Korean J Neurotrauma. 2023 Mar;19(1):82-89
https://doi.org/10.13004/kjnt.2023.19.e11
pISSN 2234-8999·eISSN 2288-2243

KJNT

Clinical Article

The Role of the Glucose Potassium Ratio in the Management of Traumatic Brain Injury

Joaquín Ignacio Marini [1] **and Matías Emmanuel Sein** [2,3]

[1]Department of Neurological Surgery, Hospital Dr. Luis Güemes, Buenos Aires, Argentina
[2]Department of Neurosurgery, Hospital San Martín, La Plata, Argentina
[3]Department of Neurosurgery, Hospital Italiano, La Plata, Argentina

Received: Aug 25, 2022
Revised: Jan 10, 2023
Accepted: Jan 11, 2023
Published online: Mar 15, 2023

Address for correspondence:
Joaquín Ignacio Marini
Department of Neurological Surgery, Hospital Dr. Luis Güemes, Rivadavia 15000, Buenos Aires 1706, Argentina.
Email: jmarini137@gmail.com

ORCID iDs
Joaquín Ignacio Marini
https://orcid.org/0000-0001-7151-1201
Matías Emmanuel Sein
https://orcid.org/0000-0003-0155-8615

Conflict of Interest
The authors have no financial conflicts of interest.

ABSTRACT

Objective: Traumatic brain injury (TBI) has become a worldwide public health issue, raising concerns about which tool might be useful to guide initial management at hospital admission, especially to decide whether the patient would benefit from an opportune surgical intervention. Recently, the glucose-to-potassium ratio has more accurate predictive values than other biomarkers and is useful for its simplicity to obtain. To correlate each biomarker with the outcome for every patient with TBI.
Methods: The analysis included patients treated in a single institution between 2020 and 2021, diagnosed with mild TBI that required neurosurgery, moderate or severe TBI. Blood samples were obtained at admission, and the glucose-to-potassium ratio was calculated retrospectively. Then, these values and other variables were compared with the outcome at 6 and 12 months. Extracranial lesions that directly contributed to the outcome, a Glasgow Coma Scale of 3 and below, hemodynamic instability, and cardiac arrest were exclusion criteria.
Results: forty-seven patients who reached the criteria were examined, 35 (74%) had a favorable outcome and 12 (26%) a poor one. The only biomarker significantly related to the outcome was the glucose-to-potassium ratio in both the bivariate and multivariate analysis (p=0.04; odds ratio, 8.61; 95% confidence interval, 1.07–69.6).
Conclusion: An increase in the glucose-to-potassium ratio was the only biomarker associated with poor outcomes and increased mortality.

Keywords: Glucose; Potassium; Traumatic brain injury; Biomarkers

INTRODUCTION

Traumatic brain injury (TBI) is one of the leading causes of death and disability, and is a major concern worldwide but particularly in developing countries. In Argentina, national statistics show that traumatic injuries represent approximately 3% of the total deaths and around half of these occur between the first and third decades of life. TBI makes a significant contribution not only to the years of lost potential life, but also to the reduction in life expectancy.[17] Moreover, disability related to this injury has become a major concern due to its impact on the economically active population.

Many biomarkers have been proposed as predictors of outcome in TBI. Coagulation disorders are reported in the early stages after trauma,[9] although sometimes delays in laboratory results make it difficult for markers such as the D dimer to become relevant enough. Therefore, several parameters measured in blood samples with readily achievable results have proven helpful in decision making at hospital admission.[6,14,18] Serum glucose and electrolytes such as sodium, calcium, and potassium are some examples.

TBI leads to changes in the parameters mentioned above because of cellular membrane mechanic disruption and membrane potential arousal, involving neurotransmitters and ion distribution between the intracellular space and extracellular matrix. In the acute phase (first hour), considerable glutamate release from the presynaptic axon terminals destabilize the membrane potential, precipitating a significant increase in potassium levels in the extracellular space, which correlates directly with the severity of TBI.[1,7]

Increases in serum glucose levels immediately after TBI involves a rapid increase in the cerebral metabolic rate associated with cellular ATP use that attempts to reestablish ion balance and cell membrane potential. This early hyperglycemic stage is followed by hypoglycemia, as observed in human and experimental models that reported in murine samples a serum glucose decrease lasting 5, 10 or 14 days in mild, moderate and severe TBI, respectively.[5,12] Moreover, the level of consciousness (Glasgow Coma Scale [GCS]) is related to glucose metabolic use rates in the thalamus, brainstem, and cerebellum.[4]

Recently, a ratio based on serum glucose and potassium levels has been proposed with the aim of studying 2 instead of only one indicator, thus avoiding false positive test results that appear, for example, in patients with diabetes while analyzing serum glucose values.[16] This ratio includes one biomarker with a rapid serum peak level (potassium) and one with steady serum level (glucose). We studied the glucose-potassium ratio as a prognostic factor in TBI by conducting a retrospective analysis of patients admitted to our institution over a period of one year, diagnosed with moderate or severe TBI, and including some cases of patients with mild TBI who underwent surgery. This study aims to understand the relationship between the glucose-potassium ratio and the outcome in patients with moderate or severe TBI.

MATERIALS AND METHODS

The database examination was carried out with the software “Consulta Practica” and produced 251 patients with TBI diagnosis between 2020 and 2021, without considering the severity of the trauma. Then, those with moderate, severe, or mild TBI requiring surgery were filtered, identifying 76 patients.

Exclusion criteria included cases of fatal polytraumatism with extracranial lesions, death related to nontraumatic extracranial lesions, GCS 3/15, nonreactive bilateral mydriasis, absence of brainstem reflexes, and cardiac arrest, age less than 16. Comorbidities included 3 cases of type 2 diabetes mellitus, 3 cases of alcoholism, 7 cases of hypertension, and 1 hormone therapy for breast cancer.

Laboratory samples were obtained at the moment of hospital admission in the resuscitation room, measuring blood glucose levels, electrolytes, lactate, and white blood cell count. Results were available within 60 minutes, approximately.

https://doi.org/10.13004/kjnt.2023.19.e11

To evaluate the result in TBI, the "modified ranking score" was calculated through a telephone interview carried out 6 and 12 months after trauma. The collected data were uploaded to the IBM SPSS statistics computer program (IBM Corp., Armonk, NY, USA). The retrospective data were then confirmed by a third-party reviewer.

Having categorized patients according to their progress, a cutoff point of modified ranking scale (mRS) ≤3 was envisaged as a favorable outcome, and an mRS ≥4 was considered unfavorable. Furthermore, a value of 50 was decided as the cutoff point for the glucose-potassium ratio; this was established in previous articles,[16] and the value was adopted for its calculation simplicity and because of its proximity to the standard mean.

Continuous variables with a normal distribution were expressed as mean ± standard deviation, and those variables without a normal distribution are presented with median and interquartile ranges 25–50–75. A descriptive statistical analysis was performed using asymmetry, kurtosis, Kolmogorov-Smirnov, and Shapiro-Wilk tests. The correlation between variables was evaluated by applying the Pearson correlation test for those normally distributed, and the Spearman correlation test for those not normally distributed. A $p>0.05$ was considered statistically significant.

Finally, logistic regression and cross-data analyzes were used for variables expressed in a mutually excluding dichotomic way.

An Institutional Review Board and/or an Ethics Committee approval was not necessary, given the retrospective nature of the study and the absence of patient identification data. Therefore, patient consent was not required.

RESULTS

During the study period, of the 76 patients included in the study and diagnosed with moderate, severe, or mild TBI that required surgical intervention, 1 presented with an extracranial (thoracic) lesion that directly contributed to the outcome, 3 died several months after surgery due to extracranial causes (1 with acute kidney injury and 2 with nosocomial pneumonia), 5 had a GCS of 3 at admission, 1 had hemodynamic instability and 5 presented with a cardiac arrest, and were excluded. Being our nosocomy is an adult-only hospital, there were no patients under 16 years of age. In addition to these 15 patients, we were unable to contact 14 cases during the follow-up period. Therefore, the evaluation included 47 patients (42 men, 5 women, between 16 and 68 years of age, mean 36 years old) (**FIGURE 1**).

The 47 patients were categorized by personal data, the severity of the TBI (mild, moderate, severe) according to the GCS, the presence of a surgical intervention, and a clinical outcome according to the mRS. Six presented with a mild TBI requiring surgical intervention (all underwent evacuation of extradural hematoma), 21 had moderate TBI, of whom 13 were treated surgically, 3 needed intracranial pressure monitoring, and 5 were controlled clinically. Of 20 with severe TBI, 13 required surgery, 6 required placement of intracranial pressure monitors, and one patient with cerebral hemorrhagic contusions was clinically maintained because of his rapid recovery.

https://doi.org/10.13004/kjnt.2023.19.e11

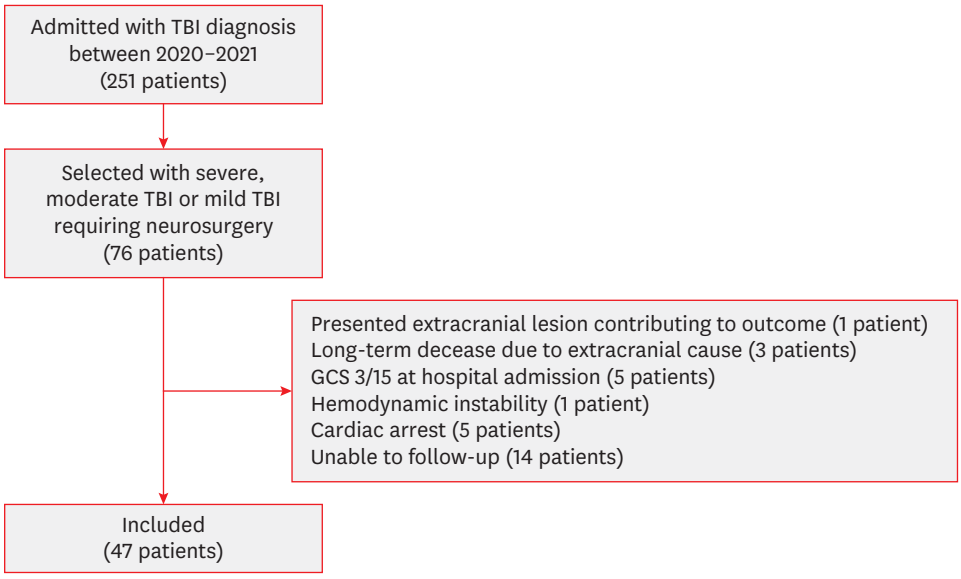

FIGURE 1. Flow diagram of patient selection.
TBI: traumatic brain injury, GCS: Glasgow Coma Scale.

The majority of surgical interventions were primary decompressive craniectomies for removable lesions (n=24); nevertheless a few cases of decompressive craniectomy secondary to refractory intracranial hypertension were included (n=4). Other surgical interventions included surgical toilette debridement and frontal sinus cranialization (n=5). In addition, 10 patients were monitored using an intracranial pressure catheter, all receiving medical treatment. Five cases of moderate TBI and one of severe TBI were clinically monitored due to their great general appearance associated with mild tomographic lesions (Marshall score of diffuse injury I–II).[10]

Thirty-five (74%) of the 47 patients achieved a favorable outcome (mRS ≤3) and 12 (26%) had unpromising outcomes, the mean age of the former was 34 and the latter 41. The total mean age was 36 (±15). The entire number of cases of mild TBI that required surgery had a favorable outcome, as well as 18 (78%) with moderate TBI and 11 (61%) of with severe TBI. Eight (44%) patients with severe TBI and 4 (17%) with moderate TBI had unfavorable outcomes. Moderate TBI was associated with a favorable outcome (odds ratio [OR], 0.529; 95% CI, 0.134–2.086; p=0.363), and severe TBI was associated with a detrimental outcome (OR, 0.261; 95% CI, 0.07–1.04; p=0.05).

The mean values obtained were for glucose 148 (±39), for potassium 3.65 (±0.5), for the glucose-potassium ratio 41 (±12), for leukocytes 15,472 (±5,388) and for lactate a median of 18 (interquartile ranges 25–50–75: 12–18–24.8). We decided, for practical reasons, the cutoff point for glucose was 150 mg/dL, 3.65 mEq/L for potassium, 50 for the glucose-potassium ratio, 15,500 for leukocytes, and 18 for lactate.

First, the characteristics of the patients, including if they undergo surgery, were analyzed and compared with the outcome (**TABLE 1**).

Logistic regression analysis was performed by addressing laboratory test values of the blood samples collected at hospital admission and those collected 2 hours after the trauma, and

https://doi.org/10.13004/kjnt.2023.19.e11

TABLE 1. Compared outcome to patient characteristics

Variables	TBI	Good outcome (mRS ≤3)	Poor outcome (mRS ≥4)	OR (95% CI)	*p*-value
Number of patients	47	35 (74.5)	12 (25.5)	-	-
Age (range)	35.70 (16–68)	34 (18–63)	40.67 (16–68)	-	-
Feminine sex	5 (10.6)	3 (8.6)	2 (16.7)	2.13 (0.31–14.62)	0.44
Surgery	41 (87.2)	29 (82.9)	12 (100)	1.41 (1.16–1.72)	0.01
Glycemia >150 mg/dL	25	14 (40.0)	11 (91.7)	16.50 (1.91–142.49)	0.01
Gly/K >50	11	3 (8.6)	8 (66.7)	21.33 (3.95–115.10)	0.00
K >3.65 mEq/L	23	17 (48.6)	6 (50.0)	1.06 (0.29–3.93)	0.93

Values are presented as number (%). Surgery: intracranial pressure monitoring sensor insertion, decompressive craniectomy, removal of hemorrhagic lesions, surgical toilette debridement and frontal sinus cranialization
Gly/K: serum glucose/potassium ratio, K: kalemia, OR: odds ratio, CI: confidence interval, mRS: modified ranking scale.

matched with the outcome of each patient. Glucose-potassium ratios >50 were related to unfavorable outcomes, while values < 50 related to average outcomes (OR, 21.33; 95% CI, 3.95–115.1; p= 0.000). Similarly, glycemic levels >150 were linked to an adverse evolution (OR, 16.50; 95% CI, 1.91–142.49; p= 0.01) likewise age >50 (OR, 4.83; 95% CI, 1.15–20.26; p= 0.03), presence of subdural hematoma (OR, 4.73; 95% CI, 1.17–19.02; p= 0.03), subarachnoid hemorrhage (OR, 6.77; 95% CI, 1.59–28.72; p= 0.01), skull base fracture (OR, 10.85; 95% CI, 2.30–51.10; p= 0.00), and demand for monitoring of intracranial pressure (OR, 13.06; 95% CI, 1.52–112.41; p= 0.02). The remaining indicators did not produce conclusive results. Serum potassium levels are slightly correlated with an unfavorable outcome (OR, 1.059; 95% CI, 0.29–3.93; p= 0.93), as did leukocytes (OR, 1.97; 95% CI, 0.49–7.99; p= 0.34) and lactate (OR, 1.25; 95% CI, 0.32–4.88; p= 0.75), but these results were not significant to the prognosis. The R-square acquired for the model was 0.59 (**TABLE 2**). Finally, we recognized through

TABLE 2. Bivariate and multivariate analysis with outcome results

Variables	No. of patients (n=47)	Bivariate				Multivariate			
		OR	95% CI		*p*-value	OR	95% CI		*p*-value
			Inferior	Superior			Inferior	Superior	
Age >50	12	4.83	1.15	20.26	0.03	32	0.44	12.32	0.32
Feminine sex	5	2.13	0.31	14.62	0.44				
Subdural hematoma	15	4.73	1.17	19.02	0.03	3.51	0.70	17.65	0.13
Extradural hematoma	20	0.19	0.04	0.99	0.05				
Subarachnoid hemorrhage	13	6.77	1.59	28.72	0.01	3.46	0.65	18.35	0.14
Cerebral contusion	20	1.50	0.40	5.61	0.55				
Diffuse axonal injury	6	1.55	0.25	9.77	0.64				
Depressed skull fractura	10	1.33	0.28	6.26	0.72				
Linear skull fractura	22	1.87	0.49	7.05	0.36				
Skull base fracture	11	10.85	2.30	51.10	0.00	4.70	0.85	26.10	0.08
Endocranial hypertension	23	2.67	0.68	10.54	0.16				
Anisocoria	10	1.01	0.62	1.64	0.97				
Mild TBI	6	0.00	0.00	0.00	0.99				
Moderate TBI	21	0.53	0.13	2.09	0.36				
Severe TBI	20	3.83	0.96	15.37	0.06	3.13	0.35	27.85	0.31
Early DC	14	3.38	0.85	13.41	0.08				
Late DC	3	6.8	0.56	83.00	0.13				
Hematoma evacuation	24	0.94	0.25	3.51	0.93				
Surgical toilette	10	0.26	0.03	2.33	0.23				
ICP monitoring	27	13.06	1.52	112.41	0.02	5.46	0.36	82.11	0.22
Glycemia >150 mg/dL	25	16.50	1.91	142.49	0.01	7.52	0.56	100.19	0.13
Gly/K >50	11	21.33	3.95	115.10	0.00	8.61	1.07	69.60	0.04
K >3.65 mEq/L	23	1.06	0.29	3.93	0.93				
Lactate >18 mmol/L	18	1.25	0.32	4.88	0.75				
WBC >15.500/mm^3	23	1.97	0.49	7.99	0.34				

TBI: traumatic brain injury, DC: decompressive craniectomy, ICP: intracranial pressure, Gly/K: serum glucose/potassium ratio, K: kalemia, WBC: white blood cells count, OR: odds ratio, CI: confidence interval.

https://doi.org/10.13004/kjnt.2023.19.e11

the examination of 28 patients who underwent surgery due to intracranial hypertension (either emergency, late, primary, or secondary decompressive craniectomy) that the glucose-potassium ratio could predict the outcome in 23 patients (82%) (OR, 9.44; 95% CI, 1.43–62.24; p= 0.02). Furthermore, 4 of the 6 patients who demonstrated an adverse outcome were either treated clinically or underwent surgery 72 hours after the traumatic incident and revealed a glucose-potassium ratio >50. However, the 15 cases that presented with a non-life-threatening outcome had a glucose-potassium ratio <50.

DISCUSSION

If TBI is considered preventable, with an annual approximate incidence of 1%[3] and generating innumerable direct and indirect health related expenses (nearly 75 million dollars in the US[13]), it emerges as a major concern including not only medical and scientific issues, but also public health care matters.

TBI is a dynamic process, and involves the conversion of kinetic energy into brain parenchymal damage. Primary lesions occur passively because of initial trauma by direct impact or by accelerating and decelerating head movements. Furthermore, these lesions can be classified as diffuse (brain contusions, diffuse axonal injury, cerebral edema) and focal (scalp laceration, fractures, and vascular lesions).[13]

After these initial events, a second process of biochemical events occurs and has been studied primarily in murine models. Several theories have been proposed and one in particular is becoming increasingly widespread. It claims that the effects of trauma threshold action potentials are caused by the release of neurotransmitters (such as glutamate) from neuronal presynaptic membranes, as well as by the activation of N-methyl-D-aspartate ion channel receptors, which results in intracellular calcium and sodium influx, as well as extracellular potassium diffusion. Through energy consumption, a sodium-potassium-ATPase pump accurately and tightly controls this electrolytic imbalance and membrane potential. Furthermore, cerebral glucose intake declines, directly proportionate to TBI severity, and patient age, thereby precipitating hyperglycemia and as a consequence radical hypoglycemic therapies were endorsed in a bid to protect the neuron from glucose toxicity. This is the case for insulin treatment, not yet clinically approved.[13]

The potassium counter effect to trauma is still a concern, as only hyperkalemia has been reported in animal models, but in human assays hypokalemia and hyperkalemia have both been observed, as well as a rebound phenomenon exposing initial hypokalemia followed by increasing levels of serum potassium.[6]

These processes include cerebral edema that is essentially cytotoxic, modified excitotoxicity and voltage changes caused by cellular membrane rupture, apoptosis induced by intracellular glutamate and calcium, altered mitochondrial permeability, DNA damage, free radical release, cellular necrosis, and enzymatic lysis, among others, as secondary lesions in conjunction with cerebral hypoxia.[13]

The glucose-potassium ratio has been reviewed for its prognostic value not only in TBI, but also in aneurismatic subarachnoid hemorrhage,[11,15] intracerebral hemorrhage,[19] and carbon monoxide poisoning.[2] In these cases, it was considered a more accurate prognostic factor

https://doi.org/10.13004/kjnt.2023.19.e11

than glucose and potassium itself. On the basis of this and considering the neurochemical processes, we decided to study its applicability in TBI. Moreover, the lack of publications of this kind in Latin America, as well as the epidemiological contrast between studies from Europe and Asia, prompted us to start our investigation.

As commented before, we have concluded that there is a close correlation between an unfavorable outcome and a value higher than 50 in the glucose-potassium ratio, and between a cipher lower than 50 and benign progression. Furthermore, we found 4 patients with an elevated glucose-potassium index and a poor outcome who were not surgically treated, at least in the first 24 hours after admission, and for whom an early surgical intervention could have reported better prospects in terms of disability.

With regard to the limitations of this study, the retrospective nature of the analysis is acknowledged, as well as the reliance on a limited sample size. Nevertheless, the results correspond to those obtained in similar studies with larger samples. Furthermore, the follow-up for patients was only 6 months, so we did not include long-term complications.

CONCLUSIONS

We aim to contribute to the pursuit of an effective biomarker in TBI to help healthcare professionals make expedient decisions at hospital admission and within the first 3 hours after the trauma occurred. We firmly recommend adopting the use of these biomarkers combined with widely recognized scales, including the Glasgow Coma, Marshall and Rotterdam Scales.[8] In conclusion, in view of the results obtained, and with reference to similar studies, the glucose-to-potassium ratio has been shown to be one of the most promising biomarkers in TBI.

REFERENCES

1. Bergsneider M, Hovda DA, Lee SM, Kelly DF, McArthur DL, Vespa PM, et al. Dissociation of cerebral glucose metabolism and level of consciousness during the period of metabolic depression following human traumatic brain injury. **J Neurotrauma** 17:389-401, 2000
PUBMED | CROSSREF
2. Demirtaş E, Korkmaz İ, Tekin YK, Demirtaş E, Çaltekin İ. Assessment of serum glucose/potassium ratio as a predictor for delayed neuropsychiatric syndrome of carbon monoxide poisoning. **Hum Exp Toxicol** 40:207-213, 2021
PUBMED | CROSSREF
3. Dewan MC, Rattani A, Gupta S, Baticulon RE, Hung YC, Punchak M, et al. Estimating the global incidence of traumatic brain injury. **J Neurosurg** 130:1080-1097, 2019
PUBMED | CROSSREF
4. Hattori N, Huang SC, Wu HM, Yeh E, Glenn TC, Vespa PM, et al. Correlation of regional metabolic rates of glucose with glasgow coma scale after traumatic brain injury. **J Nucl Med** 44:1709-1716, 2003
PUBMED
5. Hovda DA, Le HM, Lifshitz J, Berry JA, Badie H, Yoshino A, et al. Long-term changes in metabolic rates for glucose following mild, moderate and severe concussive head injuries in adult rats. **J Neurosci** 20:845, 1994
6. Katayama Y, Becker DP, Tamura T, Hovda DA. Massive increases in extracellular potassium and the indiscriminate release of glutamate following concussive brain injury. **J Neurosurg** 73:889-900, 1990
PUBMED | CROSSREF
7. Kawamata T, Katayama Y, Hovda DA, Yoshino A, Becker DP. Administration of excitatory amino acid antagonists via microdialysis attenuates the increase in glucose utilization seen following concussive brain injury. **J Cereb Blood Flow Metab** 12:12-24, 1992
PUBMED | CROSSREF

https://doi.org/10.13004/kjnt.2023.19.e11

8. Maas AI, Hukkelhoven CW, Marshall LF, Steyerberg EW. Prediction of outcome in traumatic brain injury with computed tomographic characteristics: a comparison between the computed tomographic classification and combinations of computed tomographic predictors. **Neurosurgery** 57:1173-1182, 2005
PUBMED | CROSSREF
9. MacLeod JB, Lynn M, McKenney MG, Cohn SM, Murtha M. Early coagulopathy predicts mortality in trauma. **J Trauma** 55:39-44, 2003
PUBMED | CROSSREF
10. Marshall LF, Marshall SB, Klauber MR, Van Berkum Clark M, Eisenberg H, Jane JA, et al. The diagnosis of head injury requires a classification based on computed axial tomography. **J Neurotrauma** 9 Suppl 1: S287-S292, 1992
PUBMED
11. Matano F, Fujiki Y, Mizunari T, Koketsu 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** 28:1951-1957, 2019
PUBMED | CROSSREF
12. O'Connell MT, Seal A, Nortje J, Al-Rawi PG, Coles JP, Fryer TD, et al. Glucose metabolism in traumatic brain injury: a combined microdialysis and [^{18}F]-2-fluoro-2-deoxy-D-glucose-positron emission tomography (FDG-PET) study. **Acta Neurochir Suppl** 95:165-168, 2005
PUBMED | CROSSREF
13. Prins M, Greco T, Alexander D, Giza CC. The pathophysiology of traumatic brain injury at a glance. **Dis Model Mech** 6:1307-1315, 2013
PUBMED | CROSSREF
14. Wang RR, He M, Ou XF, Xie XQ, Kang Y. The predictive value of RDW in AKI and mortality in patients with traumatic brain injury. **J Clin Lab Anal** 34:e23373, 2020
PUBMED | CROSSREF
15. Sharma R, Phalak M, Sharma P. Letter to the Editor. Is the serum glucose/potassium ratio a reliable prognostic factor for aneurysmal SAH? **J Neurosurg** 129:1098-1099, 2018
PUBMED | CROSSREF
16. Shibata A, Matano F, Saito N, Fujiki Y, Matsumoto H, Mizunari T, et al. Serum glucose-to-potassium ratio as a prognostic predictor of severe traumatic brain injury. **J Nippon Med Sch** 88:342-346, 2021
PUBMED | CROSSREF
17. Ministry of Health Argentina. Statistical synthesis No. 6 (2020): birth and mortality in 2018. Buenos Aires: Ministry of Health Argentina, 2020 (https://www.argentina.gob.ar/salud/deis/publicaciones) [Accessed May 17, 2022]
18. Svedung Wettervik T, Engquist H, Howells T, Rostami E, Hillered L, Enblad P, et al. Arterial lactate in traumatic brain injury - relation to intracranial pressure dynamics, cerebral energy metabolism and clinical outcome. **J Crit Care** 60:218-225, 2020
PUBMED | CROSSREF
19. Wu XY, Zhuang YK, Cai Y, Dong XQ, Wang KY, Du Q, et al. Serum glucose and potassium ratio as a predictive factor for prognosis of acute intracerebral hemorrhage. **J Int Med Res** 49:3000605211009689, 2021
PUBMED | CROSSREF