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Signa Vitae

ORIGINAL RESEARCH

Prognostic value of glucose-to-potassium ratio and other biomarkers in in-hospital cardiac arrest

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Abstract

Background: This study aimed to evaluate the prognostic value of the serum glucose/potassium ratio (GPR) for sustained return of spontaneous circulation (ROSC) and 30-day mortality in patients with in-hospital cardiac arrest (IHCA). **Methods**: Patients aged 18 years or older who underwent cardiopulmonary resuscitation (CPR) for cardiac arrest in the emergency department (ED) were included. Routine laboratory parameters were obtained from the first blood sample collected during CPR in the ED. The neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR) were calculated by dividing the absolute number of neutrophils and platelets, respectively, by the absolute number of lymphocytes. The GPR was calculated as serum glucose divided by potassium levels. **Results**: The 30-day mortality rate was 82.2% (n = 291). Multivariate logistic regression analysis identified lactate, NLR, PLR and GPR as independent predictors of mortality. Among these markers, lactate exhibited the highest predictive power for mortality, with an area under the curve (AUC) of 0.817, compared to NLR (AUC: 0.676), PLR (AUC: 0.679) and GPR (AUC: 0.688). The optimal cut-off values for predicting mortality were 7.83 for lactate (sensitivity: 75.3%, specificity: 89.4%), 1.68 for NLR (sensitivity: 78.8%, specificity: 71.7%), 199.26 for PLR (sensitivity: 76.4%, specificity: 92.3%) and 57.81 for GPR (sensitivity: 71.8%, specificity: 84.1%). **Conclusions**: Our findings suggest that GPR is a promising prognostic marker for predicting mortality in patients with IHCA.

Keywords

In-hospital cardiac arrest; Mortality; Glucose/potassium ratio; Emergency department

1. Introduction

In the United States, approximately 292,000 adults experience in-hospital cardiac arrest (IHCA) annually [1–3]. Globally, IHCA affects 1.5–2.8 individuals per 1000 hospitalizations in Europe and 6–7 individuals per 1000 worldwide, highlighting its significant health burden [3, 4]. Despite advancements in treatment modalities, the incidence of IHCA continues to rise, while the associated mortality rate remains alarmingly high [4, 5]. Although treatment options have improved in recent years, the proportion of patients achieving return of spontaneous circulation (ROSC) after IHCA remains low, which contributes to IHCA being a leading cause of death and disability worldwide [2–7].

The prognosis of patients who achieve ROSC following cardiac arrest is often influenced by ischemic reperfusion injury, which triggers the release of numerous cytokines and biomarkers [4, 7]. Thus, accurate and timely prediction of outcomes in patients with IHCA is essential for improving ROSC rates and optimizing clinical interventions [2, 8]. Cytokines and other biomarkers, along with their associated inflammatory pathways, are increasingly being studied for their potential to predict ROSC and assist in clinical decision-making.

Among the laboratory parameters routinely assessed in critical conditions such as acute myocardial infarction, sepsis, trauma, and hemorrhagic shock, changes in serum glucose and potassium levels have been widely reported [6, 7]. The serum glucose/potassium ratio (GPR), calculated as the serum glucose level divided by the serum potassium level, is a simple and cost-effective parameter. During cardiac arrest, the heightened stress response leads to increased catecholamine release, causing elevated serum glucose levels and reduced serum potassium levels [8, 9]. GPR has been shown to predict morbidity and mortality in various conditions, including pulmonary embolism, subarachnoid hemorrhage, traumatic brain injury, and other traumas [9–12]. Notably, studies suggest that while hyperglycemia alone is associated with increased mortality in critical illnesses, GPR offers greater predictive accuracy than serum glucose or potassium levels measured individually [8, 11].

Given the intense release of cytokines and catecholamines during cardiac arrest, GPR may serve as a valuable prognostic marker in IHCA patients. In this study, we aimed to investigate the utility of GPR as a predictor of ROSC and as a prognostic marker in patients with IHCA.

2. Materials and methods

2.1 Study design and participants

This retrospective study was conducted between 01 January 2018, and 31 December 2024 at Konya City Hospital, a tertiary care facility serving an average of 50,000 patients per month, and comprised of patients who experienced cardiac arrest and underwent cardiopulmonary resuscitation (CPR) in the emergency department (ED). Ethical approval for the study was obtained from the Faculty of Medicine of Karatay University ethics committee (approval no: 2023-06/004).

Patients were eligible for inclusion if they were 18 years of age or older and experienced cardiac arrest during their treatment in the hospital. Exclusion criteria included inaccessible records, out-of-hospital cardiac arrest, administration of epinephrine before routine blood samples were collected, or unclear clinical information and medication history. Additional exclusions were patients with diabetes mellitus, hyperthyroidism, hypothyroidism, renal failure, severe malnutrition, extensive burns, liver cirrhosis, those with hemolyzed blood samples, pregnancy, or those younger than 18 years of age. Patients taking antihypertensive or potassium-regulating medications were also excluded.

2.2 Data collection

All patients received in-hospital basic and advanced life support, as well as post-CPR care, in accordance with the American Heart Association (AHA) guidelines [13]. Patients who achieved ROSC were admitted to either the ED's red zone or the intensive care unit, where they were managed using standard treatment protocols. These protocols included hemodynamic support, invasive monitoring, mechanical ventilation, sedation, analgesia and intensive care support.

Demographic data, clinical characteristics, and laboratory findings were retrieved from the hospital's electronic medical database, including variables such as age, gender, resuscitation time, rhythm type at diagnosis, comorbidities, duration of CPR, and ROSC status. Based on 30-day outcomes, patients were classified into two groups: those who survived (alive) and those who did not (ex).

2.3 Laboratory analysis

Routine laboratory tests were conducted on the initial blood sample collected during CPR in the ED, with hematological parameters analyzed using an automatic analyzer (Mindray BC-6000, Shenzhen, Guangdong, China). The neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR) were calculated by dividing the absolute counts of neutrophils or platelets by the absolute count of lymphocytes, respectively. GPR was determined by dividing the serum glucose level by the serum potassium level.

2.4 Statistical analysis

Statistical analysis was performed using IBM SPSS version 27.0 (IBM Corp., Armonk, NY, USA). The study variables were summarized using frequency, percentage, mean, standard deviation, median, minimum, maximum and interquartile range (IQR). Qualitative data comparisons were made using the Chi-Square (χ^2) test, while data distribution was evaluated using the Kolmogorov-Smirnov test, along with skewness, kurtosis, and graphical methods such as histograms, Q-Q plots, stem-and-leaf plots and boxplots. For data following a normal distribution, the Independent Samples *t*-test was used for group comparisons, whereas the Mann-Whitney U test was applied to non-normally distributed data. Logistic regression analysis was employed to determine risk ratios for variables, and receiver operating characteristic (ROC) analysis was conducted to calculate sensitivity, specificity and optimal cut-off values. The threshold for statistical significance was set at $\alpha = 0.05$.

3. Results

This study included 354 patients who met the inclusion criteria, as outlined in the study diagram (Fig. 1). The mean age of the patients was 67.5 $\pm$ 15.2 years, with 43.2% (n = 153) being female. The mean duration of CPR was 26 $\pm$ 9 minutes. Demographic, clinical, and laboratory characteristics of the patients are presented in Table 1. The overall 30-day mortality rate was 82.2% (n = 291).

The rate of spontaneous respiration following CPR was significantly higher in the survivor group compared to the deceased group ($p < 0.05$). Additionally, survivors demonstrated higher values of WBC, neutrophils, potassium and NLR, while lower values of lymphocytes, glucose, lactate, PLR and GPR were observed in the same group ($p < 0.05$). No statistically significant differences were observed between the groups for other variables ($p > 0.05$).

Statistically significant parameters for mortality in CPR were initially analyzed using a univariate logistic regression test. Among these, variables considered to be more clinically significant and showing high correlations (WBC, lactate, NLR, PLR and GPR) were subjected to further analysis. Statistically significant variables identified in the univariate analysis (lactate, NLR, PLR and GPR) were subsequently analyzed using a multivariate logistic regression test. The multivariate logistic regression analysis confirmed that lactate, NLR, PLR and GPR were independent predictors of mortality (Table 2).

The risk of mortality was found to be approximately 2.3 times higher in patients with elevated lactate levels compared to those with lower levels. Similarly, the risk was about 1.59 times higher in individuals with low NLR values, 1.01 times higher in those with elevated PLR values, and 1.03 times higher in patients with increased GPR values. To further evaluate the predictive power of these parameters for mortality, ROC analysis was conducted (Fig. 2). Among the predictors, lactate demonstrated the highest ability to predict mortality, with an area under the curve (AUC) of 0.817, compared to NLR (AUC: 0.676), PLR (AUC: 0.679) and GPR (AUC: 0.688) (Table 3). The optimal cut-off values for predicting mortality were determined as follows: lactate, 7.83 (sensi-

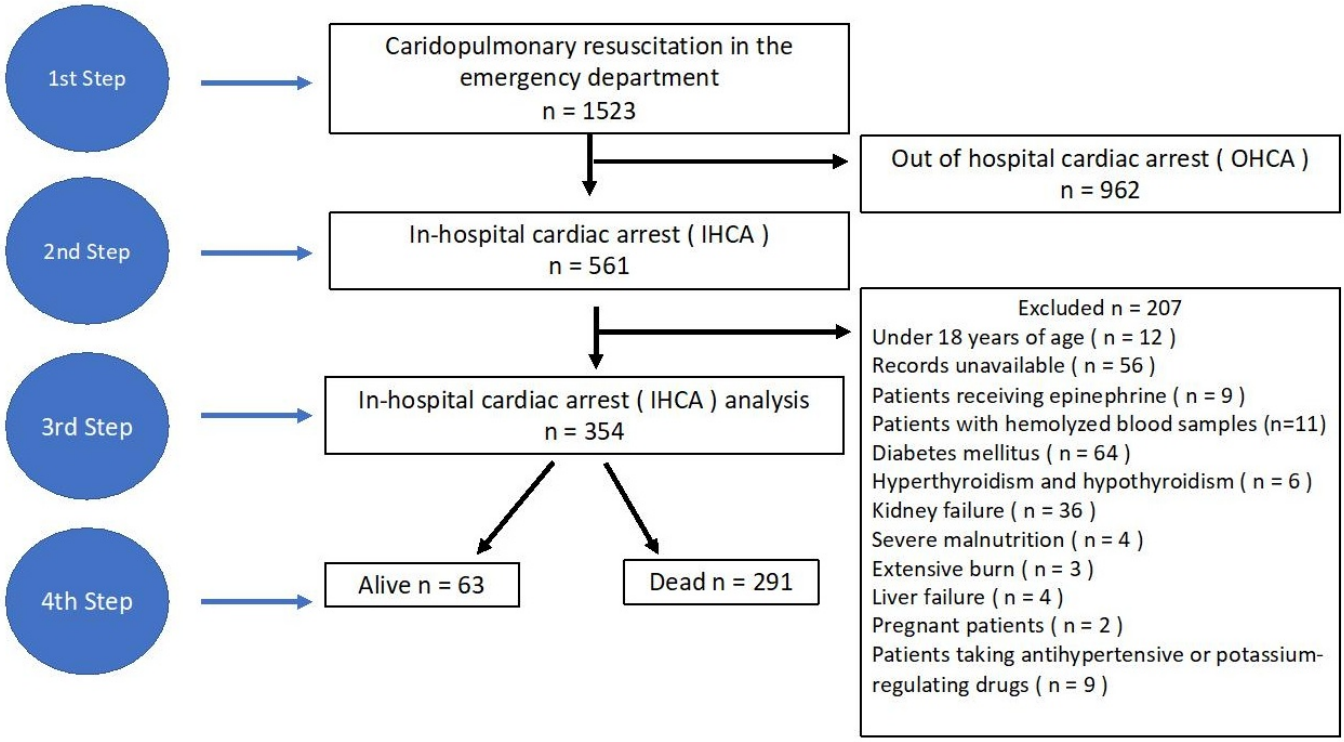

FIGURE 1. Flowchart of patients in-hospital cardiac arrest.

TABLE 1. Comparison of demographic, clinical characteristics and laboratory parameters of patients with cardiac arrest and mortality-related factors.

		Mortality		*p*
	General (n = 354)	Alive (n = 63)	Dead (n = 291)	
Sex				
Female	153 (43.2%)	22 (34.9%)	131 (45.0%)	0.142[a]
Male	201 (56.8%)	41 (65.1%)	160 (55.0%)	
Age (yr)	67.5 ± 15.2	64.6 ± 16.7	68.1 ± 14.9	0.134[b]
Comorbidity				
No	187 (52.8%)	38 (60.3%)	149 (51.2%)	0.189[a]
Yes	167 (47.2%)	25 (39.7%)	142 (48.8%)	
HT	78 (22.0%)	10 (15.9%)	68 (23.4%)	0.257[a]
CAD	67 (18.9%)	12 (19.0%)	55 (18.9%)	1.000[a]
COLD	20 (5.6%)	4 (6.3%)	16 (5.5%)	0.765[a]
Other	45 (12.7%)	5 (7.9%)	40 (13.7%)	0.295[a]
Shockable rhythm	85 (24.0%)	35 (55.5%)	50 (18.9%)	**<0.001**
CPR time (min)	26 ± 9	17 ± 6	31 ± 10	**<0.001**
Spontaneous Respiration After CPR				
No	289 (81.6%)	3 (4.8%)	286 (98.3%)	**<0.001**[a]
Yes	65 (18.4%)	60 (95.2%)	5 (1.7%)	
WBC, $\times 10^9$/L	13.6 ± 5.8	15.3 ± 7.0	13.2 ± 5.4	**0.033**[b]
Neutrophil, $\times 10^9$/L	7.1 ± 4.1	9.3 ± 6.1	6.6 ± 3.3	**0.001**[b]
Lymphocyte, $\times 10^9$/L	5.1 ± 2.1	4.5 ± 1.9	5.2 ± 2.1	**0.008**[b]
Hemoglobin, g/dL	11.9 ± 3.0	12.4 ± 3.7	11.8 ± 2.9	0.269[b]
Glucose, mg/dL	247.4 ± 121.0	206.0 ± 71.7	256.4 ± 127.5	**<0.001**[b]
Sodium, mmol/L	139.9 ± 12.2	138.5 ± 6.5	140.2 ± 13.1	0.317[b]

TABLE 1. Continued.

		Mortality		*p*
	General (n = 354)	Alive (n = 63)	Dead (n = 291)	
Potassium, mmol/L	4.4 ± 1.2	5.1 ± 0.8	4.3 ± 1.2	<**0.001**[b]
BUN, mg/dL	25.0 (16.0–46.0)	25.0 (18.0–45.0)	24.0 (16.0–46.0)	0.787[c]
Creatinine, mg/dL	1.0 (0.8–1.5)	1.1 (0.9–1.6)	1.0 (0.7–1.5)	0.928[c]
Lactate, mmol/L	7.22 ± 2.03	5.22 ± 1.53	7.64 ± 1.83	<**0.001**[b]
NLR	1.70 ± 1.21	2.32 ± 1.42	1.52 ± 1.13	<**0.001**[b]
PLR	159.71 ± 60.12	131.33 ± 42.64	165.83 ± 61.62	<**0.001**[b]
GPR	60.93 ± 35.22	41.72 ± 16.52	65.05 ± 36.81	<**0.001**[b]

HT: Hypertension; CAD: coronary artery disease; COLD: chronic obstructive lung disease; CPR: cardiopulmonary resuscitation; WBC: White blood count; BUN: blood urea nitrogen; NLR: neutrophil lymphocyte ratio; PLR: platelet lymphocyte ratio; GLR: glucose potassium ratio. Bold when p value < 0.05. [a]: Chi-Square Test (n/%), [b]: Independent Samples t Test (Mean ± SD), [c]: Mann-Whitney U Test (Median (Q1–Q3)).

TABLE 2. Univariate and multivariate analysis of predictive factors for mortality.

	Univariate Logistic Regression Analysis						Multivariate Logistic Regression Analysis					
Risk Factor	*β*	SE	Wald	Odds	95% CI	*p*[a]	B	SE	Wald	Odds	95% CI	*p*[b]
WBC	0.058	0.023	6.389	1.06	1.01–1.11	**0.011**	–	–	–	–	–	–
Lactate	0.820	0.114	52.206	2.27	1.82–2.84	<**0.001**	0.827	0.124	44.293	2.29	1.79–2.92	<**0.001**
NLR	0.465	0.105	19.677	1.59	1.30–1.95	<**0.001**	0.369	0.144	6.623	1.45	1.09–1.92	**0.010**
PLR	0.013	0.003	19.283	1.01	1.01–1.02	<**0.001**	0.013	0.004	12.162	1.01	1.01–1.02	<**0.001**
GPR	0.029	0.007	19.686	1.03	1.02–1.04	<**0.001**	0.021	0.008	6.513	1.02	1.00–1.04	**0.011**

WBC: White blood cell; NLR: neutrophil-lymphocyte ratio; PLR: platelet-lymphocyte ratio; GPR: glucose/potassium ratio; B: β coefficient; SE: Standard Error; CI: confidence interval. [a]: Univariate Logistic Regression Test, [b]: Multivariate Logistic Regression Test (Nagelkerke R^2 = 0.534, Hosmer and Lemeshow Test = 0.206). Bold when p value < 0.05.

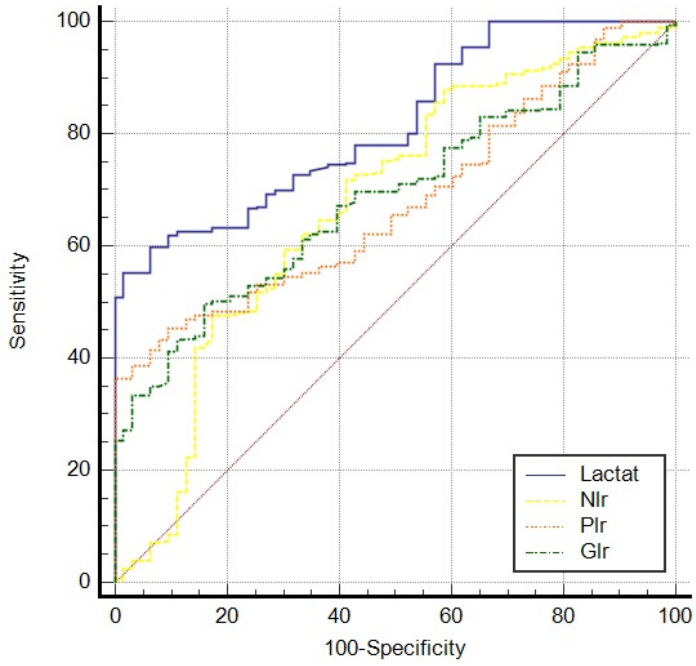

FIGURE 2. Receiver-operating characteristic curves of lactate, NLR, PLR and GPR for mortality. NLR: neutrophil-lymphocyte ratio; PLR: platelet-lymphocyte ratio; GPR: glucose/potassium ratio.

TABLE 3. Analysis of the area under the ROC curve for mortality in IHCA.

	AUC	95% CI	Cut Off	Sensitivity	Specificity	Youden index	PPV	NPV	*p*
Lactate	0.817	0.773–0.856	>7.83	75.3	89.4	0.647	89.4	72.3	<**0.001**
NLR	0.676	0.625–0.725	≤1.68	78.8	71.7	0.506	88.9	71.1	<**0.001**
PLR	0.679	0.628–0.728	>199.26	76.4	92.3	0.687	92.0	75.4	<**0.001**
GPR	0.688	0.637–0.736	>57.81	71.8	84.1	0.56	93.5	66.6	<**0.001**

ROC: receiver operating characteristic; AUC: area under the curve; CI: confidence interval; PPV: positive predictive value; NPV: negative predictive value; NLR: neutrophil-lymphocyte ratio; PLR: platelet-lymphocyte ratio; GPR: glucose/potassium ratio. Bold when p value < 0.05.

tivity: 75.3%, specificity: 89.4%); NLR, 1.68 (sensitivity: 78.8%, specificity: 71.7%); PLR, 199.26 (sensitivity: 76.4%, specificity: 92.3%); and GPR, 57.81 (sensitivity: 71.8%, specificity: 84.1%).

4. Discussion

IHCA represents a significant global health challenge [3–6]. In the United States, annual IHCA cases range between 370,000 and 750,000, and the incidence in Europe continues to rise each year [14]. The likelihood of ROSC in IHCA patients varies between 36% and 54%, while 30-day survival rates range from 15% to 34% [4, 5]. Numerous studies have investigated laboratory parameters and neuroimaging methods to predict survival in ROSC patients [2, 6]. Despite these efforts, survival rates have not significantly improved over the past 30 years. The incorporation of simpler, cost-effective, and easily measurable new markers, alongside existing risk models, could enhance prognosis prediction and aid in treatment management [6]. In IHCA, the body's stress response is activated, often leading to alterations in glucose and potassium levels [6, 15]. Therefore, in this retrospective study, we explored whether GPR could serve as a potential marker for predicting mortality in IHCA patients. To our knowledge, this is the first study to assess the prognostic value of GPR in ROSC. Our findings demonstrated that lactate, NLR, PLR and GPR are significant prognostic indicators of mortality in ROSC patients. These results suggest that GPR may be a valuable addition to existing prognostic models for IHCA.

GPR may be associated with several pathophysiological mechanisms, including inflammation, endothelial dysfunction and oxidative stress [9]. Inflammation plays a significant role in the pathophysiology of IHCA by promoting the release of pro-inflammatory cytokines and initiating a systemic inflammatory response [9, 10]. Endothelial dysfunction, in turn, disrupts vascular tone and increases vascular permeability, leading to impaired tissue perfusion and oxygenation in IHCA patients [11]. Oxidative stress further exacerbates the condition by causing cell damage and organ dysfunction through excessive free radical production and a weakened antioxidant defense system [12]. Despite these knowledge, further studies are needed to explore the relationship between GPR and these mechanisms, as well as their collective impact on mortality in IHCA patients. Moreover, GPR can reflect the body's stress response, which is activated in IHCA due to hypoxia and ischemia, causing changes in glucose and potassium levels [6, 15]. Elevated glucose levels may result from increased insulin resistance and gluconeogenesis [16–18], while high potassium levels may indicate cell damage and tissue destruction [19]. Consequently, an elevated GPR may indicate a heightened stress response and a worse prognosis in IHCA patients. Similarly, our study found that a GPR value above 57.81 predicted mortality with a sensitivity of 71.8% and a specificity of 84.1%.

Lactate is recognized as an indicator of tissue hypoperfusion and anaerobic metabolism and is commonly used to predict mortality in patients with OHCA [1, 19]. It is also a well-established prognostic marker associated with increased mortality risk in patients with IHCA [1, 20, 21]. In our study, lactate levels were significantly higher in the mortality group compared to the survivor group (7.64 ± 1.83 *vs.* 5.22 ± 1.53) and demonstrated the highest predictive power among the evaluated parameters, with an AUC of 0.817 in the ROC analysis. Furthermore, in the multivariate analysis, lactate showed an independent predictive power (Odds Ratio (OR): 2.27, 95% CI (Confidence Interval): 1.82–2.84), which was greater than that of GPR. These findings align with previous studies, confirming the role of lactate as a crucial marker in assessing prognosis in patients with IHCA.

The prognostic value of biomarkers associated with the inflammatory response following IHCA is particularly significant. As demonstrated in the study by Woodhouse *et al.* [22], the systemic inflammatory response triggered after cardiac arrest is closely linked to catecholaminergic activation, resulting in notable alterations in neutrophil, lymphocyte and platelet counts [23]. Patel *et al.* [24] reported that the risk of death in IHCA patients increased when the NLR cut-off value exceeded 4.5. Similarly, Huang *et al.* [25] identified NLR and PLR as prognostic biomarkers in IHCA patients who achieved ROSC. In our study, NLR values were significantly lower (OR: 1.59 ± 1.13), and PLR values were higher (165.83 ± 61.62) in the mortality group, highlighting the association between the severity of the systemic inflammatory response and mortality. When the performance of these inflammatory markers was evaluated using ROC analysis, the cut-off value for NLR was determined to be 1.68, with a sensitivity of 78.8% and specificity of 71.7%. The cut-off value for PLR was 199.26, with a sensitivity of 76.4% and specificity of 92.3%. These findings support the prognostic significance of inflammatory markers in assessing mortality risk in IHCA patients.

Our study has several strengths, most notably being the first

to identify GPR as a potential marker for predicting mortality in IHCA patients. Additionally, the inclusion of a large sample size strengthens the reliability of our findings. However, some limitations should be acknowledged. Firstly, the retrospective design of the study limits our ability to establish causal relationships. Secondly, as the study was conducted at a single center, the generalizability of the findings to broader populations may be restricted. Thirdly, the lack of direct measurements of stress-response parameters, such as catecholamine levels, hindered a more comprehensive evaluation of the ability of GPR to reflect catecholaminergic activation. Moreover, while it is unclear whether the use of inhaled corticosteroids (ICS) induces hyperglycemia, the potential influence of ICS on glucose levels may have introduced bias into the data. Finally, the study provided only a limited comparison of GPR with other biomarkers for predicting mortality, which constrained a more thorough assessment of its prognostic value.

5. Conclusions

In conclusion, this study indicates that GPR is a potential marker for predicting mortality in patients with IHCA. As an easily measurable and widely accessible parameter, GPR holds promise for use in the risk assessment of IHCA patients and in guiding treatment strategies. However, prospective studies are required to validate these findings and to establish the clinical utility of GPR in routine practice.

AVAILABILITY OF DATA AND MATERIALS

Data used in the study will be made available by the author upon reasonable request.

AUTHOR CONTRIBUTIONS

EFV and HM—conceived and designed the study. HM, OLD, FCM and EK—undertook data acquisition, carried out data analysis and data interpretation. HM, RY, BB and MG—provided statistical expertise. HM, MA, FCM and EFV—wrote the manuscript draft. HM, BB, RY, EFV and OLD—undertook critical revision of the manuscript for important intellectual content. All authors participated and contributed to the critical revision of the manuscript and gave final approval of version submitted for publication.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

Our study was conducted in accordance with the Declaration of Helsinki and was approved by the Ethics Committee of the Faculty of Medicine of Karatay University (approval number: 2023-06/004). Due to the retrospective nature of the study, the requirement for informed consent by the above-mentioned institution/IRB was waived.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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