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Usefulness of plasma glucose to potassium ratio in predicting the short-term mortality of patients with aneurysmal subarachnoid hemorrhage

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ABSTRACT

Rationale and objectives: To investigate the relationship between the glucose/potassium ratio (GPR) at admission and 30-day mortality in patients diagnosed with aneurysmal subarachnoid hemorrhage (SAH) in the emergency department (ED). *Materials and methods:* Patients with a modified Rankin Scale (mRS) score of \leq 2 before SAH and patients aged 18 years or older were included in the study. The patients were divided into two groups based on their functional outcomes (poor-good) and 30-day mortality rates (survivor and

non-survivor) and their clinical and laboratory values were compared. *Results:* The study included 134 patients with a mean age of 65.9 ± 16.7 years, of whom 68 (50.7 %) were female. The mean glucose and GPR levels in the poor functional outcome group were significantly higher than those in the good functional outcome group (p = 0.003, p = 0.03, respectively). The mean glucose and GPR levels in the non-survivor group were significantly higher than those in the survivor group (p = 0.004, p = 0.023, respectively). Multivariate logistic regression analysis identified GPR as an independent predictor of 30-day mortality (p = 0.043, OR: 4.041, 95 % CI: 1.45–26.147), alongside the Rankin Scale score (p = 0.002, OR: 12.714, 95 % CI: 2.578–62.706). Other variables, including age, Hunt-Hess score, and Glasgow Coma Scale, were not statistically significant.

Conclusion: The findings indicate that the GPR is a significant independent predictor of short-term mortality in patients with aneurysmal subarachnoid hemorrhage. The translation of these findings into clinical practice may help achieve better outcomes in the management of SAH patients.

1. Introduction

Subarachnoid hemorrhage (SAH) is a neurological emergency pathology. The leading source of SAH is trauma, whereas aneurysms are the primary source of spontaneous SAH, representing 75–80 % of occurrences. Spontaneous SAH is characterized by the direct passage of blood into the subarachnoid space due to the rupture of an artery or vein. The rupture of an intracranial aneurysm is the most frequent source of spontaneous SAH [1–3].

Aneurysmal SAH can occur at any age without a specific age range. Its incidence varies depending on age, gender, and geographic

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region worldwide, and it is not possible to give an exact and definitive value on its frequency. In North America, approximately 30,000 patients are diagnosed with SAH annually, and the incidence of SAH is between 2 and 25 cases per 100,000 people. While lower numbers of 2 cases per 100,000 are reported in China and 4 cases per 100,000 in South and Central America, higher rates of 19–23 cases per 100,000 are reported in Finland and Japan [4,5].

Despite variations in data among studies, it is reported that the 30-day mortality rate after SAH reaches up to 50 %. When looking at the mortality rates, it is reported that the majority of cases are lost within the first week, 10 % during bleeding, and 25 % within the first 24 h [6-8].

Emergency Departments (EDs) are the first points of contact for patients dealing with critical and urgent situations. Conditions requiring critical care, such as SAH, can lead to life-threatening complications that necessitate early intervention and treatment. Therefore, early identification and appropriate management of these patients is of great importance. By doing so, the quality of life and survival rates for patients will increase, while also reducing the burden on the healthcare system.

In SAH, which has high mortality rates, it is important for the managing physician to diagnose patients with poor prognosis early. For this purpose, the roles of various biochemical markers such as glucose, lactate, and C-reactive protein in predicting the prognosis of SAH patients have been investigated [9–11]. Glucose/potassium ratio (GPR) has been highlighted as a biomarker used for this purpose. In situations that create stress in the body, such as SAH, plasma glucose levels will increase due to hormonal changes, while potassium levels will decrease, and therefore the plasma GPR will increase. Increased GPR has been reported to be associated with poor functional outcomes and mortality among SAH patients [12].

The aim of this study is to investigate the relationship between the GPR at admission and 30-day mortality in patients diagnosed with aneurysmal SAH in the ED.

2. Methods

This retrospective cohort study was conducted in the ED of a tertiary hospital (an 650-bed institution with ~223,000 ED visits annually). The study was approved by the Şişli Hamidiye Etfal Education and Research Hospital ethics committee (Decision Number: 2207, Date: December 27, 2022) without the need for informed consent due to its retrospective nature. Patients who presented to the ED between December 2021 and December 2022 and were diagnosed with aneurysmal SAH, had a modified Rankin Scale (mRS) score of \leq 2 before SAH, and were aged 18 years or older were included in the study. The exclusion criteria for the study were as follows: patients with traumatic SAH, patients diagnosed with a condition other than SAH, patients transferred from another hospital, patients with a pre-SAH mRS score >2, patients for whom GPR could not be calculated, patients with diabetes mellitus-acute or chronic renal failure, and patients who could not be followed up. Patients with diabetes mellitus, persistent hyperglycemia is common, and in renal failure, potassium imbalance frequently occurs. The inclusion of these patients could have distorted GPR, thus affecting its validity as a prognostic marker in this study.

The patients' demographic data, laboratory results, and clinical findings were recorded by using the hospital's electronic medical

Table 1

Characteristics and out	tcomes of the	study po	pulation.
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		Min-Max			Median	$\text{Mean} \pm \text{SE}$	0/n-%	
Age (years)		26.0	-	94.0	69.0	65.9	±	16.7
Gender	Woman Man		-			68 66	-	50.7 % 49.3 %
Sistolic blood pressure (r	nmHg)	77.0	_	252.0	150.0	153.3	±	33.3
Diastolic blood pressure	(mmHg)	32.0	-	200.0	82.0	85.9	±	22.5
Heart rate (bpm)		52.0	_	140.0	85.0	87.0	±	17.8
Hunt-Hess score		1.0	_	5.0	3.0	2.9	±	1.5
Glasgow coma scale		3.0	_	15.0	15.0	11.7	±	4.2
Rankin scale		0.0	-	6.0	4.5	3.3	±	2.5
White blood cell (\times 10/µl)		4.0	-	31.8	10.8	11.5	±	4.9
Hemoglobin (g/dl)		6.5	-	28.0	13.1	13.1	±	2.5
Platelet (x10/µl)		10.0	-	524.0	244.0	241.9	±	78.3
Glucose (mmol/L)		4.1	-	24.6	7.8	8.8	±	3.4
Potassium (mmol/L)		2.9	-	5.9	4.0	4.1	±	0.6
Glucose/Potassium raito		1.0	_	5.9	2.0	2.2	±	0.9
Treatment						6		14.0.0/
Endovascular Coiling						6		14.3 %
Neurosurgical Clipping						36		85.7 %
Functional outcome	Good (mRS 0-2)					64		47.8 %
	Poor (mRS 3-6)					70		52.2 %
30-day mortality	(-)					70		52.2 %
	(+)					64		47.8 %

mRS: modified Rankin scale.

record system. A mRS score between 3 and 6 was defined as a poor functional outcome. GPR was simply calculated by dividing serum glucose by serum potassium. The primary outcome of the study was all-cause 30-day mortality. The secondary outcome was the functional outcome status at 30 days.

2.1. Statistical analysis

The data was analysed using various descriptive statistics such as mean, standard deviation, median, minimum, maximum, frequency, and ratio values. The distribution of variables was determined through the application of the Kolmogorov-Smirnov test. To assess the quantitative independent variables, the Independent Sample *t*-test and Mann-Whitney *U* test were utilized. For the qualitative independent variables, the Chi-square test and Fisher's exact test were implemented when the conditions of the chi-square test were not fulfilled. SPSS 28.0 software (IBM Corporation, Armonk, New York, United States) was used for the analysis. The statistical significance of the results was established as p < 0.05. In addition, a multivariate logistic regression analysis was performed to identify independent predictors of 30-day mortality. Variables with a p-value less than 0.2 in the univariate analysis were included in the multivariate model. Odds ratios (OR) with 95 % confidence intervals (CI) were calculated. Model fit was evaluated using the Hosmer-Lemeshow test, chi-square, Nagelkerke R^2 , and Cox-Snell R^2 values to ensure robustness.

3. Results

After excluding 18 patients with traumatic SAH, 4 patients with a diagnosis other than SAH, 12 patients transferred from another hospital, 7 patients with an mRS score >2 before SAH, 1 patient with uncalculable GPR, 12 patients with diabetes mellitus, and 11 patients with acute or chronic renal failure, the study was completed with 134 patients.

The mean age of the patients was 65.9 ± 16.7 , and 68 of them were female (50.7 %). Among the included patients, 64 (47.8 %) died within 30 days (Table 1). The patients were categorized into poor-good functional outcome groups and clinical and laboratory values were compared. The ages of the patients in the poor functional outcome group were significantly (p < 0.05) higher than those in the good functional outcome group. The Rankin scores of the poor functional outcome group were significantly (p < 0.05) higher than those of the good functional outcome group. The mean glucose and GPR levels in the poor functional outcome group were significantly (p = 0.003) higher than those in the good functional outcome group (Table 2).

Patients were categorized into survivor and non-survivor groups based on the 30-day mortality rate, and their clinical and laboratory values were compared. The ages of the patients in the non-survivor group were significantly (p < 0.05) higher than those in the survivor group. There was no significant (p > 0.05) difference in gender distribution between the survivor and non-survivor groups. The Rankin score of the non-survivor group was significantly (p < 0.05) higher than that of the survivor group. In the non-survivor group, glucose and GPR averages were significantly (p = 0.004, p = 0.023) higher than those in the survivor group (Table 3).

In the multivariate logistic regression analysis, the glucose/potassium ratio (p = 0.043, OR: 4.041, 95 % CI: 1.45–26.147) and the Rankin Scale score (p = 0.002, OR: 12.714, 95 % CI: 2.578–62.706) were identified as significant independent predictors of 30-day mortality in patients with aneurysmal subarachnoid hemorrhage (Table 4). Other variables, including age (p = 0.563), Hunt-Hess

Table 2

Comparison of laboratory and clinical data of patients according to functional outcome.

		Functional Outcome (Good)				F Functio	P value				
		Mean ± 5	SD/n-%		Median	Mean ±	Mean \pm SD/n-%		Median		
Age (years	s)	60.6	±	14.6	59.0	70.7	±	17.2	73.0	0.000	b
Gender	Woman Man	27 37	_	42.2 % 57.8 %		41 29	_	58.6 % 41.4 %		0.058	c
Sistolic blo	ood pressure (mmHg)	153.1	±	32.3	151.0	153.6	±	34.3	150.0	0.932	a
Diastolic b	blood pressure (mmHg)	85.1	\pm	21.0	80.0	86.7	±	23.9	82.5	0.619	b
Heart rate	e (bpm)	86.8	\pm	15.6	86.0	87.2	±	19.7	81.0	0.521	b
Hunt-Hess	s score	2.0	\pm	1.2	2.0	3.7	±	1.4	4.0	0.000	ь
Glasgow c	coma scale	14.0	\pm	2.5	15.0	9.5	±	4.3	9.0	0.000	ь
Rankin sca	ale	0.95	\pm	1.19	1.00	5.41	±	0.84	6.00	0.000	ь
White bloo	od cell ($ imes$ 10/µl)	11.6	\pm	4.2	11.0	11.4	±	5.5	10.5	0.378	ь
Hemoglob	oin (g/dl)	13.5	\pm	1.8	13.4	12.7	±	3.0	12.5	0.058	а
Platelet (x	:10/µl)	253.9	\pm	72.0	253.5	231.0	±	82.6	228.0	0.091	а
Glucose (n	nmol/L)	7.8	\pm	2.0	7.4	9.8	±	4.1	8.9	0.003	а
Potassium	(mmol/L)	4.0	\pm	0.5	4.0	4.2	±	0.6	4.1	0.040	а
Glucose/P	otassium raito	2.0	±	0.6	1.8	2.4	±	1.0	2.2	0.030	b
Treatment	t		_				_				-
Endovascu	ılar Coiling	4		19.0 %		2		9.5 %		0.378	с
Neurosurg	gical Clipping	17		81.0 %		19		90.5 %			

^a Independent sample *t*-test/

^b Mann-Whitney u test∕

^c Chi-Square test.

Table 3

Comparison of laboratory and clinical data of patients according to 30-day mortality.

		30-day Mortality (–)				30-day M	P value				
		Mean ± 3	SD/n-%		Median	Mean \pm SD/n-%		Medyan			
Age (years)		62.3	±	15.0	63.0	69.8	±	17.7	73.0	0.003	b
Gender	Woman Man	31 39	_	44.3 % 55.7 %		37 27	—	57.8 % 42.2 %		0.118	c
Sistolic blood pressure	(mmHg)	153.1	±	31.4	149.0	153.5	±	35.4	150.5	0.944	a
Diastolic blood pressure	e (mmHg)	85.5	±	20.5	81.5	86.4	±	24.7	83.0	0.862	b
Heart rate (bpm)		86.3	±	15.2	86.0	87.8	\pm	20.3	81.0	0.762	b
Hunt-Hess score		2.1	±	1.2	2.0	3.9	\pm	1.3	4.0	0.000	b
Glasgow coma scale		14.0	±	2.2	15.0	9.1	\pm	4.3	8.0	0.000	b
Rankin scale		1.21	±	1.42	1.00	5.55	\pm	0.75	6.00	0.000	b
White blood cell (\times 10)/µl)	11.2	\pm	4.2	10.7	11.8	±	5.6	10.8	0.784	b
Hemoglobin (g/dl)		13.4	±	1.8	13.3	12.6	\pm	3.1	12.5	0.071	а
Platelet (x10/µl)		248.2	±	72.3	249.0	235.0	\pm	84.4	234.5	0.332	а
Glucose (mmol/L)		7.9	±	2.0	7.4	9.9	\pm	4.2	8.7	0.004	b
Potassium (mmol/L)		4.0	±	0.5	4.0	4.2	\pm	0.6	4.1	0.223	а
Glucose/Potassium rait	0	2.0	±	0.6	1.8	2.4	±	1.1	2.2	0.023	b
Treatment			_								-
Endovascular Coiling		5		19.2 %		1		6.3 %		0.243	с
Neurosurgical Clipping		21		80.8 %		15		93.8 %			
Functional Outcome	Good	62		88.6 %		2		3.1 %		0.000	с
	Poor	8		11.4 %		62		96.9 %			

^a Independent sample *t*-test/

^b Mann-Whitney u test/

^c Chi-Square test.

score (p = 0.582), Glasgow Coma Scale (p = 0.984), hemoglobin (p = 0.178), and gender (p = 0.777), were not statistically significant. The Hosmer-Lemeshow goodness-of-fit test yielded a p-value of 0.567, indicating a good fit for the model. The chi-square value was 0.894, with Nagelkerke R^2 and Cox-Snell R^2 values of 0.911 and 0.683, respectively, indicating the model's strength in explaining the variance of the outcome.

4. Discussion

In this study, the relationship between GPR ratio, which is examined during admission to the ED, and mortality and functional outcomes of patients with aneurysmal SAH was investigated. It was concluded that high GPR is associated with mortality and poor functional outcome.

Despite the relatively decreased mortality rates of SAH patients due to the facilitation of access to healthcare services and developments in treatment methods, only half of the patients can survive. Additionally, a significant proportion of these surviving patients experience long-term disabilities. Therefore, early identification of patients with poor prognosis is crucial [13].

In EDs, the training of healthcare professionals should be supported by up-to-date protocols, and they should be skilled in using appropriate screening tools for critically ill patients.

Biomarkers can assist clinicians in making informed treatment decisions based on the severity of subarachnoid hemorrhage, and can help predict whether a patient is likely to have adverse outcomes, thereby improving their chances of successful recovery. According to research, GPR has been shown to be strongly associated with cerebral vasospasm and overall clinical results [12]. The relationship between GPR and poor prognosis has not been clearly explained. However, physiological changes occurring in the body during stress situations, such as SAH, may help explain this situation. Catecholamine release occurs in stress situations. This leads to increased heart rate, myocardial contraction, and blood pressure. The acceleration and increase in the heart direct blood flow to the

Table 4
Multivariate logistic regression analysis for 30-day mortality predictors in aneurysmal subarachnoid hem-
orrhage patients.

Variable	p-value	OR (95 % CI)
Age (years)	0.563	0.976 (0.900-1.059)
Hunt-Hess Score	0.582	1.333 (0.480-3.700)
Glasgow Coma Scale	0.984	0.996 (0.650-1.525)
Hemoglobin	0.178	0.797 (0.573-1.109)
Glucose/Potassium Ratio	0.043	4.041 (1.450-26.147)
Gender	0.777	1.333 (0.182–9.743)
Rankin Scale	0.002	12.714 (2.578–62.706)

skeletal system and cause increased cell metabolism throughout the body. Increased metabolism enhances glucose metabolism from the liver through glycogenolysis, gluconeogenesis, lipolysis, and ketogenesis. As a result, hyperglycemia occurs in the body. To further increase the resulting hyperglycemia, insulin release is reduced by stimulating alpha-adrenergic pancreatic receptors. On the other hand, the hypothalamic-pituitary-adrenal axis is activated due to the stress response, and as a result, the amount of corticotropin-releasing hormone increases. The increased corticotropin-releasing hormone is released through the hypothalamus and increases the release of adrenocorticotropic hormones in the systemic circulation. Aldosterone is a hormone that regulates extracellular volume and blood pressure. Its release binds to the mineralocorticoid receptors of the principal cells in the renal collecting tubules, which facilitate sodium reabsorption and potassium excretion. As a result, the body retains sodium and excretes potassium in an attempt to regulate blood pressure. Consequently, hyperglycemia and hypokalemia are expected due to the stress response. These mechanisms explain the increase in GPR [14].

The most important finding of this study is the association of increased GPR with mortality and poor functional outcomes in SAH patients. Similar results are present in the literature. In the study by Jung et al., it was emphasized that GPR could be used as an independent tool for predicting 3-month mortality in SAH patients [12]. In a study conducted in Japan, aneurysmal SAH patients were categorized into two groups, severe and non-severe, according to the Hunt-Kosnik Grade, and various characteristics were compared. The study concluded that GPR was higher in the severe SAH group [15]. There are also studies examining the prognostic roles of glucose and potassium, the two components of GPR, in SAH patients. These two components have advantages such as being quickly accessible, cost-effective and giving rational results. In the study by Beşoğlu et al., it was reported that hyperglycemia developing after aneurysmal SAH patients, it was reported that 69 of these patients (34.9 %) had hypokalemia [17]. In a study conducted in Iran, the relationship between serum electrolytes and SAH was investigated, and it was emphasized that hypokalemia was associated with poor outcomes [18]. In line with our findings, a study by Wang et al. also demonstrated that elevated GPR levels are associated with poor outcomes in patients with SAH. Specifically, their study found that higher GPR levels were significantly correlated with increased risk of preoperative rebleeding and poor outcomes at 90 days post-aSAH [19].

The significant association between a high GPR and poor outcomes in SAH patients can be understood through the physiological changes that occur during the body's stress response. In stressful situations, such as SAH, there is an increase in catecholamine release, which elevates glucose levels by promoting glycogenolysis and gluconeogenesis. Concurrently, aldosterone release increases potassium excretion through the kidneys, leading to hypokalemia. The resulting increase in the GPR reflects the intensity of the stress response and may correlate with the severity of the patient's condition, thereby serving as a predictor of mortality and poor functional outcomes.

Considering the promising results of our study, GPR has the potential to be integrated into clinical practice as a simple and costeffective prognostic marker. However, before it can be widely adopted, further validation is necessary through prospective, multicenter studies. These studies would help to confirm the robustness of GPR as a predictor of outcomes in SAH patients and address the limitations of the current study, such as its retrospective design and single-center setting. Additionally, the clinical utility of GPR should be evaluated in conjunction with other established prognostic markers to determine its relative importance and feasibility in everyday clinical use. If further studies confirm the prognostic significance of the glucose/potassium ratio, therapeutic strategies aimed at maintaining optimal glucose and potassium levels in SAH patients could be developed. Early interventions to stabilize these biomarkers might reduce the risk of poor outcomes, particularly in high-risk patients. For example, glucose management protocols and potassium supplementation could be tailored to individual patient needs, based on their GPR at admission.

There are some limitations to this study. This study's retrospective and single-center design may introduce potential biases. However, uniform data collection criteria were applied across all patients, and statistical methods were utilized to control for confounding variables. Despite these efforts, the generalizability of the findings may be limited, and further multi-center prospective studies are recommended to confirm these results. In addition, we did not separately investigate hormone levels, such as cortisol or glucagon, which could affect serum glucose levels, or the use of medications like beta-blockers or diuretics that could affect potassium levels. Lastly, we only examined the GPR measurement results during the initial admission to the ED, without looking at serial GPR measurements, which could have provided prognostic contributions.

5. Conclusion

In this study, we found that the GPR is an independent predictor of short-term mortality in patients with aneurysmal subarachnoid hemorrhage. Alongside functional status, GPR emerged as a key factor in identifying patients at higher risk. These findings suggest that GPR could be integrated into clinical practice to enhance risk stratification and improve patient outcomes. Future studies are necessary to validate these results and establish GPR's role in clinical decision-making.

Data availability statement

The data associated with this study has not been deposited into a publicly available repository. The data underlying this article will be shared on reasonable request to the corresponding author.

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CRediT authorship contribution statement

Halil Alışkan: Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Software, Resources, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation. Mazlum Kılıç: Writing – review & editing, Writing – original draft, Methodology, Formal analysis, Data curation, Conceptualization. Rohat Ak: Writing – review & editing, Writing – original draft, Methodology, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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