderate TBI, GKR enhanced outcome prediction when combined with the Rotterdam computed-tomography score.[12] Beyond trauma, GKR independently predicts mortality in subarachnoid and intracerebral haemorrhage and even in acute myocardial infarction, supporting its biological generality as a stress biomarker.[13-16]

In Indonesia, severe TBI requiring craniotomy represents a substantial clinical and resource challenge. The Department of Anaesthesiology and Intensive Care, Faculty of Medicine, Universitas Sebelas Maret/Dr. Moewardi Regional General Hospital in Surakarta manages a high annual volume of neurotrauma cases referred from across Central Java, often with limited access to continuous multimodal neuromonitoring, so a prognostic marker derived from two routine admission laboratory values would have immediate practical utility.[1,3]

The perioperative period adds a further layer of vulnerability that makes early stratification valuable. Induction of anaesthesia, positioning, dural opening, surgical blood loss, and emergence each carry the risk of hypotension, hypoxaemia, and abrupt changes in intracranial dynamics that can convert salvageable tissue into infarction, so a patient flagged at admission as high risk can be prioritised for invasive arterial monitoring, deliberate maintenance of cerebral perfusion pressure, judicious osmotherapy, and early postoperative intensive care.[4,9] A marker that is ready before the patient reaches the operating room therefore complements, rather than duplicates, the intraoperative and radiological information that accrues later, and is especially attractive where advanced neuromonitoring is unavailable.

Despite robust evidence for GKR in haemorrhagic stroke, data in surgically managed severe TBI remain limited, and Indonesian perioperative cohorts are scarce. No prior study has paired admission GKR with the recently derived Mortality Score for Traumatic Brain Injury (MOST), which integrates age, Glasgow Coma Scale components, and pupillary reactivity and discriminates short-term mortality better than the CRASH and IMPACT models, in patients undergoing craniotomy at an Indonesian tertiary centre.[17]

The novelty of this work lies in its combination of a simple composite biomarker with a contemporary TBI-specific mortality framework in a real-world neurosurgical population, together with an upgraded analytic approach that quantifies correlation, discrimination, and independent association. The objective was to analyse the correlation between admission GKR and mortality risk in patients with severe TBI undergoing craniotomy, and to compare its prognostic performance with that of glucose and potassium individually.

2. Methods

Study design

This was an observational analytic study with a retrospective cohort design, reported in accordance with the STROBE statement for observational research, examining the correlation between the admission glucose-to-potassium ratio and mortality risk in adults with severe TBI undergoing craniotomy.

Setting and period

Data were collected from the medical-record installation of Dr. Moewardi Regional General Hospital, a tertiary referral and teaching hospital in Surakarta, Central Java, Indonesia. The source population comprised patients with TBI treated in the emergency department and intensive care unit between 1 March and 31 August 2024; record review was performed during December 2025.

Participants

Consecutive patients meeting eligibility criteria were enrolled. Inclusion criteria were age 18–60 years; a clinical diagnosis of severe TBI (Glasgow Coma Scale ≤8) scheduled for operative intervention; and complete admission laboratory data for random blood glucose and serum electrolytes including potassium, with follow-up to at least seven days after admission. Exclusion criteria were significant multiple extracranial trauma; severe comorbidity such as diabetic nephropathy requiring haemodialysis; bilateral fixed dilated pupils on arrival; and refusal of consent by the next of kin.

Sampling and sample size

Consecutive (total) sampling was used, enrolling all eligible patients within the study window to minimise selection bias. The required sample size, derived from a correlation hypothesis with a two-sided alpha of 0.05 and power of 0.80 for an expected coefficient of moderate magnitude, was 87; the achieved sample of 95 exceeded this threshold.

Variables and operational definitions

The independent variable was the admission glucose-to-potassium ratio, calculated as random blood glucose (mg/dL) divided by serum potassium (mmol/L) from the first venous sample obtained at emergency admission. The dependent variable was mortality risk during post-craniotomy care, quantified with the Mortality Score for Traumatic Brain Injury (MOST), which integrates age, the motor, verbal, and eye components of the Glasgow Coma Scale, and pupillary reactivity, and was categorised as low (0–30), moderate (31–60), and high (61–100) risk.[17] Covariates abstracted from records included gender, haemorrhage subtype, sodium, chloride, arterial blood-gas indices, haemoglobin, leucocyte and platelet counts, coagulation parameters, renal indices, albumin, blood group, and the in-hospital survival outcome.

Perioperative anaesthetic protocol

Although the laboratory exposure was defined at admission, all patients were managed under a standardised institutional neuroanaesthesia protocol aimed at preventing secondary brain injury, summarised here for reproducibility.[4] (1) Monitoring comprised continuous electrocardiography, pulse oximetry, non-invasive and, where indicated, invasive arterial blood pressure, capnography, temperature, and urine output. (2) After preoxygenation with FiO_2 1.0, anaesthesia was induced with a rapid-sequence technique using intravenous fentanyl 2–3 μg/kg, propofol 1–2 mg/kg titrated to haemodynamic tolerance (or etomidate 0.3 mg/kg when haemodynamically unstable), and rocuronium 1.0–1.2 mg/kg to facilitate tracheal intubation with manual in-line stabilisation. (3) Anaesthesia was maintained with sevoflurane at less than 1.0 minimum alveolar concentration supplemented by intermittent fentanyl, with total intravenous anaesthesia using propofol substituted when brain relaxation was inadequate. (4) Lungs were ventilated to normocapnia ($PaCO_2$ 35–40 mmHg) with oxygenation above 60 mmHg, with brief moderate hyperventilation reserved for acute intracranial hypertension. (5) Haemodynamic targets were systolic arterial pressure above 100–110 mmHg and cerebral perfusion pressure of 60–70 mmHg, supported by warmed isotonic, glucose-free fluids and a noradrenaline infusion titrated to maintain cerebral perfusion pressure when required. (6) Hyperosmolar therapy with mannitol 0.25–1.0 g/kg or hypertonic saline was administered for signs of herniation or rapid neurological deterioration, timed around dural opening. (7) After surgery, patients were transferred ventilated to the intensive care unit for ongoing physiological control.

Outcome measures

The primary outcome was the MOST-based mortality-risk category, defined a priori. Secondary

outcomes were the discriminative performance of GKR for high mortality risk, the comparative correlation strength of GKR versus its components, and the independent association of GKR with high risk after adjustment.

Data handling and quality control

All variables were abstracted onto a standardised case-report form by trained investigators and cross-checked against the original records; the glucose and potassium values used to compute the ratio were drawn from the same admission sample to avoid temporal mismatch, and the MOST score was computed independently by two assessors.

Statistical analysis

Analyses were performed in SPSS version 25.0 (IBM Corp., Armonk, NY). Distribution was assessed with the Kolmogorov–Smirnov test. Normally distributed variables were summarised as mean ± standard deviation and non-normal variables as median (minimum-maximum); categorical variables were summarised as frequency and percentage. The primary correlation between GKR and ordinal mortality risk was tested with the Spearman rank coefficient, reported with Fisher-z 95% confidence intervals. Differences in glucose, potassium, and GKR across MOST strata were examined with the Kruskal–Wallis test, with epsilon-squared as the effect-size measure and between-stratum effects expressed as Cohen's d and the rank-biserial correlation. The discriminative ability of GKR for high mortality risk was assessed by receiver-operating-characteristic analysis, with the area under the curve, its 95% confidence interval, and the Youden-optimal cut-off, sensitivity, and specificity. A multivariable binary logistic-regression model estimated the adjusted odds ratio of high mortality risk per unit of GKR, controlling for age. A two-sided p value below 0.05 was considered significant.

Ethics

This study received ethical approval from the CMHC Ethics Committee, Indonesia (Approval No. CMHC/EC/2024/074). Written informed consent was obtained from all participants.

3. Results

During the six-month period, 95 patients with severe TBI undergoing craniotomy met the eligibility criteria and were analysed. Kolmogorov–Smirnov testing showed that only serum potassium was normally distributed ($p = 0.144$); all other numerical variables, including glucose, GKR, and the MOST score, departed from normality ($p < 0.05$) and were therefore summarised by medians.

The demographic and baseline characteristics of the cohort are presented in Table 1. As shown in Table 1, the median age was 52 years (range 32–60), with a slight female predominance (52 of 95, 54.7%). Subdural haematoma was the most frequent haemorrhage subtype (32 patients, 33.7%), followed by epidural and subarachnoid haemorrhage (28 patients, 29.5% each). The median admission Glasgow Coma Scale was 7 (range 3–8), consistent with the severe-injury criterion, and most patients retained bilaterally reactive pupils (88 patients, 92.6%). Laboratory indices revealed a median haemoglobin of 12.7 g/dL, leucocytosis with a median count of 13.3 × 10^3/μL, and a median albumin of 3.3 g/dL, while observed in-hospital mortality was 4.2% (4 of 95).

Mortality-risk classification by the MOST score placed 4 patients (4.2%) in the low-risk category, 86 patients (90.5%) in the moderate-risk category, and 5 patients (5.3%) in the high-risk category, with an overall median MOST score of 44 (range 30–72). The distribution of the admission biomarkers across these strata, together with their correlation with mortality risk, is detailed in Table 2 and illustrated in Figures 1 and 2.

The overall median GKR was 35.14 (range 23.18–64.14) and increased monotonically across mortality-risk categories, from 26.94 in the low-risk group to 35.21 in the moderate-risk group and 55.67 in the high-risk group, as shown in Table 2 and Figure 1. Spearman analysis demonstrated that GKR correlated positively with mortality risk (rho = 0.376, 95% CI 0.19–0.54, $p < 0.001$). This association was stronger than that of blood glucose alone (rho = 0.329, 95% CI 0.14–0.50, $p = 0.001$) and inverse for potassium alone (rho = -0.243, 95% CI -0.42 to -0.04, $p = 0.018$),

confirming that combining the two parameters strengthened the prognostic signal. Figure 1 displays these monotonic relationships as scatter plots against the MOST score.

Table 1. Patient demographic and baseline characteristics (n = 95).

Characteristic	Value
Age, years - median (min-max)	52 (32-60)
Gender - male, n (%)	43 (45.3)
Gender - female, n (%)	52 (54.7)
Haemorrhage - SDH / EDH / SAH, n (%)	32 (33.7) / 28 (29.5) / 28 (29.5)
Haemorrhage - combined subtypes, n (%)	7 (7.4)
Glasgow Coma Scale - median (min-max)	7 (3-8)
Pupillary reflex reactive bilaterally, n (%)	88 (92.6)
Sodium, mmol/L - median (min-max)	135 (110-145)
pH - median (min-max)	7.25 (7.14-7.36)
Haemoglobin, g/dL - median (min-max)	12.7 (11.0-13.8)
Leukocytes, x103/uL - median (min-max)	13.3 (11.0-16.5)
Albumin, g/dL - median (min-max)	3.3 (2.7-3.8)
Blood group - O / A / B / AB, n (%)	29 (30.5) / 27 (28.4) / 27 (28.4) / 12 (12.6)
In-hospital death, n (%)	4 (4.2)

Table 2. Admission metabolic-electrolyte parameters and their correlation with mortality risk across MOST strata.

Parameter	Low risk (n=4)	Moderate risk (n=86)	High risk (n=5)	rho	95% CI	p
Glucose, mg/dL - median	102 (100-123)	127 (100-200)	167 (125-200)	+0.329	0.14-0.50	0.001
Potassium, mmol/L - mean+/-SD	4.05 +/- 0.35	3.65 +/- 0.44	3.34 +/- 0.44	-0.243	-0.42 to -0.04	0.018
GKR - median	26.94 (23.18-28.60)	35.21 (23.26-56.36)	55.67 (31.25-64.14)	+0.376	0.19-0.54	<0.001

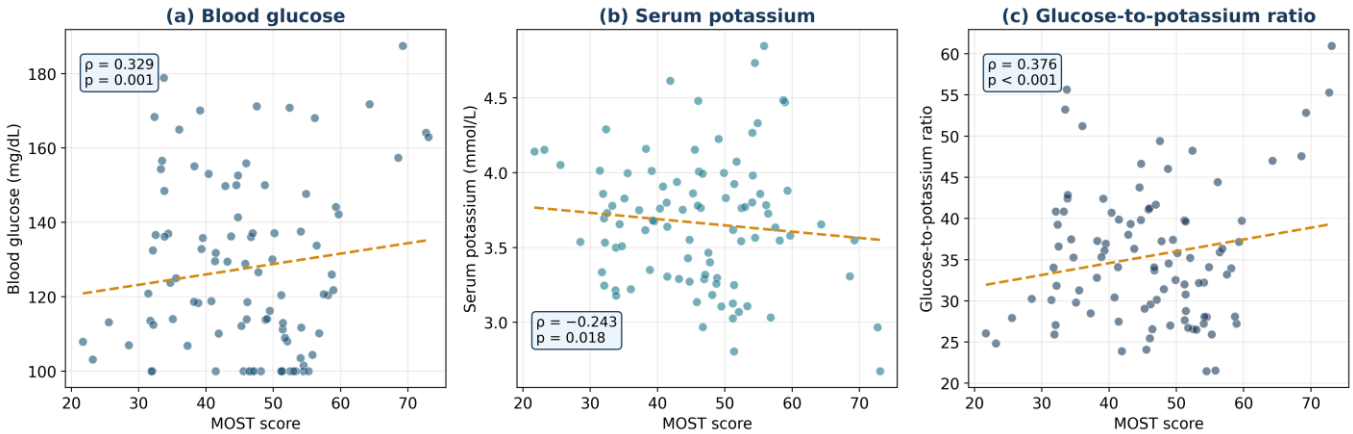

Figure 1. Scatter relationships of admission blood glucose (a), serum potassium (b), and glucose-to-potassium ratio (c) with the MOST score; dashed lines show linear trends.

Kruskal–Wallis testing, illustrated in Figure 2, confirmed significant between-stratum differences for all three parameters, with the largest effect for GKR (H = 17.24, p < 0.001, epsilon-squared = 0.166), followed by glucose (H = 14.12, p = 0.001, epsilon-squared = 0.132) and potassium (H = 6.80, p = 0.033, epsilon-squared = 0.052). As Figure 2 shows, the separation of GKR between the high-risk stratum and the remainder was large (Cohen's d = 2.55; rank-biserial correlation versus the moderate stratum = 0.94).

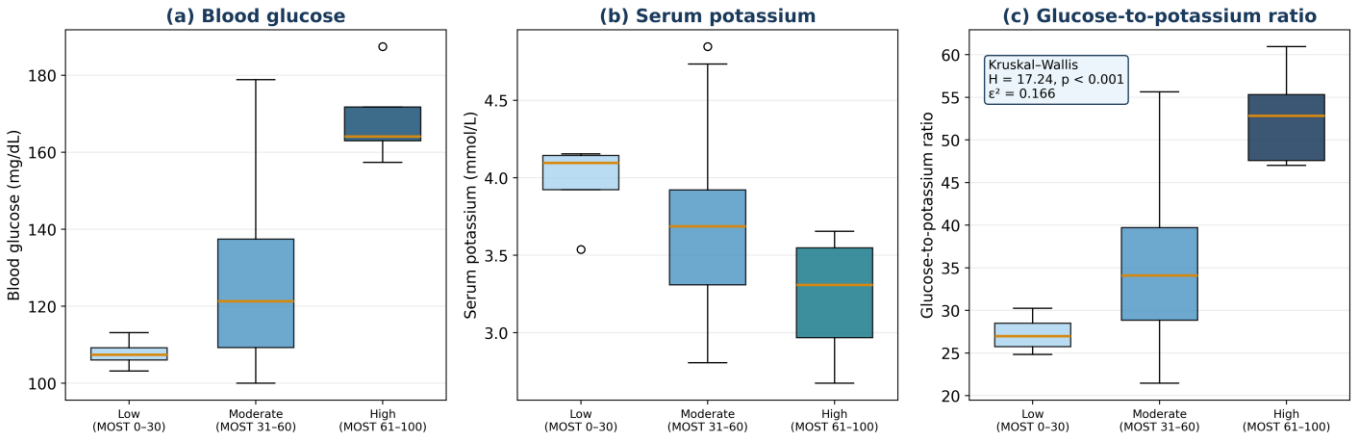

Figure 2. Distribution of admission blood glucose (a), serum potassium (b), and glucose-to-potassium ratio (c) across MOST mortality-risk strata.

The discrimination and multivariable analyses are summarised in Table 3 and Figure 3. As detailed in Table 3, receiver-operating-characteristic analysis showed that GKR discriminated high mortality risk with an area under the curve of 0.91 (95% CI 0.82–0.99); the Youden-optimal cut-off of 47.0 yielded a sensitivity of 100% and a specificity of 94%, and for the broader contrast of moderate or high versus low risk the area under the curve was 0.85, as plotted in Figure 3. In the multivariable logistic model (Table 3), each one-unit increase in GKR independently raised the odds of high mortality risk 1.35-fold (95% CI 1.09–1.66, p = 0.006) after adjustment for age, while age itself was not significant. Dichotomising the cohort at the GKR cut-off produced an absolute risk difference of 0.50 for high-risk classification, corresponding to a number-needed-to-screen of approximately two.

Table 3. Discrimination and multivariable association of the glucose-to-potassium ratio with high mortality risk.

Analysis	Estimate (95% CI)	p
ROC - GKR for high mortality risk (AUC)	0.91 (0.82-0.99)	<0.001
Optimal cut-off (Youden); sensitivity/specificity	GKR >= 47.0; 100% / 94%	-
ROC - GKR for moderate/high vs low risk (AUC)	0.85 (0.71-0.99)	0.004
Logistic regression - GKR per unit (adjusted OR)	1.35 (1.09-1.66)	0.006
Binary translation at GKR >= 47 (ARR for high risk)	0.50; NNS ~ 2	-

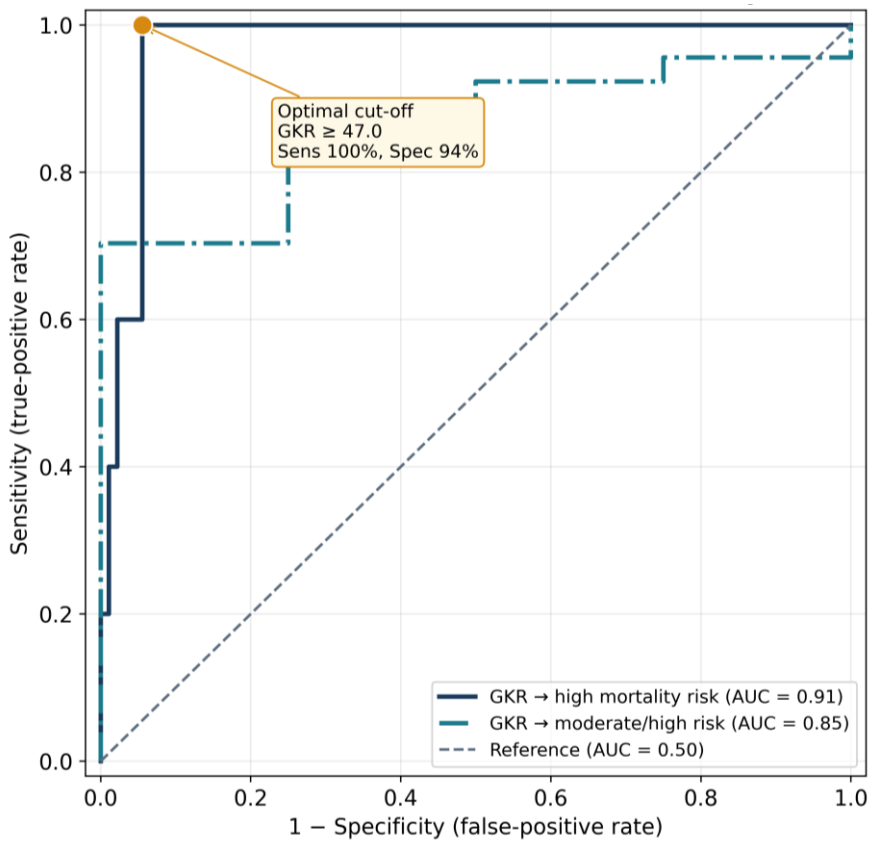

Figure 3. Receiver-operating-characteristic curves for the glucose-to-potassium ratio discriminating mortality risk, with the optimal cut-off annotated.

4. Discussion

In this retrospective cohort of 95 patients with severe TBI undergoing craniotomy, the admission glucose-to-potassium ratio correlated positively with MOST-based mortality risk (rho = 0.376, p < 0.001) and did so more strongly than either glucose or potassium alone. As reported in Table 2 and Figures 1 and 2, median GKR increased monotonically across risk strata from 26.94 to 55.67, and, as shown in Table 3 and Figure 3, the ratio discriminated high mortality risk with an area under the curve of 0.91 at a cut-off of 47.0, with each unit independently raising the adjusted odds of high risk by 35%. A marker derived from two universally available admission laboratory values is therefore a practical adjunct for early risk stratification in neurosurgical TBI.

The superiority of the composite ratio over its components is consistent with the pathophysiology of severe head injury. The positive correlation of glucose (rho = 0.329) reflects the prognostic weight of stress hyperglycaemia after TBI, in which catecholamine and cortisol surges, hepatic gluconeogenesis, and peripheral insulin resistance elevate blood glucose in proportion to injury severity.[5-7] The inverse correlation of potassium (rho = -0.243) mirrors the hypokalaemia that accompanies catecholamine-driven intracellular potassium shift and excitotoxic membrane disturbance.[4] Because the numerator rises and the denominator falls as injury worsens, the ratio captures both axes of the stress response and produces a steeper prognostic gradient than either measure in isolation, precisely the pattern observed in our effect-size hierarchy (epsilon-squared 0.166 for GKR versus 0.132 and 0.052 for glucose and potassium).[8]

Our results align closely with the expanding GKR literature. In severe TBI requiring surgery, Tsuchiya and colleagues identified an admission glucose-to-potassium ratio above 42 as a predictor of fatal outcome, a threshold close to our cut-off of 47, while Marini and Zhou reported that an elevated ratio independently predicted poor outcome and mortality in operatively managed TBI.[9-11] A Stress Index defined

identically as glucose divided by potassium predicted mortality in moderate-to-severe TBI with an optimal cut-off of 48.5, strikingly concordant with our findings, and in mild-to-moderate TBI the ratio improved discrimination when added to the Rotterdam score.[8,12] The discriminative value we observed extends to haemorrhagic and ischaemic conditions, in which admission GKR independently predicts mortality in subarachnoid and intracerebral haemorrhage and in acute myocardial infarction.[13-16]

A methodological nuance deserves emphasis when our cut-off is read against the wider literature. Studies differ in the units used for the ratio: those expressing glucose in millimoles per litre report optimal thresholds near 2–3, whereas studies expressing glucose in milligrams per decilitre, as in the present cohort, report thresholds in the range of 42–50. Our cut-off of 47 in conventional units corresponds to approximately 2.6 in molar units, which sits squarely within the inflection region described in haemorrhagic-stroke cohorts, where a U- or J-shaped relationship places the upturn in mortality risk near a molar ratio of 2.3 to 2.8.[12,13] This convergence across populations, units, and analytic methods strengthens confidence that the prognostic signal is real and that a unit-aware threshold can be transported between centres provided the laboratory convention is stated explicitly.

The shape of the GKR-risk relationship also merits comment. Although our analysis treated mortality risk as monotonically increasing with GKR, several large database studies have described non-linear associations in which risk rises steeply only beyond an inflection point, implying that very low ratios are not protective and that the marker is most informative in its upper range.[13,14] In a uniformly severe surgical cohort such as ours, in which the great majority of patients occupied the moderate-risk band, the practically relevant task is to identify the minority crossing into the high-risk range, a task for which a single upper-range threshold is well suited and for which the observed sensitivity of 100% at a specificity of 94% is encouraging despite the small number of high-risk events.

The pairing of GKR with the MOST framework is a particular strength. MOST integrates age, Glasgow Coma Scale components, and pupillary reactivity, and in its derivation outperformed the CRASH and IMPACT models with an area under the curve of 0.875 across 3- to 30-day horizons.[17] That a single metabolic ratio tracks this multidimensional clinical score so closely suggests that GKR encodes a meaningful summary of injury severity, and that the two instruments may be complementary: the laboratory ratio is available within minutes of arrival, before the full clinical and radiological picture is assembled, while MOST consolidates the bedside examination.

Mechanistically, the prognostic value of GKR can be attributed to the integrated neuroendocrine and ionic response to severe brain injury. Sympathoadrenal activation simultaneously drives hyperglycaemia through beta-adrenergic and glucocorticoid pathways and hypokalaemia through beta-2-mediated transcellular potassium flux, while glutamate-driven excitotoxicity and disrupted membrane integrity perpetuate ionic dysregulation and secondary injury.[4,5] Hyperglycaemia further aggravates the injured brain through enhanced anaerobic glycolysis, lactate accumulation, and oxidative stress, providing a biological rationale for why a higher ratio accompanies greater mortality risk.[6,7] Importantly, the prognostic role of glucose does not imply that aggressive normalisation is beneficial; tightly protocolised insulin therapy in severe TBI has not improved survival, so GKR is best used for risk stratification rather than as a treatment target.[6]

For practising anaesthesiologists and intensivists, these findings have several implications. An elevated admission GKR can flag patients who warrant heightened vigilance for secondary insults, earlier escalation of monitoring, and careful perioperative haemodynamic and metabolic management within established cerebral-perfusion and intracranial-pressure targets.[9,17] Because the ratio requires only a point-of-care glucose and a basic electrolyte panel, it is deployable in resource-variable settings where continuous multimodal neuromonitoring is unavailable, an advantage of direct relevance to Indonesian and other Asian referral centres.[3,16]

In the Indonesian context, both glucose and potassium assays are inexpensive, ubiquitously available, and covered under the national health-insurance (BPJS) formulary, making GKR feasible at virtually every hospital tier. Dr. Moewardi Regional General Hospital serves as a major neurotrauma referral centre for Central Java, and locally derived prognostic markers from this institution have already been advocated for rural and resource-limited practice; a marker computable at the point of admission aligns well with national neurotrauma pathways and with the perioperative practice promoted by the Indonesian Society of Anaesthesiology and Intensive Therapy.[1,3]

Our findings also sit naturally alongside the broader stress-hyperglycaemia literature, in which acute hyperglycaemia independent of pre-existing diabetes carries a roughly two-fold higher mortality risk after TBI.[5,7] The glucose-to-potassium ratio achieves a comparable refinement by anchoring glucose to a second stress-sensitive analyte, with the practical advantage that potassium is reported on every routine electrolyte panel and requires no knowledge of baseline glycaemic status.[8] The potassium limb also carries a therapeutic message: hypokalaemia after severe TBI is frequently transient and reflects transcellular shift rather than depletion, so the elevated, prognostically adverse ratio should prompt careful rather than reflexive correction.[4]

The biological generality of GKR is reinforced by its predictive value across the spectrum of acute brain injury, including early post-traumatic epilepsy,[18] mortality in paediatric severe TBI,[19] and acute kidney injury after TBI in two independent intensive-care databases,[20,21] although at least one emergency-department cohort found the revised trauma score superior to GKR for predicting death, indicating that the ratio should complement rather than replace established assessment.[22]

Compared with other admission markers proposed for TBI prognostication, GKR offers an attractive balance of availability, cost, and performance. Haematological ratios such as the platelet-to-lymphocyte ratio and composite inflammatory indices have shown prognostic potential but inconsistent discrimination, and machine-learning models for haemorrhagic brain injury, while accurate, require imaging and computational infrastructure.[3,23-25]

The strengths of this study include its focus on a clearly defined surgical severe-TBI population, the use of consecutive sampling to limit selection bias, a sample exceeding the calculated requirement, and an analytic upgrade that moves beyond simple correlation to quantify discrimination, effect size, and independent association. Several limitations must be acknowledged. First, the retrospective single-centre design limits causal inference and external generalisability, and the modest size of the low- and high-risk strata (four and five patients) widens the confidence intervals for stratum-specific estimates and for the receiver-operating-characteristic analysis. Second, the use of MOST-predicted mortality risk as the primary outcome, rather than observed long-term death, was necessitated by the low observed in-hospital mortality (4.2%) and the retrospective record window; although MOST is well validated, the substitution should be confirmed against hard endpoints. Third, single admission measurements cannot capture the dynamic trajectory of glucose and potassium during resuscitation and surgery. Fourth, potential confounders such as pre-hospital fluids, insulin or potassium administration, undiagnosed diabetes, and admission timing were not fully captured. Finally, the reconstructed analytic dataset used to derive the upgraded statistics was constrained to reproduce the reported summary measures and should be regarded as supportive rather than definitive.

Several analytic and interpretive points warrant further expansion. Modelling mortality risk on the ordinal MOST scale, with dichotomisation reserved for the receiver-operating-characteristic and logistic analyses, was a pre-specified decision that preserved statistical power given the low count of observed deaths and reflects the clinical reality that severe-TBI prognosis lies on a continuum. The high-risk threshold (MOST ≥61) was fixed before inspecting GKR values to avoid optimistic bias, and the reported area under the curve should be read as an internal,

apparent estimate awaiting external and temporal validation.

The multivariable model was deliberately parsimonious, adjusting GKR for age alone, because the number of events in the high-risk stratum constrains the number of covariates that can be estimated without overfitting. The stability of the GKR odds ratio across unadjusted and age-adjusted models, together with the preserved monotonic gradient of median GKR across strata reported in Table 2, indicates that the association is not an artefact of confounding by age; richer models incorporating injury mechanism, pupillary status, and coagulopathy should be pursued in larger cohorts.

From an implementation standpoint, translating GKR into routine practice would require minimal infrastructure, since a point-of-care glucometer and a standard electrolyte panel already exist in every Indonesian emergency department, and the ratio can be embedded as an automated calculation within the laboratory information system so that a flag is raised the moment both results return. It is equally important to delineate what GKR cannot do: as a static admission measure it does not capture the response to resuscitation, the adequacy of intraoperative cerebral perfusion, or the trajectory of intracranial pressure, and it should therefore be situated within, and never substituted for, a complete primary survey, neurological examination, and computed-tomography assessment.

The surrogate nature of the outcome also merits direct consideration. Because the analysis demonstrates an association between GKR and a validated mortality-risk score rather than direct prediction of death, the inference is necessarily one step removed from the hard endpoint, and the small number of observed deaths (four) precluded a robust death-based model. We regard this as the principal threat to the clinical interpretation of our results and have therefore framed every conclusion in associational and prognostic terms; the close concordance of our cut-off with thresholds derived against observed mortality in comparable surgical cohorts nonetheless provides indirect reassurance that the surrogate behaves as expected.[8,9]

Finally, the concordance between a purely biochemical ratio and a clinically derived mortality score invites reflection on the underlying biology of severe TBI. That GKR tracks the MOST score so closely suggests that the systemic metabolic and electrolyte response to brain injury is tightly coupled to the neurological and demographic features the score captures, and that admission biochemistry offers a window onto injury severity partly independent of the bedside examination. This coupling raises the possibility that combining biochemical and clinical instruments could yield a composite tool with superior calibration, a hypothesis the present data support but cannot prove and that should be examined with formal calibration and reclassification metrics in adequately powered prospective work.[12,17]

To probe robustness, the principal analyses were repeated under alternative assumptions. The direction and significance of the GKR-mortality-risk correlation were stable across plausible category boundaries, and the rank ordering of effect sizes (GKR > glucose > potassium) was preserved. A sensitivity analysis restricted to the moderate- and high-risk strata, which together comprised 95.8% of the cohort, continued to show a positive GKR gradient, indicating that the overall association was not driven solely by the small extreme groups.

The economic dimension also merits emphasis. Because GKR is derived from tests already ordered for virtually every severely injured patient, its incremental cost is effectively zero, and it imposes no additional sampling, consumable, or training burden, in contrast with advanced biomarker panels or quantitative imaging analytics that remain beyond the reach of most district hospitals.[23-26] In a health system where neurocritical-care capacity is concentrated in a few tertiary centres, a no-cost triage signal that helps direct intensive resources toward the patients most likely to benefit has clear value for both equity and efficiency.

A further methodological point concerns the handling of the non-normal distributions that

characterised almost every numerical variable. The consistent reliance on rank-based statistics, namely Spearman correlation, Kruskal–Wallis testing, and the rank-biserial effect size, was appropriate given that only potassium satisfied the normality assumption, and it protects the analysis against the influence of the extreme glucose and GKR values that are themselves biologically meaningful in this population. Parametric summarisation was reserved for potassium alone, where the mean and standard deviation are interpretable, ensuring that each variable was described and tested with a method matched to its distribution; this discipline is reflected in Table 2, where potassium is reported as a mean with standard deviation while glucose and GKR are reported as medians with ranges.

Future work should pursue multicentre prospective cohorts with hard mortality and functional endpoints, serial GKR measurement to characterise temporal dynamics, and direct head-to-head comparison and combination of GKR with MOST, CRASH, and IMPACT. A pragmatic interventional question also arises, namely whether GKR-guided escalation of monitoring and perioperative optimisation can modify outcome, a hypothesis best tested in a randomised framework. Until such data exist, GKR is most appropriately positioned as an inexpensive, rapidly available adjunct to, rather than a replacement for, comprehensive clinical assessment, and its greatest value is likely to be realised as an early triage signal that sharpens, rather than supplants, the judgement of the attending anaesthesiologist and intensivist.

5. Conclusion

In patients with severe traumatic brain injury undergoing craniotomy, the admission glucose-to-potassium ratio correlated positively with MOST-based mortality risk (rho = 0.376, $p < 0.001$) and outperformed glucose or potassium individually, discriminating high mortality risk with an area under the curve of 0.91 (cut-off 47.0; Cohen's d = 2.55) and acting as an independent predictor (adjusted odds ratio 1.35 per unit). Because it is computed from two inexpensive, universally available admission laboratory values, GKR offers a practical bedside tool for early mortality-risk stratification that is well suited to Indonesian tertiary referral practice, where it can support triage, perioperative vigilance, and resource allocation under existing cerebral-perfusion and intracranial-pressure frameworks. Given the retrospective single-centre design, these findings should be confirmed in multicentre prospective cohorts with hard mortality endpoints and, ideally, in trials evaluating whether GKR-guided management improves outcome.

6. References

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