The Relationship of Blood Gas Values With Prognosis in Pediatric Patients With Severe Head Trauma

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Abstract

Purpose: Severe head traumas in the childhood age group has an important place in terms of mortality and morbidity among all traumas. Our aim in this study is to consider the relationship between the severity of severe head trauma and arterial blood gas (ABG) parameter levels in children.

Methods: In our study, patients with head trauma, intubation and Glasgow Coma Score (GCS) ≤ 8 were retrospectively analyzed. Showing homogeneous distribution; Independent sample T test was used for pH, PCO₂ and base extract (BE) parameters, and Mann Whitney U test was used for PO₂ and lactate parameters not showing homogeneous distribution. ROC analysis was done.

Results: Of the 48 patients included in the study, 17 died while lying in the intensive care unit. There was no statistical difference when looking at the relationship between age, gender, surgical methods and mortality of the patients ($p > 0.05$). The relationship of patients with acidosis, hypercapnia and hyperlactatemia in ABG with mortality was statistically significant ($p \leq 0.05$). No statistically significant difference was found between surviving and deceased in ABG of patients regarding BE and hypoxia ($p = 0.075$; 0.175). There was a statistically significant difference between the mean values of PCO₂ and lactate which are among the quantitative values and those who survived and died ($p < 0.05$).

Conclusion: Acidosis, the presence hypercapnia and hyperlactatemia were found to be associated with mortality in children with severe head trauma in ABG analysis. This study shows that PCO₂ and lactate levels in ABG can be a biomarker in determining the prognosis in pediatric patients with traumatic brain injury (TBI).

Background

Traumatic injuries are responsible for $> 50\%$ of childhood deaths [1]. Head injuries are the most common among admissions due to trauma in the childhood age group [2]. Deaths due to head trauma comprise 80% of all trauma related deaths [3].

GCS is widely used in head traumas to measure the severity of trauma. According to GCS, 80% of head injuries are mild ($GCS \geq 14$), 10% moderate ($8 < GCS < 14$), and 10% severe ($GCS \leq 8$) [4]. The mortality rate is approximately 30% in patients with severe head trauma [5].

Traumatic brain injury (TBI) is divided into primary and secondary damage according to its physiopathology. Primary TBI, with the effect of mechanical forces; it occurs as a result of damage to neuronal and vascular tissue. Physical damage in the cell membranes causes the homeostasis to deteriorate and the permeability of the membrane to increase. This situation can lead to neuronal edema, hypoperfusion and neurotoxicity. Secondary TBI is defined as the result of process such as ischemia, reperfusion and hypoxia in the damaged areas of the brain in the period after the first injury [6]. The aim of treatment is to correct intracranial and extracranial factors that cause secondary TBI and to minimize brain damage [7].

After head trauma knowing the biomarkers affecting the prognosis in patients progressing TBI makes it easier to determine the treatment priority, to detect patients who may be at risk early, and to predict the clinical outcome [8]. In determining the prognosis in TBI; Studies have been conducted with some biomarkers such as glucose, S100B, lactate, and thiol [9–11].

ABG analysis is an examination that provides an overview of the acid-base balance in patients with TBI and can show the oxygen demand and requirements in the brain [12].

Studies on the prognostic effects of ABG analysis have mostly been done on cardiovascular and respiratory system diseases. Our aim in this study is to evaluate the changes in ABG parameters in pediatric age group patients with severe head trauma and to find their effects to prognosis, if any.

Methods

Ethical approval was obtained from the Ethics Committee of Kafkas University Faculty of Medicine for the study. Patients between the ages of 0–17 who were admitted to the emergency service of the Caucasian University Health Research and Practice Hospital between 2014 and 2019, with a GCS ≤ 8 , with proven intracranial pathology by computed tomography (CT), were retrospectively analyzed.

Patients included in the study had all intubated. The demographic characteristics of the patients, their GCS at the time of admission, values of pH, PCO₂, PO₂, lactate and BE parameters in ABG were obtained by scanning the patient files. Patients with diabetes, electrolyte disturbances, chronic lung diseases, severe hypovolemia, anemia and infections, which will alter blood gas results and severe multiple trauma accompanying head trauma (thoracic, abdominal organ injuries, major bone fractures and cardiac injuries) were excluded from the study. In addition, patients with blood gas results obtained 20 minutes after their admission to the hospital were excluded from the study.

In ABG parameters; pH: 7.35–7.45, PaO₂: 80–100 mmHg, PaCO₂: 35–45 mmHg, lactate level up to 2 mmol / L and BE: up to ± 3 mmol / L were referenced as normal values [13]. In analyzing ABG data; "Medcalc: Acid-base Calculator" calculation software was used. Ph < 7.35 was defined as acidosis, PaO₂ < 80 mmHg as hypoxia, PaCO₂ > 45 mmHg as hypercapnia, lactate > 2 mmol / L as hyperlactatemia. The values of BE $\geq \pm 3$ mEq / L in ABG were accepted as having a base deficit.

Statistical Analysis

The data obtained were analyzed with IBM SPSS Statistics 22 program. Quantitative (quantitative) data were calculated as mean $\pm$ standard deviation (SD). Categorical variables were presented as numbers and percentages. Statistically; Chi-square test was used to evaluate the relationship between categorical variables and mortality. Shapiro Wilk test was applied to find the quantitative values showing homogeneous distribution. In order to evaluate the relationship between quantitative variables and mortality, independent sample T test was used for homogeneously distributed parameters (age, GCS, pH, PCO₂ and BE), and Mann Whitney U test was used for parameters that did not show homogeneous

distribution (PO2 and lactate). ROC Curve (receiver process characteristic curve) analysis was performed for ABG parameters. Using the ROC curve extracted from AUC (area under the curve), an analysis was performed regarding the estimated values of the tested parameters. In all statistical tests, $p < 0.05$ was considered significant. In order to evaluate the effect size of the study, Düsseldorf University's G * Power: Statistical Power Analyzes program was used and Cohen's d test was applied.

Results

In our study, the data of a total of 48 patients were examined. The most common intracranial pathologies were intracerebral hematoma (22.9%), subdural hematoma (16.6%) and traumatic subarachnoid hemorrhage (14.5%), respectively (Fig. 1). The mean age of all patients included in the study was 7.56 ± 4.28 years, 6.29 ± 4.57 years for those who died, and 8.25 ± 4.02 years for those who survived. There were 16 (33.3%) female patients and 32 (66.6%) male patients according to their gender (Table 1). Our mortality rate in all patients was 35.41%. There was no difference in age and gender between the deceased and the survivors. Likewise, there was no statistically significant effect of surgery on mortality or life. The GCS mean of all patients was 6.04 ± 1.68 , the deceased was 4.82 ± 1.7 , and the survivors were 6.70 ± 1.27 . Low GCS values were associated with mortality and were significant.

Table 1: Demographic and clinical data

	All (n=48)	Non Survivors (n=17)	Survivors (n=31)	p value
Boys	32(%66,7)	13(%40,6)	19(%59,4)	0.350
Girls	16(%33,3)	4(%25)	12(%75)	
Mean age	7,56±4,28	6,29±4,57	8,25±4,02	0.149
<i>Boys</i>	7,68±4,36	6,38±4,95	8,57±3,81	0.190
<i>Girls</i>	7,31±4,25	6±3,82	7,75±4,45	0.495
Mean admission GCS	6,04±1,68	4,82±1,7	6,70±1,27	0.000
<i>Boys</i>	6,37±1,66	5,15±1,77	7,21±0,91	0.001
<i>Girls</i>	5,37±1,58	3,75±0,95	5,91±1,37	0.012
Intracranial Operation				
<i>Yes</i>	14 (%29,2)	6 (%42,9)	8(%57,1)	0.478
<i>No</i>	34(%74,8)	11((%32,4)	23(%67,6)	

When we compared the ABG values of living and deceased patients, we obtained many significant statistical results. We found that 14 (53.8%) patients in the deceased group and 12 (46.2%) patients in the surviving group had acidosis. 3 (13.6%) of 22 (45.8%) patients who were normal or alkalosis according to the pH value died, 19 (84.6%) were alive. Mortality rate in patients with acidosis was statistically significant ($p = 0.004$). According to PaO2 values, 8 (47.1%) patients with hypoxia died and 9

(52.9%) patients were alive. According to the PCO₂ values, 13 (61.9%) patients with hypercapnia died and 8 (38.1%) patients were alive. 23 (85.2%) of 27 (56.3) patients with normal PCO₂ or hypocapnia were alive and 4 (14.8) of them died. The presence of hypercapnia differed statistically between patients who died and survived ($p = 0.001$). While 14 (73.7%) patients with high lactate levels died, 5 (26.3%) patients were alive. 26 (83.9%) of 29 (60.4%) patients with normal or low lactate levels were alive. The presence of hyperlactatemia differed statistically between patients who died and survived ($p = 0.000$). 6 (60.0%) of 10 (20.8%) patients with BE died and 4 (40.0%) were alive. BE did not differ significantly between patients who died and survived (Table 2).

Table 2: Proportions and p values of ABG results in living and deceased patient groups

	<u>All</u>	<u>Non- Survivors</u> (n=17)	<u>Survivors</u> (n=31)	<u>OR</u>	<u>%95 CI</u>	<u>p value</u>	
Acidosis							
Yes	26 54,2%	14 29,2%	12 25,2%	7,389	1,748-31,225	0.004	
No	22 45,8%	3 6,3%	19 39,6%				
Hypoxia							
Yes	17 35,4%	8 16,7%	9 18,8%	2,173	0,636-7,420	0.212	
No	31 64,6%	9 18,8%	22 45,8%				
Hypercapnia							
Yes	21 43,8%	13 27,1%	8 16,7%	9,344	2,352-37,123	0.001	
No	27 56,3%	4 8,3%	23 47,9%				
Hyperlactataemia							
Yes	19 39,6%	14 29,2%	5 10,4%	24,267	5,039-116,865	0.000	
No	29 60,4%	3 6,3%	26 54,2%				
BD	Yes	10 20,8%	6 12,5%	4 22,9%	3,682	,867-15,640	0.068
	No	38 79,2%	4 8,3%	27 56,3%			

BD: Base Deficit, OR: Odds Ratio

Quantitative values in ABG differed significantly between patients who died and survived (Table 3).

Table 3: Average values in ABG

	<u>All</u>	<u>Non-Survivors</u>	<u>Survivors</u>	<u>P value</u>
Ph	7,56±0 ,13	7,15±0 ,12	7,35± 0,08	0,00
PO2 (mmHg)	81,66± 15,38	71,17± 17,12	87,41± 10,86	0,02
PCO2 (mmHg)	40,89± 8,03	49,29± 5,59	36,29± 4,75	0,00
Lactate (mmol/L)	3,54± 2,52	6,25± 2,05	2,06± 1,12	0,00
BD (mmol/L)	2,52± 1,56	3,75 ±1,07	1,85 ±1,36	0,00

In the ROC analysis which was made, sensitivity, specificity, 95% confidence interval of sensitivity, 95% confidence interval (CI) of specificity, 95% confidence interval of AUC and AUC were evaluated for ABG parameters in patients who died. Although the presence of acidosis had a statistically significant effect on mortality, no comment could be made on the relationship between pH values and mortality in ROC analysis. In the ROC analysis of PO2 and BE values, AUC values were found below 0.500 (Fig. 2, Table 4).

Table 4
AUC values of ABG parameters in patients who died

Area Under the Curve					
Test Result Variable(s)	Area	Std. Error ^a	Asymptotic Sig. ^b	Asymptotic 95% CI	
				Lower Bound	Upper Bound
PH	0,114	0,058	0,000	0,000	0,228
PO2 (mm Hg)	0,203	0,064	0,001	0,077	0,329
PCO2 (mm Hg)	0,954	0,027	0,000	0,900	1,000
Lactate (mmol/L)	0,952	0,030	0,000	0,892	1,000
BD (mmol/L)	0,847	0,054	0,000	0,741	0,954
The test result variable(s): ph, PO2, PCO2, lactate, be has at least one tie between the positive actual state group and the negative actual state group. Statistics may be biased. a. Under the nonparametric assumption b. Null hypothesis: true area = 0.5					

ROC analysis results of PCO2 and lactate values were valuable (Fig. 2). We found that the values of PCO2 mm 49 mmHg ≥ sensitivity was 58.8% for mortality and a specificity was 96.8%. Likewise, we found that the lactate values of 4.5 mmol / L ≥ had a sensitivity of 82.4% and a specificity of 93.6% for mortality.

The power analysis performed for all quantitative values resulted in Cohen's d score above 0.90.

Discussion

TBI formed after head trauma is one of the main causes of morbidity and mortality in the pediatric age group [14]. Severe head injuries were found to be more common in boys than girls; but there was no statistically significant difference. There was no statistical difference between the genders in terms of age, admission GCS and surgical application. Mortality rate was similar for both boys and girls ($p > 0.05$) (Table 1).

Since brain development continues in children, complete axonal myelization has not occurred and brain tissue has a higher water content than adults. For this reason, brain damage is more common in children, while focal damage is more common in adults. Although children with TBI have a higher survival rate compared to adults, the picture in children is more devastating in terms of sequelae and consequences [15].

The presence of acidosis may cause neural death by disrupting acid-base homeostasis in brain tissue [16]. Therefore, it has been accepted as an important indicator of morbidity and mortality in pediatric patients with TBI [17]. Kushi et al. [18] stated that jugular venous blood pH levels are useful as an early prognostic indicator in the evaluation of neurological function in patients with TBI. pH average values in our study were measured as 7.15 in non-survivors and 7.35 in survivors. The presence of acidosis and mean pH values in the cases that resulted in mortality as a result of TBI were statistically significant compared to the surviving cases.

In patients with TBI, during head injury as the cause of the damage, besides primary brain injury the efficacy of the presence of hypotension and hypoxia was mentioned [19]. Whether surgery is performed or not, adequate oxygenation is important to reduce damage. In children with severe head trauma with oxygen saturation $< 90\%$ or $\text{PaO}_2 < 60 \text{ mmHg}$, firstly, the importance of correcting hypoxia and increasing cerebral perfusion pressure was mentioned [20]. Rapid intubation and mechanical ventilation implementation are recommended to achieve this [21]. The presence of hypoxia has been associated with poor prognosis in pediatric TBI [22]. In a similar study, it has been shown that post-traumatic hypoxia significantly increases the likelihood of mortality in patients with brain damage [23]. Chiaretti et al. [24] reported that post traumatic hypoxia and hypotension are associated with poor outcome. However, there are also studies in which hypoxia is not seen as a factor that statistically affects poor prognosis [25, 26]. In a study involving severe head trauma, no significant statistical result was found in patients with normal PO_2 levels in ABG measured in the emergency department, even if they showed a better prognosis [27].

In our study, the presence of hypoxia did not differ between survivors and those who died. We think that the PO_2 values of our patients were not associated with mortality, since hypoxia was corrected at the time of the first intervention.

Hypercapnia is known to increase cerebral blood flow and volume through cerebral vasodilation; However, its effect on recovery has not been demonstrated directly in humans [6]. It has been stated that it is important to bring PCO_2 to normal as soon as possible in patients with severe head trauma [28]. Dumont et al. [29] studied 65 patients with TBI to determine the effect of prehospital hyperventilation on in-

hospital mortality; found that the survival rate in patients with normocapnia was better than in patients with hypercapnia. He stated that ABG PCO₂ levels can be used as a predictor of outcome in patients with TBI. However, in the study of Rahimi et al. [25] it was shown that hypocapnia or hypercapnia does not have a statistically significant relationship with mortality in children with severe head trauma.

In our study, PCO₂ mean value was 49.29 ± 5.59 mmHg in the deceased and 36.29 ± 4.75 mmHg in the survivors, and the difference between them was statistically significant. The presence of hypercapnia in the patients who died was statistically significant. In the ROC analysis performed to evaluate the relationship of PCO₂ values with mortality, the specificity of $49 \text{ mmHg} \geq$ values of PCO₂ for mortality was 58.8% and its sensitivity was 96.8%. Our study shows that the presence of hypercapnia and high PCO₂ values adversely affect the prognosis.

Lactate is a byproduct of anaerobic metabolism and serum lactate level reflects the degree of tissue hypoperfusion and hypoxia [8]. As the lactate level increases, its use as a cerebral energy fuel increases and this preserves cerebral glucose in patients with TBI [30]. High arterial lactate causes an increase in cerebral blood flow by vasodilation in cerebral vessels [31]. Hypertonic lactate solutions given exogenously may show cerebral edema and intracranial pressure reducing effects. In addition, it has been reported that it may have a neuroprotective effect on high lactate, intracranial pressure, cerebral blood flow and cerebral cellular metabolism [32]. However, some studies have stated that high lactate measured in ABG is associated with increased mortality in critically ill patients and after severe trauma [33, 34]. The role of lactate in TBI is not very clear, a study conducted with adults showed that high arterial lactate may impair cerebral blood flow regulation and thus negatively affect the outcome in TBI [35].

Measuring lactate level in ABG is a fast and practical method compared to serum. When we conducted a literature review, we found that studies investigating the relationship between lactate level in blood gas and determining prognosis in pediatric TBI were insufficient. Ramanathan et al. [36] found that lactate levels were high in pediatric trauma patients hospitalized in the intensive care unit. Similarly, Shah et al. [37] found that the lactate level measured after trauma was high in pediatric patients who needed intensive care. In our study, the presence of hyperlactatemia in non-alive and average lactate values in ABG were statistically significant compared to those who were alive. In ROC analysis, ABG lactate values were found to be a strong indicator for mortality. The specificity of 4.5 mmol / L values of lactate for mortality in pediatric patients with severe head trauma was 82.4% and its sensitivity was 93.6%. Our study shows that high levels of lactate in TBI are associated with mortality.

BE is known as an important indicator of tissue perfusion and hypoxia in trauma patients [38]. It has been stated that BE measurement values are important as a prognostic marker in patients with multiple trauma [39]. However, there are not many studies on BE in TBI. A study mentions the prevalence of surgery in patients with TBI with $\text{BE} > 4 \text{ mmol / L}$ [40]. In our study, we accepted BE values of $4 \text{ mmol / L} \geq$ as significant in terms of the presence of BE. The mean BE values of the non-survivors were higher than those who were alive, but the effect of the presence or absence of BE on the outcome could not be determined.

For patients with TBI, it is necessary to determine some biomarkers for good prognosis, to measure them quickly and to correct pathological values, if any. Therefore, starting the treatment process as soon as possible is important to prevent secondary damage. This study shows that some parameters in early period ABG in pediatric TBI can be a biomarker for prognosis.

Conclusions

We found that high PCO₂ and lactate levels and the presence of acidosis in ABG in the pediatric age group with severe head trauma were associated with mortality. Determination of reliable outcome determinants after head trauma is especially important for clinicians. Acidosis, hypercapnia and hyperlactatemia in ABG in patients with TBI in the pediatric age group may be an indicator for poor prognosis.

Limitations

Our study has some limitations, the first of which is the small patient population. The other limitation is that it is a retrospective study. In addition, patients with multiple trauma accompanying severe head injuries are excluded from the study. Finally, its limitation is the arterial condition for blood gas, and venous blood gas is not accepted.

Declarations

Ethics approval and consent to participate

Prior to the study, the ethics committee approval dated 25.06.2020 and numbered 233 was obtained from the Ethics Committee of Kafkas University Faculty of Medicine. Since this study was designed as a retrospective study, no written consent was required from patients [from their parents or guardians]. Since it does not contain personal medical data, it does not require written consent in retrospective research according to institutional / national laws and the Helsinki Declaration of the World Medical Association.

Consent for publication

Not applicable.

Availability of data and materials

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

L.S. designed, supervised the study and wrote the article; M. K. did the literature review, analyzed the biochemical parameters, collected the data and designed the table and wrote the article.

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Figures

Figure 1

Type of Brain Trauma Percentage of Patients. SDH: Subdural hematoma (8), EDH: Epidural hematoma (6), Contusion (5), SAH: Subarachnoid hemorrhage (7), IVH: Intraventricular hemorrhage (6), ICH: Intracerebral hemorrhage (11), Brain Edema (5)

Figure 2

ROC curve for the values of ABG parameters