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RESEARCH

A new index for predicting epileptic seizures: glucose potassium ratio

Epileptik nöbetlerin öngörülmesinde yeni bir indeks: glukoz potasyum oranı

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Abstract

Purpose: This study aimed to evaluate the glucose to potassium ratio (GPR) as a potential biomarker for diagnosing active epileptic seizures in patients presenting to the emergency department.

Materials and Methods: This study was conducted at a tertiary university hospital's emergency department from January 1, 2022, to December 31, 2022. This study included patients presenting to the emergency department with generalized motor epileptic seizure activity and witnessed seizure activity (observed by health personnel) (Group A), and adult patients with a diagnosis of epilepsy visiting the neurology outpatient clinic for routine check-ups (Group B).

Results: A total of 119 and 118 patients in groups A and B, respectively, were included. Significant differences were observed in white blood cell counts, glucose, potassium, and GPR levels between the groups. Receiver Operating Characteristic (ROC) curve analysis indicated that a GPR cut-off value of 24.8 had a sensitivity of 71.4% and specificity of 66.9% [Area Under the Curve (AUC)= 0.758]. Multivariate logistic regression revealed that GPR was an independent predictor of epileptic seizure activity [Odds ratio (OR)=0.873, 95% confidence intervarl (CI) 0.831–0.917).

Conclusion: GPR is a promising independent biomarker for predicting active epileptic seizure activity, and is significantly associated with increased seizure episodes. This study underscores the potential of GPR as a simple, effective diagnostic tool, especially in emergency settings where advanced diagnostic methods may be unavailable. Further prospective studies are recommended to validate these findings and to assess their clinical implications.

Keywords:. Epilepsy; glucose; potassium; seizures

Öz

Amaç: Bu çalışma, acil servise başvuran hastalarda aktif epileptik nöbetleri teşhis etmek için potansiyel bir biyobelirteç olarak glikoz-potasyum oranını (GPR) değerlendirmeyi amaçlamıştır.

Gereç ve Yöntem: Bu çalışma, 1 Ocak 2022-31 Aralık 2022 tarihleri arasında üçüncü basamak üniversite hastanesinin acil servisinde yürütülmüştür. Bu çalışma, acil servise jeneralize tonik-klonik epileptik nöbet aktivitesi ile başvuran ve nöbet aktivitesine tanık olan (sağlık personeli tarafından gözlemlenen) hastaları (A Grubu) ve nöroloji polikliniğine rutin kontroller için başvuran epilepsi tanılı erişkin hastaları (B Grubu) içermiştir.

Bulgular: Sırasıyla A ve B gruplarında toplam 119 ve 118 hasta dahil edilmiştir. Gruplar arasında nötrofil, glikoz, potasyum ve GPR seviyelerinde anlamlı farklılıklar gözlenmiştir. Receiver Operating Characteristic (ROC) eğrisi analizi, 24.8'lik bir GPR kesme değerinin %71.4 duyarlılık ve %66.9 özgüllüğe sahip olduğunu [Area Under the Curve (AUC)= 0.758] göstermiştir. Çok değişkenli lojistik regresyon, GPR'nin epileptik nöbet aktivitesinin bağımsız bir belirleyicisi olduğunu ortaya koymuştur [Olabilirlik oranı (OR)=0.873, %95 güven aralığı 0.831–0.917,].

Sonuç: GPR, aktif epileptik nöbet aktivitesini tahmin etmek için umut vadeden bağımsız bir biyobelirteçtir ve artmış nöbet atakları ile anlamlı bir şekilde ilişkilidir. Bu çalışma, özellikle gelişmiş tanı yöntemlerinin mevcut olmadığı acil durumlarda, GPR'nin basit ve etkili bir tanı aracı olarak potansiyelini vurgulamaktadır. Bu bulguları doğrulamak ve klinik etkilerini değerlendirmek için daha fazla prospektif çalışma önerilmektedir.

Anahtar kelimeler: Epilepsi; glikoz; nöbet; potasyum

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INTRODUCTION

Epileptic seizure is a manifestation of temporary signs and/or symptoms due to excessive, abnormal, or synchronous neuronal activity in the brain[1]. Seizures are commonly encountered, affecting 8-10% of the population over their lifetime[2]. Epileptic seizures represent a serious emergency condition that significantly affects the quality of life of affected individuals, and their diagnosis is of substantial importance in both emergency and specialized clinical settings[3]. The frequency of epileptic seizures increases with age, making diagnosis in adult populations particularly challenging in emergency departments[4]. Despite rapid advancements in both clinical and preclinical epilepsy research, the pathogenesis of epilepsy remains unclear.

Numerous clinical studies have demonstrated a correlation between plasma cortisol level and neurological disorders. Cortisol is a primary corticosteroid secreted by the adrenal cortex and is known for its proconvulsant and epileptogenic effects[5]. Additionally, cortisol plays a significant role in glucose homeostasis, especially in counter-regulatory mechanisms aimed at preventing hypoglycemia during biological stress periods[6]. The clinical presentation of epileptic seizures involves a combination of sympathetic activation, vagal activation, and sympathetic-vagal suppression mechanisms[7]. Sympathetic discharge, a part of this mechanism, activates the sodium-potassium pump, leading to the uptake of extracellular potassium into cells, causing hypokalemia[8].

In patients presenting to the emergency department with altered consciousness, seizure activity is a crucial consideration in differential diagnosis. Current diagnostic approaches for epileptic seizure management primarily focus on developing new and effective technologies, and often concentrate on the advancement of device-dependent electroencephalograms (EEG) and imaging methods[3,9].

Despite the witnessed episodes, a practical and accessible laboratory test to definitively ascertain the occurrence of a true seizure remains lacking in clinical practice. Considering the role of cortisol in the pathophysiology of epilepsy and its influence on serum glucose and potassium levels, this study aimed to evaluate the glucose to potassium ratio (GPR) in patients presenting with epileptic seizures to the emergency department as a potential simple, cost-effective, and readily available biomarker to assist in the early and accurate diagnosis of epileptic seizures.

The hypothesis of this study is that patients presenting with epileptic seizures will have a significantly elevated glucose to potassium ratio compared to non-seizure patients, reflecting underlying neuroendocrine and metabolic alterations associated with seizure activity.

MATERIALS AND METHODS

Study design and settings

This research was designed as a retrospective cross-sectional study. After obtaining ethical approval from the Ordu University Clinical Research Ethics Committee (Decision Number: 2023/48, Date: 03.02.2023), the study was conducted at Ordu University Training and Research Hospital, a tertiary healthcare institution. The hospital operates an integrated digital medical record system that ensures data reliability, completeness, and traceability for retrospective studies.

The study included two groups of patients between January 1, 2022, and December 31, 2022:

Group A consisted of adult patients presenting to the adult emergency department with generalized motor epileptic seizure activity witnessed by healthcare professionals, including emergency physicians and nurses. Seizure activity was confirmed either by direct observation, video documentation by an emergency medicine specialist, or through neurology consultation.

Group B included adult patients with a confirmed diagnosis of epilepsy based on EEG recordings, who had a history of generalized motor seizures but had experienced no seizure episodes in the past month and were attending the neurology outpatient clinic for routine follow-up.

All evaluations, observations, and clinical decisions in the emergency department were performed by board-certified emergency medicine specialists. Similarly, outpatient assessments were conducted by neurologists

Sample

Based on the study data, patients were included in the study if they met one of the following criteria:

(1) Adults over 18 years of age presenting to the emergency department with generalized motor epileptic seizure activity witnessed by healthcare professionals, with seizure activity confirmed by direct observation, video recording, or verified by an emergency medicine specialist or a neurologist;

(2) Adults over 18 years of age with a prior history of generalized motor seizures, who had not experienced a seizure in the past month, and had a confirmed diagnosis of epilepsy based on EEG findings.

Patients under 18 years of age, those with known diseases causing blood glucose metabolism disorders [diabetes mellitus (DM) and other endocrine metabolism diseases], blood potassium metabolism disorders [acute renal failure (ARF), chronic renal failure (CRF), gastrointestinal disorders, hypo- or hyperaldosteronism, nausea, vomiting, diarrhea], pregnant women, those receiving treatment or diagnosed with intracranial mass (ICM) or other intracranial pathologies [(ICM, ischemic/hemorrhagic cerebrovascular events (CVE)] on neuroimaging, patients who did not have witnessed seizures (not observed by any healthcare professional), and those with insufficient data in the record forms were excluded from the study.

A pilot study was conducted with 30 participants to estimate the sample size required for detecting a significant difference in GPR between patients with generalized motor seizures and a control group. Based on the pilot data, the effect size (Cohen's d) was calculated to be 0.67. Using this effect size, with a significance level (α) of 0.05 and a statistical power of 0.90 ($1-\beta = 0.90$), the minimum required sample size was determined using G*Power software. The analysis indicated that at least 37 patients were needed in the seizure group and 73 in the control group, totaling 110 participants.

Data collection

Demographic information (age, sex, chronic diseases requiring continuous medical treatment) and treatments applied to patients were obtained from patient record forms created and approved by our hospital and the Ministry of Health, and entered into a data collection form. All the procedures in this study adhered to the principles of the Declaration of Helsinki.

Routine diagnostic and therapeutic blood biochemistry tests performed at hospital admission were recorded, including serum alanine aminotransferase (ALT), aspartate aminotransferase (AST), blood urea nitrogen (BUN), creatinine, sodium, glucose, and potassium levels, as well as white blood cell (WBC), hemoglobin and platelet counts from complete blood counts. The GPR was calculated and entered into the data collection form.

Statistical analysis

All statistical analyses were completed using IBM Statistical Package for the Social Sciences Statistics for Windows version 23.0 software (IBM, Armonk, NY, USA). Correlations between categorical variables were performed using the Chi-square test. The Kolmogorov-Smirnov test was used to assess the normality of the data. Student's t-test was used for normally distributed data and the results are shown as mean±standard deviation. Mann-Whitney U test was used for non-normally distributed data and the results are shown as Median (25^{th} and 75^{th} percentile). Logistic regression analysis was performed using both univariate and multivariate models to assess the independent effect of GPR in predicting generalized motor seizure activity, and the corresponding odds ratios (ORs) were calculated. In the multivariate analysis (Model 2), age and sex were included as potential confounding variables, and the predictive value of GPR for generalized motor seizures was estimated. In Model 3, WBC count — which was found to be statistically significant in hypothesis testing (mann whitney u test) — was also added to the model in addition to age and sex. Although serum glucose and potassium levels were also significant in hypothesis testing (mann whitney u test), they were excluded from Model 3 to avoid multicollinearity, as they are direct components of the GPR calculation.

Receiving operating characteristics (ROC) curve analysis was performed to create area under the curve (AUC) scores for the diagnosis of active epileptic seizures by calculating GPR. This analysis was used to evaluate the optimal cut-off scores for maximizing sensitivity and specificity of the tools. AUC, sensitivity, specificity, positive likehood ratio (+LR), negative likehood ratio (-LR), positive predictive value (PPV), negative predictive value (NPV) and cut-off values were calculated. $p<0.05$ was determined as the level of statistical significant.

RESULTS

The study initially included 172 patients in Group A and 213 patients in Group B, totaling 385 patients.

After the examining patient data, 66 patients in Group A were excluded for meeting the exclusion criteria (9 with CRF, 17 previously diagnosed with DM, 18 with known or newly diagnosed ICM, 6 with ischemic CVE and 3 with hemorrhagic CVE). After reviewing the data of Group B patients, 95 were excluded (19 with a DM diagnosis, 13 with CRF, 24 with a previous diagnosis of CVE, 21 diagnosed with ICM, and 18 due to inaccessible blood test results). The flowchart of the patient and control groups included in our study is shown in Figure 1.

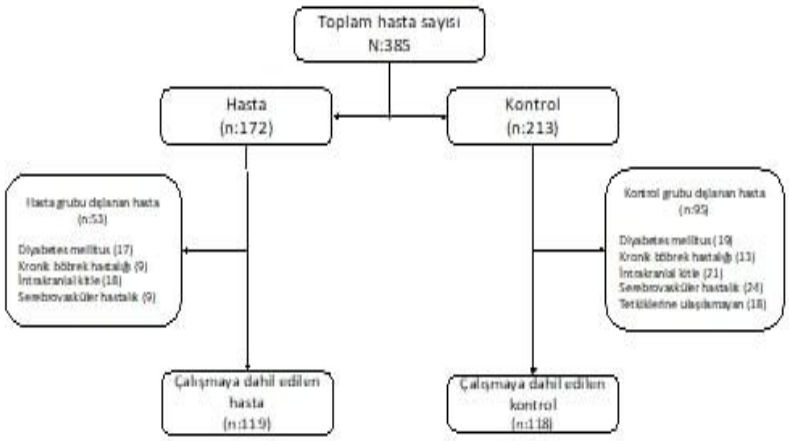

Figure 1. Flow chart for patients enrollment.

Table 1. Demographic and laboratory findings of the epilepsy groups

Variables	Epilepsy		*p*-value
	Group A (119)	Group B (118)	
Age (year)	49 (33-64)	48.5 (32.75-64.25)	0.767[α]
Gender (F/M)	51/68	53/65	0.750*
ALT (U/L)	15 (10-25)	15 (10-20.25)	0.288[α]
AST (U/L)	20 (15-26)	18 (14-22)	0.010[α]
BUN (mg/dL)	12.5 (9.19-17.30)	12.5 (9.57-18.88)	0.669[α]
Creatinine (mg/dL)	0.8 (0.60-0.92)	0.7 (0.60-0.91)	0.350[α]
WBC (x103 /mm3)	8.8 (6.50-12.42)	7.4 (6.17-9.16)	**0.005[α]**
Hemoglobin (g/dL)	13.3±1.9	13.4±1.7	0.671[μ]
Platelet (x103 /mm3)	243.9±88.5	250.4±81.8	0.554[μ]
Sodium (mmol/L)	138 (136-140)	138.7 (137-140)	0.090[α]
Glucose (mg/dL)	113 (99-143)	98 (89-108)	**<0.001[α]**
Potassium (mmol/L)	4.1±0.5	4.3±0.4	**<0.001[μ]**
GPR	28.1 (23.76-36.34)	22.1 (20.14-26.83)	**<0.001[α]**

ALT: Alanine aminotransferase; AST: Aspartate aminotransferase; BUN: Blood urea nitrogen; F: Female, GPR: Glucose to potassium ratio, K: Serum potassium levels, M: Male, WBC: White blood cell.

[μ] : Student t-test was used for normally distributed data and resuts showed as Mean±standart deviation

[α] : Mann-Whitney U test used for non-normally distributed data and resuts showed as Median (25^{th} and 75^{th} percentile)

* : Gender variable was analyzed by the Chi-Square test

119 patients were included in Group A and 118 in Group B for the study. Sociodemographic data and blood laboratory values of the patients are shown in Table 1. Group A included 51 females and 68 males, while Group B included 53 females and 65 males. The median age of seizure cases was 49 (33-64), while the non-seizure group was found to be 48.5 (32.75-64.25). Statistically significant differences were observed in WBC, glucose levels, potassium, and GPR between Groups A and B (p=0.005, p<0.001, p<0.001, p<0.001, respectively), with increased levels of WBC, glucose, and GPR found in Group A patients.

The ROC curve analysis of GPR in patients with epileptic seizures is shown in Table 2 and Figure 2. GPR, with a cut-off value of 24.8, demonstrated a predictive value for active epileptic seizures with a sensitivity of 71.4%, specificity of 66.9% and an AUC value of 0.758.

Table 2. ORs of the GPR for predicting epileptic seizures.

	Model 1		Model 2		Model 3	
	OR (%95 CI)	p value	OR (%95 CI)	p value	OR (%95 CI)	p value
GPR	0.871 (0.831-0.912)	<0.001	0.870 (0.830-0.912)	<0.001	0.873 (0.831-0.917)	<0.001

Model 1: unadjusted model
Model 2: adjusted for age and sex.
Model 3: each marker was adjusted for WBC, age and sex.
CI: confidence interval, GPR: Glucose Potassium Ration, OR: Odds ratio, WBC: White blood cells count.

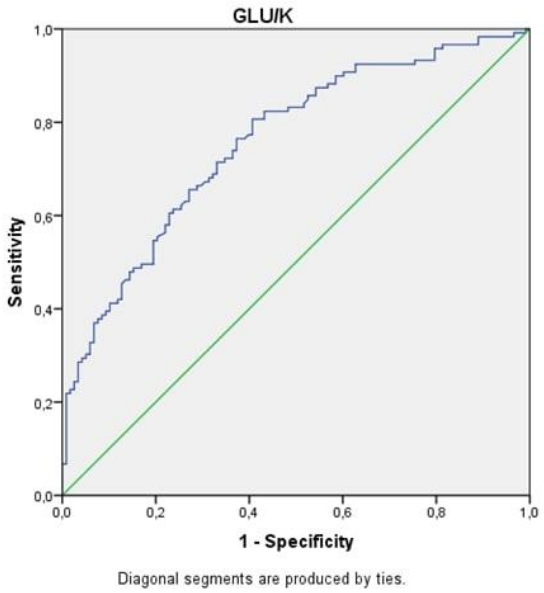

Figure 2. ROC analysis of GPR ratio

.A Multivariate logistic regression analysis was performed to assess the independent effect of GPR on the prediction of epileptic seizure activity. In the unadjusted model, the OR for GPR was 0.871 (95% CI 0.831–0.912); in the model adjusted for age and sex, the OR for GPR was 0.870 (95% CI 0.830–0.912), and 0.873 (95% CI 0.831–0.917) in the model adjusted for age, gender, and WBC. GPR was an independent predictor of epileptic seizure activity in both the adjusted and unadjusted models ($p < 0.001$)

Table 3. ROC curve analysis of GPR in epileptic seizure patients.

Parameter	AUC (95% CI)	Cut off	p value	Sensitivity	Specificity	+LR	-LR	PPV	NPV
GPR	0.758 (0.697-0.818)	21.4	<0.001	0.899	0.415	1.537	0.243	0.608	0.803
GPR	0.758 (0.697-0.818)	24.8	<0.001	0.714	0.669	2.157	0.428	0.685	0.699
GPR	0.758 (0.697-0.818)	30.2	<0.001	0.462	0.864	3.397	0.623	0.774	0.614

AUC: Areas under the curve, CI: Confidence interval, GPR: Glucose to potassium ratio, +LR: Positive likelihood ratio, -LR: Negative likelihood ratio, NPV: Negative predictive value, PPV: Positive predictive value, ROC: Receiving operating characteristics.

DISCUSSION

To the best of our knowledge, this is the first study in the literature to investigate the role of GPR on epileptic seizure activity. The findings of this study demonstrated that GPR was higher in patients with generalized motor seizure activity than in seizure-free patients followed up with a diagnosis of epilepsy. In multivariable logistic regression analysis, GPR independently predicted generalized motor seizure activity.

Circadian rhythms of plasma cortisol concentrations can influence the balance of neuronal excitability and inhibition, and there is a correlation between plasma cortisol and epilepsy susceptibility[10,11]. In a study conducted by Rider et al., serum cortisol levels were compared between patients with epileptic seizures and those with psychogenic non-epileptic seizures, revealing higher serum cortisol levels in patients with epileptic seizures. Although the same study identified significant variations in cortisol levels due to circadian rhythm in both groups, it also demonstrated that the cortisol response triggered by seizure activity differed significantly between the groups. Specifically, in the group with epileptic seizure activity, an acute increase in cortisol levels was observed within the first hour following the seizure[12].

Cortisol also induces lipolysis in adipose tissues and promotes hepatic ketogenesis and gluconeogenesis[13]. Patients with acute neurological illnesses exhibit excessive catecholamine production due to sympathetic activation, which is strongly associated with the severity of the illness[14]. Catecholamines, including dopamine, epinephrine, and norepinephrine, are among the most crucial subgroups of neurotransmitters in the central nervous system (CNS). They affect the highest levels of mental function and play a key role in neurological disorders like epilepsy[15]. Catecholamines not only activate the release of glucagon following stress and injury but also inhibit the secretion of insulin, further elevating serum glucose levels[16]. Stress and sympathetic discharge occurring during an epileptic seizure stimulate the adrenal medulla, leading to the release of catecholamines, such as epinephrine. Epinephrine, through ß2 receptors, stimulates the Na-K ATPase pump, driving potassium into the cells[7,17]. Consequently, hyperglycemia and hypokalemia are predictable conditions. A study in a pediatric age group found that patients experiencing their first febrile convulsion had higher serum glucose levels, along with lower serum sodium and chloride levels[18]. Our study also demonstrated that increased GPR is associated with active epileptic seizure activity. Additionally, there was an increase in serum glucose levels and decrease in serum potassium levels during epileptic seizure activity.

Although the mechanisms underlying the pathophysiology of epilepsy are still unelucidated, studies continue to be conducted to diagnose epileptic seizures. In patients with early-onset seizures, higher levels of D-dimer, apolipoprotein CIII, neural cell adhesion molecule, and Fas ligand, and lower levels of heat shock 70kDa protein-8 (Hsc70) and tumor necrosis factor receptor 1 have been identified [19]. However, the utility of these parameters is limited owing to their susceptibility to various factors. When assessing the etiology of a seizure (epileptic or non-epileptic), prolactin levels obtained shortly after a seizure typically increase three to four times, with a higher likelihood of occurrence in generalized tonic-clonic seizures than in other seizure types. However, significant variability in prolactin levels hinders their routine clinical use. Although the American Academy of Neurology has reported that serum prolactin levels may be useful in diagnosing epileptic seizures, their use in differential

diagnosis, particularly in conditions causing altered consciousness such as syncope, is not recommended [4,20]. While serum levels of anticonvulsant agents can be studied to determine baseline levels, potential toxicity, lack of efficacy, treatment non-compliance, and/or autoinduction or pharmacokinetic changes, cerebrospinal fluid examination and imaging methods are used in patients with suppressed CNS, suspected meningitis or encephalitis. To establish epileptic seizure activity, neuroimaging assessments (e.g., magnetic resonance imaging, computed tomography) and EEG are required. Clinical diagnosis can be confirmed by abnormalities in interictal EEG; however, these abnormalities can also be present in healthy individuals, and their absence does not exclude an epilepsy diagnosis. Video-EEG monitoring is the standard test for classifying the type of seizure or syndrome or diagnosing psychogenic non-epileptic seizures (i.e., making a definitive diagnosis of seizures with loss of consciousness). This technique is also used to characterize seizure types and epileptic syndromes to optimize pharmacological treatment and pre-surgical evaluation [20]. However, implementing these tests is often not feasible for patients who have had an active seizure, in terms of both the time and resources of the healthcare facility.

Emergency department visits with a history of suspected seizures are very common. While the history provided by the patient and witnesses often guides the diagnosis of epileptic seizures, diagnosis in patients without witnesses who are unconscious or prone to sleepiness presents a challenging situation for clinicians. Considering these uncertainties, there is a clear need for simple tests or parameters to guide clinicians in early diagnosis. The serum GPR was calculated by dividing the serum glucose level by the serum potassium level. Its elevation has been reported to be highly associated with increasing severity and poor prognosis in certain pathological conditions such as carbon monoxide poisoning [21]. In literature reviews, high serum glucose and low serum potassium levels have been identified in critical illnesses such as sepsis, blunt abdominal and thoracic trauma, and acute aortic dissection [21–25]. Additionally, post-traumatic hyperglycemia and insulin resistance have been reported to be associated with increased mortality and severe clinical outcomes[26]. Additionally, increased GPR has been found in certain conditions affecting the CNS, such as severe traumatic brain injury, traumatic spinal trauma, stroke, and aneurysmal subarachnoid hemorrhage [27–30]. Our data indicate a high correlation between increased GPR and epileptic seizure activity. Additionally, multivariate logistic regression analysis demonstrated that GPR was an independent variable for predicting epileptic seizure activity.

The primary limitation of our study was that it was conducted at a single center with a limited number of patients. Although the control group consisting of epilepsy patients without any complaints or symptoms may seem to be a negative factor, it actually provided more efficient comparative results, as the medications and treatments used in these patients would generate similar metabolic activities. Therefore, we believe that this broader prospective study is beneficial.

In conclusion, this study highlights the potential of GPR as an independent biomarker for predicting epileptic seizure activity. Our findings suggest that elevated GPR is significantly associated with active seizure episodes, offering a simple yet effective diagnostic tool, especially in emergency settings where advanced neuroimaging and video-EEG monitoring may not be readily available. Given the limitations of traditional diagnostic methods and the often urgent nature of seizure-related healthcare visits, GPR could serve as a valuable addition to clinical practice, facilitating timely and accurate diagnosis. Future prospective studies are warranted to validate these findings and to explore their broader clinical implications.

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REFERENCES

1. Sazgar M, Young MG. Seizures and Epilepsy in Absolute epilepsy and EEG rotation review, (Eds Sazgar M, Young MG);9-46. Cham, Springer International Publishing. 2019.
2. Huff JS, Morris DL, Kothari RU, Gibbs MA, Emergency medicine seizure study group (EMSSG)*. Emergency department management of patients with

Sseizures: A multicenter study. Acad Emerg Med. 2001;8:622–8.
3. Restrepo-Vera JL, Coscojuela P, Fonseca E, Quintana M, Sarria-Estrada S, Santamarina E et al. Epileptic seizures in the emergency room: clinical and electroencephalographic findings associated with brain perfusion patterns on computed tomography. J Neurol. 2022;269:3761-69.
4. Verny M, Greffard S. Contribution of geriatric model to the management of an epileptic seizure or epilepsy. Geriatr Psychol Neuropsychiatr Vieil. 2019;17:21–4.
5. Li H, Chen K, Yang L, Wang Q, Zhang J, He J. The role of plasma cortisol in dementia, epilepsy, and multiple sclerosis: A Mendelian randomization study. Front Endocrinol (Lausanne). 2023;14:1107780.
6. Lee SC, Baranowski ES, Sakremath R, Saraff V, Mohamed Z. Hypoglycaemia in adrenal insufficiency. Front Endocrinol (Lausanne). 2023;14:1198519.
7. Gözübatık Çeli RG, Özkara Ç. Seizures with autonomic symptoms and sudden unexpected death in epilepsy (SUDEP). Turkish J Neurol. 2019;25:109–16.
8. Boyuk F, Çalışkan S, Yigen R. Glucose-to-potassium ratio: A novel index associated with the clinical status in acute coronary syndrome patients. Ann Clin Anal Med. 2022;13.
9. Katyayan A, Diaz-Medina G. Epilepsy: Epileptic syndromes and treatment. Neurol Clin. 2021;39:779–95.
10. Young E, abelson J, lightman S. Cortisol pulsatility and its role in stress regulation and health. Front Neuroendocrinol. 2004;25:69–76.
11. Sarabdjitsingh RA, Jezequel J, Pasricha N Mikasova L, Kerkhofs A, Karst H et al. Ultradian corticosterone pulses balance glutamatergic transmission and synaptic plasticity. Proc Natl Acad Sci. 2014;111:14265–70.
12. Rider F, Turchinets A, Druzhkova T, Kustov G, Guekht A, Gulyaeva N. Dissimilar changes in serum cortisol after epileptic and psychogenic non-epileptic seizures: a promising biomarker in the differential diagnosis of paroxysmal events? Int J Mol Sci. 2024;25:7387.
13. Tesfaye N, Seaquist ER. Neuroendocrine responses to hypoglycemia. Ann N Y Acad Sci. 2010;1212:12–28.
14. Kurtz P, Claassen J, Schmidt JM Helbok R, Hanafy K A, Presciutti M et al. Reduced brain/serum glucose ratios predict cerebral metabolic distress and mortality after severe brain injury. Neurocrit Care. 2013;19:311–9.
15. Ogura T, Satoh A, Ooigawa H, Sugiyama T, Takeda R,Fushihara G et al. Characteristics and prognostic value of acute catecholamine surge in patients with aneurysmal subarachnoid hemorrhage. Neurol Res. 2012;34:484–90.
16. Fredj Z, Sawan M. Advanced nanomaterials-based electrochemical biosensors for catecholamines detection: challenges and trends. Biosensors (Basel). 2023;13:211.
17. Lloyd C, Mohar C, Priano J. Hypokalemic cardiac arrest: narrative review of case reports and current state of science. J Emerg Nurs. 2022;48:310–6.
18. Sharawat IK, Singh J, Dawman L, Singh A. Evaluation of risk factors associated with first episode febrile seizure. J Clin Diagn Res. 2016;10:10-3.
19. Phan J, Ramos M, Soares T, Parmar MS. Poststroke seizure and epilepsy: a review of incidence, risk factors, diagnosis, pathophysiology, and pharmacological therapies. Oxid Med Cell Longev. 2022;2022:1–15.
20. Lucke-Wold BP, Nguyen L, Turner RC, Logsdon AF Chen YW, Smith KE et al. Traumatic brain injury and epilepsy: Underlying mechanisms leading to seizure. Seizure. 2015;33:13–23.
21. Demirtaş E, Korkmaz İ, Tekin Y, Demirtaş E, Çaltekin İ. Assessment of serum glucose/potassium ratio as a predictor for delayed neuropsychiatric syndrome of carbon monoxide poisoning. Hum Exp Toxicol. 2021;40:207–13.
22. Johnson KB, Wiesmann WP, Pearce FJ. The effect of hypothermia on potassium and glucose changes in isobaric hemorrhagic shock in the rat. Shock. 1996;6:223–9.
23. Chen Y, Peng Y, Zhang X, Liao J,Lin J,Chen L et al. The blood glucose-potassium ratio at admission predicts in-hospital mortality in patients with acute type A aortic dissection. Sci Rep. 2023;13:15707.
24. Katipoğlu B, Demirtaş E. Assessment of serum glucose potassium ratio as a predictor for morbidity and mortality of blunt abdominal trauma. Ulus Travma Acil Cerrahi Derg. 2022;28:134–9.
25. Turan E, Şahin A. Role of glucose/potassium ratio and shock index in predicting mortality in patients with isolated thoracoabdominal blunt trauma. Turkish journal of trauma & emergency surgery. 2022;28:1442–8.
26. Su WT, Wu SC, Chou SE, Huang CY, Hsu SY, Liu HT et al. Higher mortality rate in moderate-to-severe thoracoabdominal injury patients with admission hyperglycemia than nondiabetic normoglycemic patients. Int J Environ Res Public Health. 2019;16.
27. Marini JI, Sein ME. The role of the glucose potassium ratio in the management of traumatic brain injury. Korean J Neurotrauma. 2023;19:82–9.
28. Zhou W, Liu Y, Wang Z, Mao Z, Li M. Serum glucose/potassium ratio as a clinical risk factor for predicting the severity and prognosis of acute traumatic spinal cord injury. BMC Musculoskelet Disord. 2023;24:870.
29. Lu Y, Ma X, Zhou X, Wang Y. The association between serum glucose to potassium ratio on admission and short-term mortality in ischemic stroke patients. Sci Rep. 2022;12:8233.

30. Matano F, Fujiki Y, Mizunari T, Kotetsu K, Tamaki T, Murai Y et al. Serum glucose and potassium ratio as risk factors for cerebral vasospasm after aneurysmal subarachnoid hemorrhage. J Stroke Cerebrovasc Dis. 2019;28:1951–7.