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RESEARCH ARTICLE

Prognostic Value of the Glucose-to-Potassium Ratio for Mortality in Critically Ill COVID-19 Patients

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ABSTRACT

Objectives: Previous studies have demonstrated that an elevated serum glucose-to-potassium ratio (GPR) is associated with poor prognosis in patients with subarachnoid hemorrhage, ischemic stroke, and pulmonary embolism. This study aimed to investigate the association between GPR and mortality among critically ill patients with COVID-19.

Methods: This retrospective study included patients with a confirmed diagnosis of COVID-19 who were admitted to the intensive care unit (ICU) of a tertiary-level hospital between March 2020 and January 2022. Laboratory data and 90-day survival status following ICU admission were recorded. The glucose-to-potassium ratio was calculated for each patient.

Results: The mean GPR was significantly higher in non-survivors than in survivors (45.8±17.0 vs. 38.1±15.1, p=0.006). A significant positive correlation was observed between GPR and mortality (r=0.247, p=0.002). Binary logistic regression analysis identified GPR as an independent risk factor for mortality (odds ratio=1.08, 95% CI 1.03–1.14, p=0.002). Receiver operating characteristic (ROC) analysis determined a GPR cut-off value of >32.1, yielding a sensitivity of 81.9% and specificity of 50.0% (AUC=0.647, 95% CI 0.565–0.723, p=0.002). The prevalence of kidney disease as a comorbidity was significantly higher among non-survivors (p=0.009).

Conclusion: Elevated glucose-to-potassium ratio and the presence of kidney disease at the time of ICU admission are associated with poor prognosis in patients with COVID-19.

Keywords: COVID-19, glucose, intensive care unit, potassium, mortality

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Introduction

A subset of patients with COVID-19 infection progresses to a critical form of the disease requiring intensive care unit (ICU) admission. Mortality risk is particularly increased in those with comorbidities such as hypertension, diabetes mellitus (DM), chronic obstructive pulmonary disease (COPD), obesity, cardiovascular disease, and kidney disease.[1] During the first year of the pandemic caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), hospital mortality among critically ill patients exceeded 50%.[2] Several hematological and biochemical biomarkers have been proposed for predicting mortality in severe COVID-19, including decreased hemoglobin or platelet counts, and elevated levels of cardiac troponin I, D-dimer, C-reactive protein (CRP), lactate dehydrogenase (LDH), aspartate aminotransferase (AST), alanine aminotransferase (ALT), procalcitonin, and international normalized ratio (INR).[3,4]

The serum glucose-to-potassium ratio (GPR) has recently emerged as a novel prognostic marker in acute neurological and cardiovascular disorders. An elevated GPR has been associated with poor outcomes and increased mortality

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in patients with aneurysmal subarachnoid hemorrhage (SAH).[5–11] In aneurysmal rupture, acute stress induces catecholamine release, increased glucagon secretion, and reduced insulin activity, resulting in hyperglycemia. Hyperglycemia-induced inflammation may contribute to microvascular injury, hematoma expansion, and re-bleeding through oxidative stress mechanisms. Simultaneously, catecholamine and insulin activity lower serum potassium levels by stimulating the Na^+/K^+-ATPase pump and K-ATP channels, while systemic inflammation further exacerbates hypokalemia.[12] These processes collectively increase the glucose-to-potassium ratio, reflecting the severity of metabolic and inflammatory stress.

Given that both hyperglycemia and systemic inflammation play major roles in the pathophysiology of critical COVID-19, the GPR may serve as a simple, cost-effective biomarker for prognostic assessment. However, the prognostic significance of GPR in critically ill COVID-19 patients has not yet been evaluated. Therefore, this study aimed to investigate the association between GPR at ICU admission and mortality in critically ill patients with COVID-19. Identifying patients with elevated GPR may facilitate early recognition of those at increased risk of poor outcomes.

Methods

Study Design

This was a retrospective observational cohort study conducted in the 16-bed General Intensive Care Unit (ICU) of our hospital. Ethical approval was obtained from the Local Ethics Committee (23/02/2022, E1-22-2413) prior to data collection. This study was conducted in accordance with the principles of the Declaration of Helsinki. The study included consecutive patients aged >18 years, of either sex, who were admitted to the ICU between 18 March 2020 and 31 January 2022 with a confirmed diagnosis of COVID-19 based on reverse transcription polymerase chain reaction (RT-PCR) positivity for the SARS-CoV-2 virus.

For each patient, demographic characteristics (age and sex), comorbidities, need for mechanical ventilation, Acute Physiology and Chronic Health Evaluation II (APACHE II) score, length of ICU stay, total hospital stay, and 90-day survival status after ICU admission were recorded. Patients without available laboratory data were excluded.

Laboratory parameters, including serum glucose, potassium, lactate dehydrogenase (LDH), creatinine, platelet count, ferritin, C-reactive protein (CRP), procalcitonin, lactate, and D-dimer levels at the time of ICU admission, were retrieved from the hospital's electronic medical records. The glucose-to-potassium ratio (GPR) was calculated for each patient.

Statistical Analysis

All data were analyzed using SPSS version 25.0 (IBM Corp., Armonk, NY, USA) and MedCalc version 15.8 (MedCalc Software bvba, Ostend, Belgium). Descriptive statistics were presented as mean±standard deviation (SD) for normally distributed variables and as median (Q1–Q3) for non-normally distributed variables. The conformity of data to a normal distribution was assessed using the Kolmogorov–Smirnov test, skewness–kurtosis values, and graphical methods (histogram, Q–Q plot, stem-and-leaf plot, and boxplot).

Comparisons between two independent groups were performed using the Independent Samples t-test for normally distributed data and the Mann–Whitney U test for non-normally distributed data. The Chi-square test was used to analyze categorical variables. Receiver Operating Characteristic (ROC) curve analysis was applied to assess the discriminatory ability of variables. Survival rates were estimated using the Kaplan–Meier method, and differences between groups were compared using the Log-rank, Breslow, and Tarone–Ware tests. Correlations between variables were evaluated with Spearman's rho correlation analysis. A p value of <0.05 was considered statistically significant.

The statistical power of the study was calculated based on the GPR variable using G*Power version 3.1.9.7 (Franz Faul, University of Kiel, Germany). With sample sizes of $n_1=94$ and $n_2=56$, $\alpha=0.05$, and an effect size (d) of 0.52, the achieved power was 86%.

Results

The baseline characteristics of the 150 patients included in the study are presented in Table 1. The cohort consisted of 78 males and 72 females, with a mean age of 67.7±15.6 years. Comparisons between survivors (n=56) and non-survivors (n=94) are summarized in Table 1. The mean age of non-survivors was significantly higher than that of survivors ($p<0.001$). The prevalence of kidney disease was also significantly higher among non-survivors ($p<0.001$). Serum potassium levels were significantly lower in non-survivors compared to survivors ($p<0.05$). The mean GPR in non-survivors (45.8±17.0) was significantly higher than in survivors (38.1±15.1, $p<0.05$). A significant positive correlation was observed between GPR and mortality ($r=0.247$, $p<0.05$).

Univariate logistic regression analysis revealed that mortality risk increased by approximately 1.1-fold with older age, 1.2-fold with higher APACHE II scores, 1.1-fold with longer ICU stay, 3.4-fold with elevated creatinine, and 1.1-fold with elevated GPR, while higher potassium levels

Table 1. Comparisons of the survivor and non-survivor groups

	All patients (n=150)		Survivor (n=56)		Non-Survivor (n=94)		p
	n	%	n	%	n	%	
Gender							
Female	72		24	42.9	48	51.1	0.331[a]
Male	78		32	57.1	46	48.9	
Age (years)	67.7±15.6		62.0±16.8		71.1±13.9		<0.001[b]
Comorbidities							
None	30		16	28.6	14	14.9	0.070[a]
Present	120		40	71.4	80	85.1	
Diabetes Mellitus	55		17	30.4	38	40.4	0.288[a]
Hypertension	83		26	46.4	57	60.6	0.90[a]
Coronary artery disease	33		12	21.4	21	22.3	1.000[a]
Chronic heart failure	25		5	8.9	20	21.3	0.083[a]
Stroke	7		4	7.1	3	3.2	0.425[a]
Thyroid disorders	10		4	7.1	6	6.4	1.000[a]
Liver disease	3		2	3.6	1	1.1	0.556[a]
Kidney disease	13		0	0.0	13	13.8	0.009[a]
Chronic obstructive lung disease	27		6	10.7	21	22.3	0.116[a]
Cancer	16		4	7.1	12	12.8	0.420[a]
APACHE II	23.6±12.3		13.3±8.5		29.8±9.8		<0.001[b]
ICU stay (days)	12.8±10.5		7.0 (4.0–10.8)		12.0 (6.8–21.3)		<0.001[c]
Hospital stay (days)	17.2±11.7		17.2±10.5		17.2±12.4		0.991[b]
Breathing support							
Invasive ventilation	96		2	3.6	94	100.0	<0.001[a]
Non-invasive ventilation	2		2	3.6	0	0.0	
High flow	26		26	46.4	0	0.0	
Mask/nasal	26		26	46.4	0	0.0	
Glucose (mg/dL)	170.7±75.0		160.9±64.6		176.5±80.3		0.218 b
Potassium (mEq/L)	4.0±0.7		4.2±0.5		3.8±0.8		<0.001[b]
Glucose to potassium ratio	42.91±16.67		38.1±15.1		45.8±17.0		0.006[b]
Creatinine (mg/dL	1.33±1.28		0.83 (0.64–1.13)		1.09 (0.80–1.71)		0.002[c]
Lactate dehydrogenase (U/L)	526.8±231.8		460.1±217.1		566.6±232.2		0.006[b]
D-dimer (mg/L)	4.4±7.5		1.2 (0.7–1.9)		2.0 (1.0–4.8)		0.001[c]
Procalcitonin (µg/L)	6.9±34.0		0.10 (0.04–0.28)		0.24 (0.11–1.25)		<0.001[c]
Ferritin (µg/L)	770.4±795.2		362.0 (148.5–743.8)		670.0 (303.0–1254.8)		0.001[c]
Platelet (x10^9/L)	270.0±155.5		253.0 (190.3–371.8)		245.5 (175.8–331.3)		0.282[c]
Lactate (mmol/L)	2.6±1.3		2.5 (1.8–3.1)		2.4 (1.7–3.1)		0.933[c]
C-reactive protein (g/L)	0.12±0.10		0.08 (0.04–0.14)		0.13 (0.06–0.19)		0.009[c]

[a]: Chi-Square test (n (%)); [b]: Independent samples t test (Mean±SD); [c]: Mann-Whitney U test (Median (Q1 – Q3)). APACHE II: Acute Physiology and Chronic Health Evaluation II; ICU: Intensive care unit.

were associated with a 0.14-fold decrease in mortality risk. The predictive model correctly classified 89 of 94 non-survivors (94.7%) and 49 of 56 survivors (87.5%), with an overall accuracy of 92.0%.

Multivariate logistic regression identified GPR as an independent predictor of mortality (odds ratio=1.08, 95% CI 1.03–1.14, p<0.05) (Table 2). Receiver Operating Characteristic (ROC) analysis determined a GPR cut-off value of > 32.1, providing 81.9% sensitivity and 50.0% specificity (AUC=0.647, 95% CI 0.565–0.723, p<0.05). ROC results for significant predictors are shown in Table 3 and Figure 1.

Discussion

The present study demonstrated that the glucose-to-potassium ratio (GPR) was significantly higher in non-

Table 2. Multivariate logistic regression analysis of the risk factors

Risk factor	β	SE	Wald	Odds	95% CI	p*
Age (years)	0.079	0.029	7.310	1.08	1.02–1.15	0.007
APACHE II	0.217	0.043	25.361	1.24	1.14–1.35	<0.001
ICU stay (days)	0.098	0.044	5.041	1.10	1.01–1.20	0.025
Creatinine (mg/dL)	1.227	0.567	4.686	3.41	1.12–10.36	0.030
Lactate dehydrogenase (U/L)	0.004	0.002	4.580	1.00	1.00–1.01	0.052
Potassium (mEq/L)	-1.945	0.633	9.432	0.14	0.04–0.49	0.002
D-dimer (mg/L)	0.173	0.095	3.323	1.19	0.99–1.43	0.068
Procalcitonin (µg/L)	-0.025	0.014	3.255	0.98	0.95–1.00	0.071
GPR	0.079	0.025	9.957	1.08	1.03–1.14	0.002
Constant	-9.976	3.703	7.257			

*: Binary Logistic Regression Test (only given for variables remaining in the model), Nagelkerke R2=0.816, Hosmer and Lemeshow Test=0.840. SE: Standart error; APACHE II: Acute Physiology and Chronic Health Evaluation II; ICU: Intensive care unit; GPR: Glucose to potassium ratio.

Table 3. ROC analysis results and cut-off values of risk factors

	AUC	95% CI	Cut-off	Sensitivity	Specificity	Youden index	+PV	-PV	p*
Age (years)	0.660	0.579–0.735	>64	69.2	589	0.281	73.9	53.2	0.001
APACHE II	0.895	0.835–0.939	>19	86.2	80.4	0.665	88.0	77.6	<0.001
ICU stay (days)	0.699	0.619–0.771	>9	66.0	69.6	0.356	78.5	54.9	<0.001
Creatinine (mg/dL)	0.654	0.572–0.730	>0.94	66.0	67.9	0.338	77.5	54.3	0.001
Lactate dehydrogenase (U/L)	0.651	0.569–0.727	>539	51.1	78.6	0.296	80.0	48.9	0.001
Potassium (mEq/L)	0.698	0.617–0.770	≤3.7	52.1	87.5	0.396	87.5	52.1	<0.001
D-dimer (mg/L)	0.668	0.586–0.742	>2.13	48.9	82.1	0.311	82.1	48.9	<0.001
Procalcitonin (µg/L)	0.687	0.607–0.760	>0.14	69.2	67.9	0.370	78.3	56.7	<0.001
Ferritin (µg/L)	0.657	0.575–0.732	>895	38.3	85.7	0.240	81.8	45.3	0.001
C-reactive protein (g/L)	0.628	0.546–0.706	>0.15	41.5	85.7	0.272	83.0	46.6	0.005
GPR	0.647	0.565–0.723	>32.1	81.9	50.0	0.319	73.3	62.2	0.002

*: ROC curve analysis. AUC: Area under the curve; CI: Confidence interval; GPR: Glucose to potassium ratio; APACHE II: Acute Physiology and Chronic Health Evaluation II; ICU: Intensive care unit.

survivors compared with survivors among critically ill COVID-19 patients, and a cut-off value of 32.1 predicted mortality with moderate accuracy. Both serum glucose and potassium are routinely measured biochemical parameters, reflecting metabolic and hormonal responses to stress. Glucose homeostasis is maintained through the balance between anabolic and catabolic hormones—insulin reduces serum glucose, whereas glucagon, cortisol, and catecholamines increase it. Potassium, the major intracellular cation, is essential for cellular membrane potential and excitability, and its serum level is tightly regulated by renal and hormonal mechanisms.

Previous studies have identified GPR as a prognostic marker in various acute conditions. Matano et al.[13] reported a significant correlation between high GPR and cerebral infarction secondary to vasospasm after subarachnoid hemorrhage. Wu et al.[11] found elevated GPR levels in patients with spontaneous intracerebral hemorrhage compared with healthy controls and showed its association with larger hematoma size, lower Glasgow Coma Scale scores, and poorer 90-day outcomes. Similarly, Lu et al.[14] demonstrated a positive correlation between baseline GPR and 30-day mortality in ischemic stroke. Zhou et al.[15] and Shibata et al.[16] both reported that admission GPR independently predicted short-term mortality in severe traumatic brain injury. In addition, GPR has been linked to disease severity in other systemic conditions, including pulmonary embolism,[17] blunt abdominal trauma,[18] and carbon monoxide poisoning.[19] In organophosphate poisoning, Sharif et al.[20] reported that GPR>41.07 predicted intermediate syndrome with 93% sensitivity and specificity. Collectively, these studies support the role of GPR as a reliable and low-cost biomarker of stress-related metabolic imbalance and systemic inflammation.

Hyperglycemia, a major determinant of elevated GPR, is known to predict adverse outcomes in critical illness. Umpierrez et al.[21] observed a markedly higher mortality rate in patients with newly diagnosed hyperglycemia

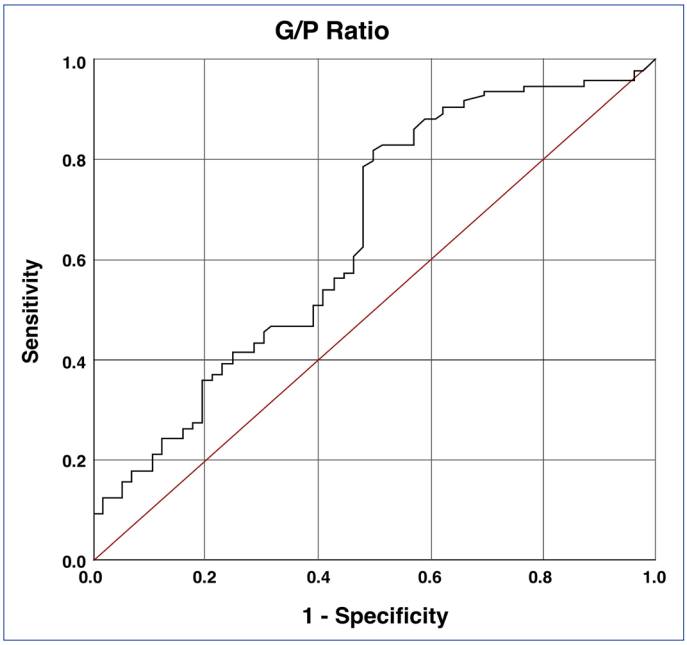

Figure 1. ROC curves for GPR.

ROC: Receiver operating characteristic; GPR: Glucose to potassium ratio.

compared with normoglycemic or previously diabetic patients. Numerous studies have similarly demonstrated that hyperglycemia on admission predicts mortality in myocardial infarction and stroke.[22–28] In COVID-19, Chen et al.[29] showed in a meta-analysis that elevated glucose levels were significantly associated with disease severity. Experimental studies have provided mechanistic insights: chronic inflammation and cytokine release (e.g., lipopolysaccharide-induced inflammation, interferon-γ activation) impair insulin signaling, leading to stress-induced hyperglycemia.[30–32] Furthermore, SARS-CoV-2 directly infects pancreatic β-cells and renal tubular cells via ACE2 receptors, potentially causing new-onset or exacerbated diabetes.[33–35] Although our study found higher glucose levels among non-survivors, this difference did not reach statistical significance, likely due to variability in glycemic control and comorbid diabetes status.

Potassium metabolism may also be disrupted in severe COVID-19. Fukui et al.[36] reported that low serum potassium predicted progression of chronic kidney disease. In our study, low potassium and high creatinine levels, together with pre-existing kidney disease, were associated with higher mortality. These findings are consistent with prior meta-analyses demonstrating increased mortality among COVID-19 patients with chronic kidney disease.[37] SARS-CoV-2–related renal injury, tubular dysfunction, and increased urinary potassium loss may contribute to hypokalemia in this setting.

Elevated inflammatory biomarkers—procalcitonin, ferritin, and CRP—were also associated with mortality in our cohort, highlighting the role of systemic inflammation in disease progression. Severe inflammation and stress-induced hormonal dysregulation may together disturb glucose and potassium balance, resulting in an increased GPR. Therefore, GPR can be considered an integrative biomarker reflecting both metabolic and inflammatory stress, and may serve as a practical prognostic indicator in critically ill COVID-19 patients.

This study has several limitations. First, its retrospective and single-center design may limit the generalizability of the findings. Second, potential confounding factors such as pre-existing diabetes mellitus, medications affecting glucose or potassium metabolism, and variations in glycemic control during ICU follow-up could not be fully adjusted. Third, only admission laboratory values were analyzed; serial measurements over time might provide a more dynamic understanding of GPR trends. Finally, the sample size, although adequate for statistical power, was relatively modest, and larger multicenter prospective studies are warranted to validate these results.

Conclusion

In conclusion, an elevated glucose-to-potassium ratio (GPR) on ICU admission was associated with increased mortality among critically ill patients with COVID-19. Given that serum glucose and potassium are routinely measured, GPR represents a simple, inexpensive, and readily available biomarker that may assist clinicians in identifying patients at higher risk of poor outcomes. Further large-scale, prospective studies are needed to confirm these findings and to explore whether GPR-guided metabolic optimization could improve survival in critically ill populations.

Limitations of this study include the relatively low number of patients and the fact that the data were obtained retrospectively.

Disclosures

Ethics Committee Approval: The study was approved by the Ankara City Hospital Ethics Committee (no: E1-22-2413, date: 23/02/2022).

Informed Consent: Informed consent was obtained from all participants.

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