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Correlation Between Serum Glucose/Potassium Ratio and The Severity of Mushroom Poisoning at The Time of Admission to The Emergency Departments

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ÖZ

Founded: 2004

Research Article	ABSTRACT
	Exposure to wild mushrooms can lead to serious toxicity and death. It is accepted that patients who ingest
	potentially lethal mushrooms typically develop toxicity signs after six hours. However, clinical manifestations of
History	poisoning that occur less than six hours after ingestion do not exclude the potential for life-threatening toxicity,
-	especially when more than one type of mushroom has been eaten. Whereas there are not any clinical
Received: 04/02/2022	parameters that help to establish the severity of mushroom poisoning. In this study, we aimed to determine the
Accepted: 25/03/2022	relationship of serum glucose/potassium ratio and the clinical severity of mushroom poisoning cases. This is a
	retrospective study which includes the mushroom poisoning 510 cases between the years 2007 - 2018. Data
	consisted of age, gender, clinical history of mushroom poisoning including time from consumption to first
	symptoms, date of presentation, discharge time and laboratory results including complete blood cell count,
	biochemistry tests for liver and renal function, and coagulation profile. Patients included in this study were

classified as mild-moderate and severe mushroom poisoning groups according to laboratory and clinical characteristics. Glucose, BUN, Creatinine, ALT, AST mean values and glucose/potassium ratio were significantly higher in the clinically severe group patients (p=0.008, p=0.01, p=0.039, p=0.037, p=0.046 and p=0.036 respectively). The sensitivity, specificity and area under curve for glucose/potassium ratio were as follows; 0.68, 0.57 (AUC %95CI) was 0.0647. Glucose/potassium ratio can predict the severity in mushroom poisonings according to our results which can helpful by management in mushroom poisonings as a laboratory result.

Keywords: Mushroom poisoning, glucose/potassium ratio, emergency department, mushroom poisoning, glucose/potassium ratio, emergency department

Acil Servislere Başvuru Anında Serum Glukoz/Potasyum Oranı ile Mantar Zehirlenmesinin Şiddeti Arasındaki İlişki

Sürec

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Yabani mantarlara maruz kalmak ciddi toksisiteye ve ölüme neden olabilir. Potansiyel olarak öldürücü mantarları yiyen hastaların tipik olarak altı saat sonra toksisite belirtileri geliştirdiği kabul edilmektedir. Bununla birlikte, yuttuktan altı saatten daha kısa bir süre sonra ortaya çıkan zehirlenmenin klinik belirtileri, özellikle birden fazla mantar türü yendiğinde, yaşamı tehdit eden toksisite potansiyelini dışlamaz. Oysa mantar zehirlenmesinin ciddiyetini belirlemeye yardımcı olan herhangi bir klinik parametre yoktur. Bu çalışmada mantar zehirlenmesi olgularının serum glukoz/potasyum oranı ile klinik şiddeti arasındaki ilişkiyi belirlemeyi amaçladık. Bu, 2007 -2018 yılları arasında 510 mantar zehirlenmesi vakasını içeren retrospektif bir çalışmadır. Veriler, yaş, cinsiyet, tüketimden ilk semptomlara kadar geçen süreyi içeren klinik mantar zehirlenmesi öyküsü, başvuru tarihi, taburculuk zamanı ve aşağıdakileri içeren laboratuvar sonuçlarını içermektedir. tam kan hücresi sayımı, karaciğer ve böbrek fonksiyonu için biyokimya testleri ve pıhtılaşma profili. Bu çalışmaya dahil edilen hastalar laboratuvar ve klinik özelliklerine göre hafif-orta ve şiddetli mantar zehirlenmesi grupları olarak sınıflandırıldı. Glukoz, BUN, Kreatinin, ALT, AST ortalama değerleri ve glukoz/potasyum oranı klinik olarak şiddetli gruptaki hastalarda anlamlı olarak daha yüksekti (sırasıyla p=0.008, p=0.01, p=0.039, p=0.037, p=0.046 ve p=0.036). Glikoz/potasyum oranı için duyarlılık, özgüllük ve eğrinin altındaki alan aşağıdaki gibidir; 0.68, 0.57 (AUC %95CI) 0.0647 idi. Glikoz/potasyum oranı, laboratuvar sonucu olarak mantar zehirlenmelerinde yönetime yardımcı olabilecek sonuçlarımıza göre mantar zehirlenmelerinin ciddiyetini tahmin edebilir.

Anahtar sözcükler: Mantar zehirlenmesi ve serum glukoz/potasyum oranı.

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Introduction

Wild mushroom poisoning is a major source of mortality-morbidity ¹, the exposures rate is increasing over the world ². While 52 types are toxic to humans, 32 types have been associated with fatalities. The wild mushroom poisoning occur generally in late spring and autumn. While there are more than 200 species, toxic and non-toxic, and can grow in the same area even trained mycologists often confuse to determine the non-toxic species. It is also difficult for the mycologist or the humans to differentiate the toxic wild mushrooms while there are no simple tests for identification ^{3,4}.

The clinical symptoms can range from slight gastrointestinal malaise to death ⁵. To define the patient as a wild mushroom intoxicated patient we can use the clinical symptoms and the background, the expected laboratory results or if we can reach the mushroom species it can be analyzed by a mycologist¹. Patients who are exhibiting intoxication symptoms within 6h after consumption of the meal, this intoxicated patients general don't have severe clinical symptoms and mostly treatment is sufficient. Whereas symptomatic consumption of mushrooms with early and late intoxication symptoms may cause confusion in the clinical state 6-11.

While the ingested type of the mushroom is unknown and the patient can eat mushrooms with early and late exhibiting clinical symptoms together it is difficult to make a risk assessment and management strategy in the emergency department (ED) quickly ¹². Also, the initial symptoms of mushroom poisoning may be misleading and mimic many diseases and may not correlate with prognosis ¹³.

It is accepted that patients who ingest potentially lethal mushrooms (e.g., Amanita virosa, Amanita phalloides, Cortinarius orellanus, Gyromitra esculenta) typically develop signs of toxicity after than six hours ingestion ^{14,15}. However, clinical manifestations of poisoning that occur before six hours after ingestion do not exclude the potential for life-threatening toxicity, especially due to the mix type mushrooms consumption. That's why there are not any clinical parameters that help to establish a relationship between presentation and severity of mushroom poisoning.

Catecholamines, glucocorticoids and growth hormones are enhanced due to acute diseases or stress. Mushroom poisoning is also a clinical state with increased stress. Due to the increased level of the hormones serum glucose level increases and after while insulin, as a counter-regulatory hormone, increases the glucose transport through the cell membrane with activation of Na⁺/K⁺-ATPase which results in hypokalemia. That's why we hypothesized that there would be a correlation between serum glucose/potassium ratio and the clinical severity of mushroom poisoning cases.

Material and Methods

This is a retrospective study which includes the mushroom poisoning patients defined according to the International Classification of Diseases, Ten Revision (ICD-10) using code T62.0 (Toxic effects of ingested mushrooms) analyzed the years between 2007 - 2018. T62.0 code included as a general mushroom poisoning code. We could not all classify the intoxication state according to the mushroom type since there is no mycologist in our university. İnitally 738 patients who admitted to the Emergency Service of Sivas Cumhuriyet University Hospital were determined. While 137 pediatric patients(≤16 years) were excluded due to their age group, 48 patients were excluded due to pre-existing causes of hyperglycemia, diabetes mellitus, metabolic syndrome, insulin resistance or taking oral hypoglycemic, insulin or beta-blocker agents. Also according to the background history of the patients' demographic and clinical data we recorded from the dedicated medical charts 43 patients who had any disease resulting in hyper or hypokalemia were also excluded and at all 510 cases were analyzed.

Data consisted of age, gender, clinical history of mushroom poisoning including time from consumption to first symptoms, date of presentation, discharge time and admission laboratory results including complete blood cell count, biochemistry tests for liver and renal function, and coagulation profile. Type of management as outpatient and inpatient, treatment(s) provided, length of stay, discharge diagnosis, or admission in the intensive care unit data were also collected.

Patients included in this study were classified as mildmoderate and severe mushroom poisoning groups according to laboratory and clinical characteristics. Nausea-vomiting and abdominal pain after mushroom consumption was the minimum criteria for patients in the mild-moderate group. The severe mushroom poisoning group was created by one of the admission or hospitalization time laboratory results: creatinine level >2 mg/dL, triple increased liver function tests (ALT>99µ/L or AST>96µ/L), alteration of mental status or twice increased the level of international normalized ratio at admission. Admission glucose and potassium levels were accepted for all groups.

Four-hundred fifty-eight patients admitted within 1 hour to emergency departments were treated with an orogastric lavage. The first dose of activated charcoal was given as 1gr/kg. the main aim in the mild-moderate group patients treatment was to maintain the fluidelectrolyte balance and to relieve the patient's symptoms. Crystallized penicillin was used for severe patients according to the literature.

Statistical Analysis

The study sample descriptive statistics were generated by using SPSS 23.0 (IBM SPSS Statistics for Windows, Armonk, NY, USA). Kolmogorov-Smirnov test was used to asses the data distribution normality. Continuous data variables are presented as the median and interquartile range ($25^{th}-75^{th}$ quartiles) and were compared with Mann Whitney U and χ^2 test. A Linear regression analysis was made for some laboratory variables (Glucose, AST, ALT, BUN, Creatinin) to present the effectivity on the hospitalization time. *P* < 0.05 levels were accepted statistically significant.

Results

In the 11-year study period, there were 510 cases (491 in the clinically mild-moderate group, 19 cases in the clinically severe group) of mushroom poisoning. While the mean age of the mild-moderate group was 38.30 ± 18.26 years, the mean age of the clinical severity group was 43.42 ± 20.70 years. There weren't any significant differences between the groups' distribution according to age, sex, and symptoms beginning times (p=0.92, p=0.82, and p=0.91 respectively). (Table 1)

Table 1 presents the frequency of symptoms and complaints at admission time according to the clinical severity. Only the confusion state of the patients was significant between the groups of mushroom poisoning. It was significantly higher among the severe group (p=0,004).

The laboratory findings of the study population with mild-moderate and severe mushroom poisonings were presented in Table 2. The hematological variables [White blood cell (WBC), Platelets, Lymphocyte, Monocytes, variables Neutrophil)], biochemical [alanine aminotransferase (ALT), aspartate aminotransferase (AST). blood urea nitrogen (BUN), Creatinine], internationalization normalization rate and glucose/potassium ratio were analyzed between the groups. Glucose, BUN, Creatinine, ALT, AST mean values and glucose/potassium ratio were significantly higher in the clinically severe group patients (p=0.008, p=0.01, p=0.039, p=0.037, p=0.046 and p=0.036 respectively).

The study aimed was to define a new biomarker ratio for the prediction of severe group mushroom poisoning. Therefore, we calculated the sensitivity, specificity and area under the curve for glucose/potassium ratio. The sensitivity was 0.68, specificity 0.57 and area under the curve (AUC %95CI) was 0.0647. (Table 3, Fig.1)

A Linear regression analysis made for Glucose, AST, ALT, BUN, Creatinin and hospitalization time. The Glucose/potassium ratio was found to be the most effective determinant (Table 4).

Discussion

Glucose plasma levels in critically patients increases due to increased insulin resistance. A hypermetabolic state exists in these patients which induces counterregulatory hormones and cytokines that are important for insulin resistance and result in hyperglycemia^{15,16}.

Hyperglycemia is used as a prognostic biomarker for cerebrovascular diseases, acute coronary syndromes, burns, and head injuries critically ill patients. High glucose decreases the endothelial nitric oxide, vascular reactivity, mitochondrial function. Hyperglycemia can lead to cardiovascular collapse due to cardiac-hemodynamic complications and kidney failure ^{15,17}.

We analvzed the serum glucose and glucose/potassium ratio in our two groups as classified according to the liver and kidney function tests among the patients with mushroom poisonings. We determine that the plasma glucose levels and glucose/potassium ratio were significantly higher in the patients who were defined as severely groups. The sensitivity and specificity for the glucose/potassium ratio were calculated as 0.68 and 0.57. Also, the area under the curve was 0.647(%95% Cl). Hospitalization time is also a marker for the clinical severity. Glucose/potassium ratio was the best variable that predicted hospitalization time.

Ia	ble 1. Distribution of mushroom-poisoned mild-moderate and severe patient group according to the frequency of
	symptoms and complaints at admission time and demographic characteristics the frequency of symptoms and
	complaints at admission time.

Characteristics	Mild-moderate Patient	Severe Patient Group	P value
Characteristics	Group (n= 491, %)	(n= 19, %)	(Chi squared test)
Age, year (Mean ± SD)	38.30 ±18.26	43.42 ±20.70	0.927*
Gender			
Male	211 (43)	11(57.9)	0.823
Female	280 (57)	8 (42.1)	
The first symptom beginning time			0.918
0-6 hours	443(90)	17(90)	
> 6 hours	48(10)	2(10)	
The first clinical finding and symptoms			
Nausea and vomiting	289(58.9)	13 (68.4)	0.279
Abdominal pain	185(37.7)	10(52.6)	0.201
Diarrhea	119(24.2)	7 (36.8)	0.222
Dizziness	83(16.9)	5 (26.3)	0.298
Headache	84(17.1)	4(21.1)	0.671**
Vision loss	16(3.3)	2(10.5)	0.095**
Urticaria	15(3.1)	0	>0.05**
Confusion	15(3.1)	4(21.1)	0.004**
Pruritis	27(5.6)	0	0.615**

*Student t-test, Fisher's exact test**

Variables	Mild-moderate Patient Group (N= 491 mean±SD)	Severe Patient Group (N= 19 mean±SD)	P-value*
White blood cell (10 ³ /µl)	10.3 ± 3.4	10.9 ± 3.5	0.536
Hemoglobin (g/dL)	13.6 ± 1.9	14.0 ± 2.5	0.598
Platelet (10 ³ /µl)	253.5 ± 61.3	245.5 ± 116.2	0.782
Glucose (mg/dl)	105.3 ± 16.5	116.6 ± 16.3	0.008
BUN(mmol/L)	13.3 ± 5.2	19.0 ± 8.7	0.011
Creatinine (mg/dL) Median (%25-75)	0.7 (0,59-0,80)	1.0 (0.85-1.3)	0.039
ALT (U/L) Median (%25-75)	17 (14-22)	94 (21-349)	0.037
AST (U/L) Median (%25-75)	22 (19-27)	124 (34-391)	0.046
Sodium(mmol/L)	137.6 ± 6.3	137.4 ± 2.5	0.756
Potassium(mmol/L)	4.0 ± 0.8	4.1 ± 0.5	0.584
INR	1.04(0.9-1.11)	1.07(1.03-1.13)	0.432
Glucose/potassium ratio	26.3 ± 6.9	28.7 ± 4.5	0.036

*Student t-test, BUN: blood urea nitrogen, ALT: alanine aminotransferase, AST: aspartate aminotransferase, INR: (International normalized ratio), Data P-value as median (25% -75% interquartile ranges).

Table 3. Cut-off value, sensitivity, and specificity of Glucose/Potassium Ratio, for predicting disease severity in patients with mushroom poisoning.

	Glucose/Potassium Ratio
Cut-off value	26.93
Sensitivity	0.68
Specificity	0.57
PPV	0,68
NPV	0,56
AUC (95% CI) [*]	0.647 (0,523-0,770)

*AUC, area under the curve; CI, confidence interval.

Table 4. Multiple regression analysis of selected variables.

Independent variables	В	95%Cl	Р
Glucose/ Potassium Ratio	0.27	0.02-0,05	0.001
AST	0.40	0.000-0,003	0.141
ALT	-0.28	-0.03-0,001	0.30
White blood cell	0.012	-0.039-0,049	0.825
BUN	0.058	-0.007-0,019	0.346
Creatinine	0.142	0.145-1,510	0.018

AST: aspartate aminotransferase, ALT: alanine aminotransferase, BUN: blood urea nitrogen





There aren't any clinical studies about the wild mushroom poisoning and glucose/K ratio. The clinical severity of wild mushroom poisonings was evaluated by different methods. Bonacini et all. the collected information from 27 patients with clinically severe hepatitis due to mushroom ingestion. They found that serum AST level <4000 IU/L and peak INR <2 were correlated with a good outcome. Whereas 3 patients with poor outcome had nadir factor V value as 9%, 13%, and 15%. Age was determined also as an indicator of mortality. The patients >65 years and women had a poor outcome ^{12,18}.

Mortality is generally related to acute liver failure and jaundice is described as the early sign. Jan et al. analyzed 18 patients who admitted to their hospital due to gastroenteritis complaints. All of them were managed as gastroenteritis until they have determined liver involvement. Delayed treatment of the patients is emphasized as the main reason for the increased mortality rate (72.3%)¹⁹.

Ganzert et al. analyzed the indications of liver transplantation after amatoxin type of mushroom intoxications. They determined that the prothrombin index and serum creatinine levels were significantly higher among the fatal outcome patients' when this was compared with serum bilirubin and alanine transferase levels. Also, less than 25% of prothrombin index which has been combined with creatinine greater than 106 mmol/l had an optimum prediction of fatal outcome and was better than the accepted King's College criteria ²⁰.

Potassium is the main electrolyte stored in the cell and plasma levels are controlled by adenosine its triphosphatase sodium/potassium pump (Na⁺/K⁺-ATPase). Serum potassium level will be decreased by catecholamines, B2 adrenergic hormones, and insulin, particularly after important injury and stress. Potassium alone as a simple prognostic indicator is evaluated by many authors. Tongyoo S et all. analyzed the serum potassium levels and outcomes in severely ill patients. The patients in intensive care unit were divided as normal or abnormal potassium groups. Most of the patients were in the hypokalemic group and had a significantly higher mortality rate among this group (24.3% vs. 39.5%, p=0.04) ²¹. Xi et al. and Goyal et all evaluated the correlation between serum potassium levels and mortality rates in acute myocardial infarctions. While Xi et al. determined that hypokalemic and hyperkalemic patients with AMI had an increased risk of mortality, Goyal et al. observed the lowest mortality in those with normal range postadmission serum potassium levels ^{22,23}.

In the literature, the serum glucose and potassium ratio are only evaluated in subarachnoidal hemorrhagic patients. Matano F. et al. found a significant correlation between cerebral vasospasm induced ischemic complications and glucose/potassium ratio. Also, there was a correlation between serum glucose/potassium ratio and poor outcome ²⁴. Fujiki et al. reviewed the records of subarachnoidal hemorrhagic patients retrospectively. They classified the severity of SAH patients according to Hunt and Kosnik classification. Hunt and Kosnik grade IV

or V were accepted as severe SAH. A positive correlation of glucose/potassium ratio and severe SAH was determined (p <0.0001) and serum glucose/potassium ratio was elevated in an H-K grade–dependent manner (Spearman's r = 0.5374, p <0.0001). The discharged patients were classified according to the Glasgow Outcome Scale and the glucose/potassium ratio was significantly higher among patients with severe disabilities.

Limitations

The most important limitation of this study is that it is a retrospective study. All patients were in a single center and the number of patients was relatively small.

Conclusion

As a consequence of increased stress and catecholamines, there was a significant difference between the mild-moderate and severe groups for the serum glucose/potassium ratio. This enables the health staff by prognostification among the wild mushroom poisonings and can be helpful by management in mushroom poisonings.

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