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Glucose-to-potassium ratio as a predictor of in-hospital mortality in patients with spontaneous intracranial hemorrhage

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Abstract

Background & Objective: Spontaneous intracranial hemorrhage (ICH) is associated with high morbidity and mortality. Early identification of patients at risk for poor outcomes is crucial to guide management and optimize intensive care utilization. Glucose-to-potassium ratio (GPR) is an emerging biomarker that may reflect combined metabolic and systemic derangements. The objective of this study is to evaluate the prognostic performance of GPR in predicting in-hospital mortality among patients with spontaneous ICH. *Methods:* In this retrospective observational study, 168 consecutive patients diagnosed with spontaneous ICH between January and December 2024 were included. Demographics, clinical parameters, laboratory results, and interventions were extracted from electronic medical records. The primary outcome was in-hospital mortality. GPR was calculated from admission serum glucose and potassium levels. Statistical analyses included Mann-Whitney U and chi-square tests for group comparisons, receiver operating characteristic (ROC) curve analysis for predictive performance, and multivariable logistic regression to identify independent mortality predictors. *Results:* Among 168 patients, 103 (61.3%) survived and 65 (38.7%) died during hospitalization. Non-survivors were older (median 64 vs. 58 years, $p = 0.017$) and had lower GCS scores (12 vs. 15, $p < 0.001$). ROC analysis of GPR yielded an area under the curve of 0.718 (95% CI, 0.637–0.800) with an optimal cut-off of ≥37.29, sensitivity of 65.6%, and specificity of 70.8%. In multivariable logistic regression, higher GPR (OR 1.04, 95% CI 1.01–1.07, $p = 0.004$), older age (OR 1.04, 95% CI 1.01–1.06, $p = 0.002$), and decompressive craniectomy (OR 3.60, 95% CI 1.52–8.53, $p = 0.004$) independently predicted in-hospital mortality.
Conclusion: GPR is a simple, cost-effective, and readily obtainable biomarker that independently predicts in-hospital mortality in patients with spontaneous ICH. When combined with established predictors such as age and surgical intervention, GPR may facilitate early risk stratification and guide clinical management. Prospective multicenter studies are warranted to further validate these findings.

Keywords: Spontaneous intracranial hemorrhage, mortality, prognostic biomarkers, critical illness, metabolic stress, hyperglycemia

INTRODUCTION

Spontaneous intracranial hemorrhage is one of the most devastating forms of stroke, associated with high rates of morbidity and mortality despite advances in neurocritical care.[1-3] Early identification of patients at risk for poor outcomes is crucial to optimize management strategies, guide surgical decisions, and allocate intensive care resources effectively. Conventional prognostic markers, such as age, Glasgow Coma Scale (GCS), hematoma volume, and need for decompressive surgery, are widely used but may not fully capture the complex pathophysiological processes contributing to mortality.[3-6]

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Among laboratory parameters, blood glucose and serum potassium levels have been independently associated with adverse outcomes in acute neurological disorders.[7,13] Stress-induced hyperglycemia frequently occurs after intracranial hemorrhage and has been linked to increased hematoma expansion, perihematomal edema, and worse neurological recovery.[7-11] Similarly, hypokalemia is common in critically ill neurological patients[12,13], potentially reflecting catecholamine surge, insulin response, and systemic inflammatory activation, all of which may predispose to arrhythmia and hemodynamic instability.

The glucose-to-potassium ratio (GPR) is a simple and easily obtainable parameter that simultaneously incorporates two readily available biochemical markers.[14-19] Previous studies have suggested its potential prognostic value in various acute conditions, including subarachnoid hemorrhage, ischemic stroke, and cardiac arrest.[16-19] However, its role in predicting outcomes in spontaneous intracranial hemorrhage has not been adequately investigated. In this study, we aimed to evaluate the diagnostic and prognostic performance of the glucose-to-potassium ratio in predicting in-hospital mortality among patients with spontaneous intracranial hemorrhage.

METHODS

Study design and population

This retrospective observational study was conducted in the emergency department and neurosurgery unit of a tertiary care hospital. A total of 168 consecutive patients diagnosed with spontaneous intracranial hemorrhage between January 1, 2024, and December 31, 2024, were included. The diagnosis of spontaneous intracranial hemorrhage was confirmed by cranial computed tomography in all patients. Patients with traumatic intracranial hemorrhage, ischemic stroke, underlying malignancy, or incomplete medical records were excluded.

Data collection

Demographic characteristics, clinical parameters (GCS, vital signs), laboratory findings, and neuroimaging results were extracted from electronic medical records. Admission blood glucose and serum potassium values were used to calculate the glucose-to-potassium ratio (GPR). The (GPR) was calculated as serum glucose (mg/dL) divided by serum potassium (mEq/L) and was analyzed as a continuous variable. Interventions such as decompressive craniectomy were recorded, along with clinical outcomes.

Outcome

The primary outcome was in-hospital mortality. Secondary outcomes included length of hospital stay and need for surgical intervention.

Statistical analysis

All analyses were performed using the jamovi statistical software (version 2.6.26.0; The jamovi project, Sydney, Australia). Continuous variables were expressed as medians with interquartile ranges (IQR), and categorical variables as frequencies and percentages. Between-group comparisons (survivors vs. non-survivors) were made using the Mann-Whitney U test for continuous variables and the chi-square or Fisher's exact test for categorical variables. Receiver operating characteristic (ROC) curve analysis was performed to assess the discriminative performance of GPR for predicting in-hospital mortality, with the optimal cut-off value determined by the Youden index. Diagnostic performance metrics included sensitivity, specificity, predictive values, likelihood ratios, accuracy, and the area under the curve (AUC) with 95% confidence intervals (CI). Multivariable logistic regression analysis was conducted to identify independent predictors of in-hospital mortality. Variables with $p < 0.10$ in univariate comparisons and clinically relevant parameters (age, GCS, decompressive craniectomy, and GPR) were entered into the model. Results were reported as regression coefficients (β), standard errors, z-values, odds ratios (OR), and 95% CI. Model calibration was tested using the Hosmer-Lemeshow goodness-of-fit test, and model explanatory power was assessed using Nagelkerke's R^2. Statistical significance was set at a two-tailed $p < 0.05$.

A priori sample size estimation was performed based on the primary outcome of in-hospital mortality and the diagnostic performance of the glucose-to-potassium ratio. Assuming an expected AUC of approximately 0.70 for GPR, an alpha level of 0.05, and a statistical power of 80%, the minimum required sample size was calculated to be approximately 150 patients. Considering potential exclusions due to missing data and to ensure adequate events

for multivariable logistic regression analysis, all eligible consecutive patients during the study period were included. The final sample of 168 patients was therefore considered sufficient to achieve adequate statistical power for the primary analyses.

RESULTS

Patient characteristics

A total of 168 patients with spontaneous intracranial hemorrhage were included in the analysis, of whom 103 (61.3%) survived and 65 (38.7%) died during hospitalization.

Baseline demographic, clinical, hematologic, biochemical, and pathological characteristics of survivors and non-survivors are presented in Table 1. Non-survivors were significantly older compared with survivors (median 64 vs. 58 years, $p = 0.017$). Initial GCS scores were markedly lower in the non-survivor group (median 12 vs. 15, $p < 0.001$). Systolic blood pressure was higher among non-survivors (165 vs. 149 mmHg, $p = 0.005$).

Among laboratory parameters, non-survivors had significantly higher white blood cell counts (11.4 vs. 9.8 $\times 10^3/\mu L$, $p = 0.013$) and neutrophil counts (7.8 vs. 6.3 $\times 10^3/\mu L$, $p = 0.012$), whereas lymphocyte counts did not differ significantly. Serum glucose levels were notably elevated in non-survivors compared to survivors (163 vs. 124 mg/dL, $p < 0.001$), while serum potassium levels tended to be lower, though this difference was not statistically significant (4.1 vs. 4.2 mEq/L, $p = 0.142$). Aspartate aminotransferase levels were modestly higher in non-survivors (24 vs. 22 U/L, $p = 0.038$). Other biochemical parameters, including creatinine, sodium, and C-reactive protein, showed no significant differences between the groups.

Pathological imaging findings, such as subdural hematoma, epidural hematoma, intracerebral hematoma, and aneurysm, did not differ significantly between groups. However, the need for decompressive craniectomy was significantly higher in non-survivors compared with survivors (46.2% vs. 14.6%, $p < 0.001$).

Regarding inflammatory indices, the neutrophil-to-lymphocyte ratio was substantially higher in non-survivors than survivors (median 43.7 vs. 30.1, $p < 0.001$). Length of hospital stay did not differ significantly between groups.

Diagnostic performance of glucose-to-potassium ratio

The diagnostic performance of the GPR for predicting in-hospital mortality is shown in Table 2. ROC curve analysis demonstrated an area under the curve (AUC) of 0.718 (95% CI, 0.637–0.800; $p < 0.001$), indicating fair discriminative ability. The optimal cut-off value of GPR for mortality prediction was ≥37.29, yielding a sensitivity of 65.6% and specificity of 70.8%. The positive predictive value was 58.8%, and the negative predictive value was 76.4%. The positive likelihood ratio was 2.25, while the negative likelihood ratio was 0.49, with an overall accuracy of 68.8%.

Multivariable logistic regression analysis

Results of the multivariable logistic regression analysis are summarized in Table 3. In the multivariable logistic regression model, the model demonstrated acceptable fit with a Nagelkerke R^2 of 0.205 and an Akaike Information Criterion of 177. Independent predictors of in-hospital mortality included older age (OR 1.04, 95% CI 1.01–1.06; $p = 0.002$), undergoing decompressive craniectomy (OR 3.60, 95% CI 1.52–8.53; $p = 0.004$), and higher GPR (OR 1.04, 95% CI 1.01–1.07; $p = 0.004$). GCS score was inversely associated with mortality in the unadjusted analysis but did not retain statistical significance in the multivariable model (OR 0.92, 95% CI 0.83–1.02; $p = 0.103$).

DISCUSSION

In this retrospective observational study, we investigated the prognostic performance of the GPR in predicting in-hospital mortality among patients with spontaneous intracranial hemorrhage. Our findings demonstrated that elevated GPR was significantly associated with mortality, with fair discriminative ability in ROC analysis and independent predictive value in multivariable regression. Alongside age and decompressive craniectomy, GPR emerged as an accessible and cost-effective biomarker that may support early risk stratification in this critically ill population.

The primary finding of our study was the significant association between higher GPR values and in-hospital mortality. ROC curve analysis identified an optimal cut-off of 37.29, yielding moderate sensitivity (65.6%) and specificity (70.8%). Previous studies have

Table 1: Patient characteristics and clinical outcomes

Characteristic	Total (N = 168)	Survivors (n = 103)	Non-survivors (n = 65)	p-value
Demographic characteristics				
Age, years, median (IQR)	60 (48-73)	58 (43.5-69)	64 (55-76)	0.017
Gender, female, n (%)	61 (36.3)	34 (33)	27 (41.5)	0.340
Clinical parameters				
Glasgow Coma Scale, median (IQR)	15 (10-15)	15 (12-15)	12 (8-15)	<0.001
Systolic blood pressure, mm Hg, median (IQR)	153 (138.5-170)	149 (134-166)	165 (144.2-175.8)	0.005
Diastolic blood pressure, mm Hg, median (IQR)	83 (74-93)	81 (72-92)	86.0 (77.2-93.8)	0.080
Heart rate, beats/min, median (IQR)	82 (74.8-91)	81 (73.2-90)	83.5 (75-91.8)	0.676
Oxygen saturation, %, median (IQR)	96 (94-98)	96 (93-98)	96 (95-98.8)	0.081
Hematologic parameters				
White blood cells, $\times 10^3/\mu L$, median (IQR)	10.3 (8.5-14.5)	9.8 (8-13.6)	11.4 (9.0-15.2)	0.013
Neutrophils, $\times 10^3/\mu L$, median (IQR)	7.1 (5.2-11.2)	6.3 (4.9-10)	7.8 (6-12)	0.012
Lymphocytes, $\times 10^3/\mu L$, median (IQR)	2.1 (1.2-3.3)	2.2 (1.3-3.2)	1.8 (1.1-3.3)	0.571
Hemoglobin, g/dL, median (IQR)	13.9 (12.4-15.1)	14.4 (12.5-15.3)	13.8 (12-15)	0.086
Hematocrit, %, median (IQR)	42.2 (37.7-45.2)	42.5 (37.9-45.4)	42.2 (36.6-44.2)	0.239
Platelets, $\times 10^3/\mu L$, median (IQR)	250 (195-290)	251 (200.2-287.2)	242 (184-291)	0.980
Biochemical parameters				
Glucose, mg/dL, median (IQR)	134 (110.2-173.2)	124 (101.5-147)	163 (128-195)	<0.001
Aspartate aminotransferase, U/L, median (IQR)	22 (18-29)	22 (17-28)	24 (19.8-31)	0.038
Alanine aminotransferase, U/L, median (IQR)	17 (13-22)	17.5 (12-23)	17 (14-22)	0.941
Blood urea nitrogen, mg/dL, median (IQR)	32.8 (27.1-39.5)	32.4 (26.0-38.5)	34.6 (27.8-41.5)	0.100
Creatinine, mg/dL, median (IQR)	0.8 (0.7-1)	0.8 (0.7-1)	0.8 (0.7-1)	0.606
Sodium, mEq/L, median (IQR)	139 (137.4-140.8)	139 (137.6-140.7)	139 (137.4-141)	0.890
Potassium, mEq/L, median (IQR)	4.2 (3.8-4.5)	4.2 (3.9-4.5)	4.1 (3.6-4.4)	0.142
International normalized ratio, median (IQR)	1 (1-1.1)	1 (1-1.1)	1 (1-1.2)	0.202
Albumin, g/L, median (IQR)	42.9 (40.4-45.3)	43.0 (40.5-44.9)	42.5 (40.4-46)	0.649
C-reactive protein, mg/L, median (IQR)	3.3 (1.6-12.3)	3.5 (1.8-12.1)	2.9 (1.5-12.3)	0.713
Pathological findings				
Subdural hematoma, n (%)	69 (41.1)	48 (46.6)	21 (32.3)	0.094
Epidural hematoma, n (%)	6 (3.6)	4 (3.9)	2 (3.1)	1.000
Intracerebral hematoma, n (%)	69 (41.1)	37 (35.9)	32 (49.2)	0.122
Aneurysm, n (%)	50 (29.8)	31 (30.1)	19 (29.2)	1.000

Table 1: (continued)

Characteristic	Total (N = 168)	Survivors (n = 103)	Non-survivors (n = 65)	p-value
Interventions				
Decompressive craniectomy, n (%)	45 (26.8)	15 (14.6)	30 (46.2)	<0.001
Door to operation time, hours, median (IQR)	8 (5-16)	11 (5-17)	7 (4-12)	0.052
Inflammatory markers				
Neutrophil-to-lymphocyte ratio, median (IQR)	34.6 (25.4-46.5)	30.1 (23.4-39)	43.7 (31.4-56.9)	<0.001
Outcome				
Length of stay, days, median (IQR)	9 (5-16)	9 (5-16)	9 (5-16)	0.540

Table 2: Diagnostic performance of glucose-to-potassium ratio for predicting in-hospital mortality

Parameter	Value (95% CI)
Area under curve	0.718 (0.637-0.800)
Optimal cut-off Value	≥37.29
Sensitivity	65.57% (52.31-77.27%)
Specificity	70.83% (60.67-79.67%)
Positive predictive value	58.82% (49.89-67.21%)
Negative predictive value	76.40% (69.12-82.41%)
Positive likelihood ratio	2.248 (1.567-3.225)
Negative likelihood ratio	0.486 (0.336-0.703)
Accuracy	68.79% (60.92-75.94%)
p-value	<0.001

investigated the prognostic value of GPR in patients with intracerebral hemorrhage. Liu *et al*.[20] demonstrated that elevated GPR was independently associated with increased short- and long-term mortality in critically ill ICH patients, while Wu *et al.*[17] reported that GPR predicted poor outcomes more effectively than glucose or potassium alone. Another recent study demonstrated an independent association between admission GPR and poor outcomes in patients with acute ischemic stroke, including unfavorable functional outcomes and mortality at follow-up. These findings support the broader relevance of GPR as a prognostic biomarker across cerebrovascular disorders.[18] However, many of these studies relied on large database cohorts, focused on long-term outcomes, or lacked integration with key clinical variables.

In contrast, the present study focuses on in-hospital mortality in a consecutive spontaneous ICH cohort and evaluates the independent prognostic value of admission GPR alongside established clinical predictors, thereby providing clinically relevant evidence for early risk stratification. From a pathophysiological

Table3: Multivariable logistic regression analysis of predictors of in-hospital mortality

Predictor	β (Coefficient)	Standard Error	Z-value	p-value	OR (95% CI)
Intercept	-3.65	1.30	-2.80	0.005	-
Age (per year)	0.038	0.012	3.15	**0.002**	1.04 (1.01-1.06)
Decompressive craniectomy (yes)	1.28	0.440	2.91	**0.004**	3.60 (1.52-8.53)
Glasgow coma scale	-0.086	0.053	-1.63	0.103	0.92 (0.83-1.02)
Glucose-to-potassium Ratio	0.039	0.013	2.91	**0.004**	1.04 (1.01-1.07)

perspective, hyperglycemia may exacerbate hematoma expansion and perihematomal edema, while hypokalemia may predispose patients to arrhythmias and hemodynamic instability; together, these mechanisms may contribute to increased mortality risk.[16-18]

Our findings are consistent with emerging literature. In a large cohort of critically ill ICH patients from the MIMIC-IV databas, elevated GPR was independently associated with increased 30-day mortality (HR 1.32), as well as 90-day and 1-year mortality (HR 1.27 and 1.22, respectively).[20] Similarly, in aneurysmal subarachnoid hemorrhage (aSAH), plasma GPR on admission predicted 3-month mortality with an AUC of 0.747 and remained an independent predictor after multivariable adjustment.[21] Another study in aSAH reported that non-survivors had significantly higher median GPR values (46.7) compared to survivors (37.8), with independent association to 30-day mortality (OR 4.04).[22] In addition, a retrospective analysis of 92 ICH patients showed that GPR, unlike glucose or potassium alone, significantly improved prognostic accuracy for 90-day functional outcomes and mortality and retained independent predictive value even after adjustment for GCS score and hematoma volume.[17] Overall, these findings confirm that elevated GPR consistently correlates with higher mortality across different forms of acute neurologic injury, supporting its use as a robust and readily measurable biomarker in neurocritical care.

In addition to GPR, our study confirmed the prognostic impact of established predictors, including advanced age and the need for decompressive craniectomy. Consistent with prior reports, older patients had significantly higher mortality risk, likely reflecting reduced physiological reserve and increased comorbid burden.[23,24] The strong association of decompressive craniectomy with mortality in our cohort probably reflects confounding by indication, as patients undergoing surgery generally presented with more severe hemorrhage and neurological compromise.[25,26] Although the GCS was significantly lower among non-survivors in univariate analyses, it did not retain significance in the multivariable model, suggesting that GPR and age may capture overlapping prognostic information beyond clinical examination alone.

This study has several limitations. First, its retrospective, single-center observational design may limit the generalizability of the findings and is inherently subject to data collection bias and unmeasured confounding factors. Second, detailed radiological parameters such as hematoma volume, location, and perihematomal edema—known to be important prognostic determinants in intracerebral hemorrhage—were not available and therefore could not be incorporated into the analyses. Third, glucose and potassium levels were assessed only at hospital admission; thus, temporal fluctuations of the glucose-to-potassium ratio during hospitalization, which may carry additional prognostic information, were not evaluated. Finally, although the study was adequately powered for the primary outcome based on a priori sample size estimation, the sample size may still be insufficient for more detailed subgroup analyses across different hemorrhage subtypes, which should be addressed in future multicenter prospective studies.

In conclusion, the GPR represents a simple, readily available, and cost-effective biomarker that provides valuable prognostic information in patients with spontaneous intracranial hemorrhage. Our findings suggest that GPR, when used alongside established predictors such as age and surgical intervention, may help clinicians identify high-risk patients early and optimize management strategies. Larger prospective multicenter studies are warranted to validate these results and further define the clinical utility of GPR in neurocritical care.

DISCLOSURE

Ethic: The study was approved by the local ethics committee Ümraniye Clinical Sample Research Training and Research Hospital Non-Interventional Ethics Committee Date: 09/11/2025: Approval No: 274. Given the retrospective nature of this study, the requirement for informed consent was formally waived by the Clinical Research Ethics Committee.

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