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Research Article
Use of Base Excess Value and the Blood Lactate Level in Predicting Organ Disfunction Measured by Sequential Organ Failure Assessment (SOFA) Score System: Study in the Post Trepanation Patients with Severe Traumatic Brain Injury

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Abstract

Background and Objective: Prediction of organ failure is important in the management of patient in Intensive Care Unit (ICU). This study was aimed to compare blood base excess value and blood lactate levels among patient with and without organ dysfunction. **Materials and Methods:** Twenty eight subjects aged 15-64 years who have undergone trepanation procedure due to severe traumatic brain injury involved in this study. Base excess value and blood lactate levels were measured shortly and 12, 24, 48 h after arrived in Intensive Care Unit (ICU). The presence of organ dysfunction was measure by Sequential Organ Failure Assessment (SOFA) score system. The difference of base excess value and blood lactate levels between patient with and without organ dysfunction was compared with Mann Whitney tests and the association between base excess with SOFA scores was calculated by chi-squire test. **Results:** The blood lactate value in patient with organ dysfunction was significantly higher after 24 and 48 h compared with patient without organ dysfunction ($p = 0.008$ and $p = 0.035$, respectively). The difference of base excess value can only be seen after 48 h ($p = 0.048$). A positive association was found between abnormal base excess and organ dysfunction ($p = 0.04$). **Conclusion:** Base excess value and the blood lactate levels can be used as predictor to determine the presence of organ dysfunction measured by SOFA scores.

Key words: Base excess value, blood lactate levels, post trepanation, traumatic brain injury

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Data Availability: All relevant data are within the paper and its supporting information files.

INTRODUCTION

Traumatic brain injury is a health problem that can lead to the disability and death. Although, the incidence is very high, however, it is termed as a global epidemic in disguise. Every year, about 1.5 million people died as a result of traumatic brain injury and several million others have to undergo the treatment in the hospital. It is the rationale why the management of the patients sometimes make treatment decisions based on the prognosis assessment.

A survey conducted in 2005 states that 80% of doctors believe that an accurate assessment of the prognosis is very important when they make decisions about the use of specific methods to the therapy, for instance the use of hyperventilation, barbiturates or mannitol. The same size will be selected when taking important decisions to continue or stop the aggressive therapy.

To maximize the life expectancy rate, priority should be focused on the therapeutic shock resuscitation. Inadequate oxygen network will lead to an anaerobic metabolism and develop into tissue acidosis. Resuscitation is considered complete when the lack of oxygen has been fulfilled, tissue acidosis has been lost and aerobic metabolism has been reached in all networks[1].

When there is an imbalance between demand and supply of oxygen, the anaerobic respiration started to happen that developed into a metabolic acidosis. This metabolic acidosis can be measured directly via an arterial blood gas analysis through the examination of base excess and lactate. Base excess can be used as an indicator of a decrease in oxygen delivery and clinical serves as an indicator of compensated shock. Lactate and base excess can reliably predict the outcome of the patients with head injury but it cannot be used as an indicator to determine the patient's head injury, closed head trauma[2].

Damage to the brain tissue cause the mitochondrial dysfunction in the Central Nervous System (CNS) and deformity of the membrane. This process inhibits the pyruvate entering to the Krebs cycle and produces the lactate accumulation of intra-cellular and extra-cellular. Decreasing immediately on the cerebral blood flow and cerebral ischemia on the threshold level may lead to an increase in lactate in the brain tissue. If an increase in lactate settled in the brain tissue and cerebrospinal, then it will worsen the prognosis.

A high increased lactate levels in alba substance in contusions suffered fast vasogenic edema after the experimental head trauma occur in animals cobs. Patients with bad outcomes in head trauma have a higher significant ventricular lactate levels compared to the patients with moderate disabilities or good outcomes.

An increase in cerebral lactate is one of the predictors of mortality in patients with severe head injury. However, the availability of evidence on the relationship of cerebral lactate concentration, base excess to the severity of head injuries as well as the ability to reflect the lactate arterial lactate levels in brain tissue and cerebrospinal until now have not been studyed adequately[2].

Many factors can affect the condition of the patients during being care in the ICU. Therefore, various assessments that can evaluate the changes in the patients during being treatedare needed but it must still be based on the existing evaluation models. The use of the assessment system as an aid in making decisions is logical and advisable but a system that has been validated should be chosen.

At this time, there are various models of the scoring system that can be used to estimate the mortality of the patients, such as the acute physiology and chronic health evaluation (APACHE), Simplified Acute Physiology Score (SAPS), Mortality Probability Models (MPM), Sequential Organ Failure Assessment (SOFA), Multi Organ Dysfunction Score (MODS) and Logistic Organ Dysfunction Score (LODS) that assessed and quantified in the first 24 h when the patients were admitted to the ICU[3].

The APACHE II scores correlate well with the mortality risk patient population in the ICU but it does not have the accuracy to predict the mortality in individual patients. One of the simpler value systems developed is the SOFA score that assesses six organ systems. In addition, the accuracy and precision of the SOFA score assessment have been recognized by a number of clinicians. The SOFA score is more accurate in predicting mortality due to have better discrimination and calibration compare to APACHE II score[3].

Severe neurologic injury increases the risk factor to develop into MODS. It is important to know that the development of non-neurologic organ dysfunction that is not related to the severity of neurologic injury has been associated with bad outcomes in the patients with subarachnoid hemorrhage and severe head injury[3].

Based on this background, the purpose of this study was to determine the changes dynamic of the Base Excess (BE) value, blood lactate levels and the SOFA score to the post trepanation patients with severe traumatic brain injury who were treated in the ICU.

MATERIALS AND METHODS

Study site: This study was conducted in October-December, 2013 in Intensive Care Unit (ICU) of several teaching hospitals of Hasanuddin University in Makassar.

Design and study variables: This study is a prospective cohort study to assess the correlation of the dynamics of changes in the value of BE, blood lactate levels and the SOFA score to the post trepanation patients with severe traumatic brain injury who were treated in the ICU. The study variables consisted of variable X (base excess levels and blood lactate levels), Y (SOFA score) and the control variables (increase in serum transaminase, decreased filtration rate glomerulus, age, sepsis, hip albumin and drugs).

Population and sample: The populations in this study were all the patients admitted to the ICU of Central General Hospital of Dr. Wahidin Sudirohusodo Makassar and the networking diagnosed postoperative trepanation with severe traumatic brain injury. Samples were all affordable populations diagnosed as postoperative trepanation with severe traumatic brain injury who met the study criteria. The sampling technique performed consecutive sampling until the number of the samples met. A number of samples in this study were 9 people.

Method of data collecting: Data collection method in this study was by providing an explanation to the family about the purpose of the study. If the patient was willing to follow the study, the patient was asked her/his willingness to sign an agreement to follow the study. Another method was to collect the family history (alloanamnese) so that, the respondent data includes identity, course of the disease, a history of previous disease, previous treatment history and the presence of comorbidities that can be obtained. It was collected by doing a physical examination, such as blood pressure, pulse, respiratory rate, temperature and GCS and doing some investigation laboratory tests, such as leukocytes, platelets, SGOT, SGPT, total bilirubin, creatinine, blood gas analysis, blood lactate levels, BE and assessment of the SOFA score. The checkup was carried out on when the patients did the admission to the ICU care after being diagnosed of severe traumatic brain injury. The checkups of blood lactate with portable whole-blood lactate using accu check lactate (Roche) as tool and base excess in the checkup of the blood gas analysis were performed in the laboratory.

Data analysis technique: The statistical analysis used SPSS 20 for windows. The test used was Fisher's exact test.

RESULTS

There are differences in the dynamics of blood lactic acid levels between the groups experiencing the organ dysfunction for 48 h after the treatment and the group that did not experience the organ dysfunction. In the group without the organ dysfunction, the blood lactic acid levels decreased. While the group of the dysfunctional organs, blood lactic acid levels increased. Differences in the dynamics of the blood lactic acid levels differ significantly ($p<0.05$) at the time of the observation of 24 and 48 h after the treatment (Table 1).

Table 1: Dynamic comparison of blood lactic acid levels between patient with and without organ dysfunction during 48 h observation in the intensive care unit

	Median levels of blood lactic acid		
Observation time (h)	With organ dysfunction (N = 14)	Without organ dysfunction (N = 14)	p-value
Beginning	3.55	3.30	0.721
12	3.55	1.90	0.505
24	4.05	1.45	0.008
48	5.24	0.90	0.035

Table 2: Dynamic comparison of the blood base excess values between patient with and without organ dysfunction during 48 h observation in the intensive care unit

	Median blood base excess		
Observation time (h)	With organ dysfunction (N = 14)	Without organ dysfunction (N = 14)	p-value (Mann-Whitney)
Beginning	-5.15	-5.00	0.878
12	-4.80	-3.50	0.574
24	-4.35	-2.50	0.079
48	-3.68	-1.00	0.046

There are differences in the dynamics of base excess values between groups experiencing the organ dysfunction for 48 h after the treatment and the group that did not experience the organ dysfunction. In the group without the organ dysfunction, base excess values decreased from -5.0 become -3.50 and continue to be -2.50 became -1.0 during 48 h after the treatment. While, the group of the organ dysfunction also experienced a decrease BE but it was not for those who did not experience the organ dysfunction. Base excess values were significantly different ($p<0.05$) at the time of the observation for 48 h after the treatment (Table 2).

There were differences in the dynamics of blood lactic acid levels between the groups that have abnormal base excess value and the groups with normal base excess values. In the group of abnormal base excess, blood lactic acid levels increased from 3.00-5.00 and continued to be 5.76 and 8.80 at 48 h after the treatment. Whereas, in the group of normal base excess, blood lactic acid levels decreased from 4.00-2.20 and continued to be 2.00 and 1.40 at 48 h after the treatment. There were significant differences in the dynamics of blood lactic acid levels ($p<0.05$) at 12 h observation, 24 and 48 h after the treatment (Table 3).

There were differences in the dynamics of blood lactic acid levels between the groups with normal and abnormal

Table 3: Dynamic comparison of the blood lactic acid levels between patient with normal and abnormal blood base excess during 48 h observation in the intensive care unit

	Median blood lactic acid levels		
Observation time (h)	Abnormal blood base excess (N = 7)	Normal blood base excess (N = 7)	p-value (Mann-Whitney)
Beginning	3.00	4.00	0.860
12	5.00	2.20	0.020
24	5.76	2.00	0.000
48	8.80	1.40	0.000

Table 4: Association between blood base excess in the first 24 h observation and the presence of organ dysfunction measured by SOFA scores after 48 h observation in the intensive care unit

	SOFA		
Base excess	Without organ dysfunction N = 4 (%)	With organ dysfunction N = 14 (%)	p-value (Fisher exact test)
Normal	3 (75.0)	2 (14.3)	0.040
Acid	1 (25.0)	12 (85.7)	

base excess at 48 h after the treatment. The dynamics of blood lactic acid levels decreased from the initial state to 48 h after the treatment in the group of normal base excess. While, the group with abnormal base excess increased.

There was a relationship between the blood lactic acid levels and BE. Increased blood lactic acid levels in the range of 0.0-5.0 showed a rapid decrease of the value of base excess from positive (4) to negative (7); whereas an increase in blood lactic acid levels from 5.0-15.0 did not change the value of base excess (about negative 7) and an increase in blood lactic acid levels from 15.0-20.0 rapidly lowered the value of base excess of about negative 7 to negative 16.

There was a significant relationship ($p<0.05$) between the base excess 24 h after the treatment and the SOFA score 48 h after the treatment. If the value of the base excess was normal, the potency to not experience the organ dysfunction was 75%. Meanwhile, when the value of base excess was acid, the potential to not experience the organ dysfunction was small (14.3%). The Fisher's exact test results demonstrated the p-value was 0.040 (Table 4).

DISCUSSION

This study showed that there was a significant relationship between the base excess and the SOFA score. Furthermore, there was a significant difference from the dynamics of the levels of lactate and the values of base excess tothe post trepanation patients with severe traumatic brain injury admitted to the ICU between the groups experiencing and not experiencing the dysfunctional organ and between the groups of normal and abnormal base excess. The dynamics of changes in the base excess value and the blood lactate levels were in line with the dynamics of the SOFA score.

The organ dysfunction is a condition that describes the development of the organ function impairment and a dynamic state. Traumatic brain injury is one injury or trace physiological and pathological that can lead to the organ dysfunction. Currently, various theories have been used to explain the theory of the organ dysfunction. One is the hypothesis that the actual supply adequate oxygen to the cells but the oxygen cannot be used by the cells as a result of abnormalities in mitochondrial oxidative phosphorylation pathway[4].

Traumatic brain injury is usually followed by an increase in basal metabolism and energy which is equivalent to the severity of the injury. The energy is obtained through the endogenous tissue deposits by increasing the speed of gluconeogenesis, glikogenilisis and proteolysis[5].

The severity of illness to the patients undergoing treatment in the ICU can be measured through the number of the organ failure inserted into each organ dysfunction severity. The SOFA score is made to evaluate the organ failure that focuses on the morbidity than mortality[6].

At the beginning, the SOFA score was made for sepsis patients. But at the end, its use is not only for the group but also to evaluate the presence or absence of the infection[7].

The SOFA score was formed through a process of consensus and then approved by the larger population of the ICU patient. At the first, the SOFA score was used to describe the outcome of the patients with sepsis but in recent decades, the SOFA score can also be used for other conditions. A progressive increase in total SOFA score is a marker of bad outcome in the daily evaluation of ICU patients and 48 h after the treatment. A very significant increase in SOFA score was as a marker of the patient's outcome. It was found that based on the presence or absence of the organ dysfunction, there was a significant difference ($p<0.05$) of blood lactic acid levels during 24 h of observation and 48 h after the admission to the ICU.

Traumatic brain injuries can cause metabolic changes in the brain. This metabolic change is a picture of changes in the brain energy metabolism in response to the trauma. The release of the stress hormones, such as catecholamine and adrenaline will cause an increase in metabolic rate. Increased lactate levels reflect the imbalance between the demand and supply of the energy. In addition to the influence of the brain tissue damage due to the trauma, an increased lactate levels is also affected by the changes in the chain of pathophysiological complex, such as increased intracranial pressure, impaired perfusion, damage the metabolism,

inflammation, secretion of neurotransmitter excitation and impaired balance of ionic that finally exacerbate the brain energy metabolism.

Increased lactate levels on the second and the 7th day after the trauma showed that the increased lactate levels was not only influenced by the hypoxia but also influenced by the oxidative phosphorylation disorders due to the tissue damage from the trauma. At the first, it was also affected by the inflammation, catecholamine and the response to adrenaline[4].

Base excess is the amount base in mill moles unit needed to titrate 1 L of arterial blood that has a pH of 7.40 with full oxygen saturation at a temperature of 370°C and $PaCO_2$ by 40 mmHg[8]. Base excess was related to the needs of intravascular fluid and mortality to the patients with pelvic fractures, liver trauma and hypovolemic shock. The relationship between hypervolemia and base excess allegedly was caused by the hypo perfusion and dysoxia.

There is a statistically significant relationship between the base excess and the total SOFA score, where the value of base excess can be used as a picture of the severity of illness in ICU patients. In describing the correlation between clinical outcome and severity of the disease, base excess can be trusted as a diagnostic tool metabolic acidosis[9].

Hyperlactataemia can be caused by the tissue perfusion, which is inadequate so the oxygen demand increased caused by the exposure to the endotoxin, the inhibitory activity of pyruvate dehydrogenase and increased anaerobic glycolysis, which lead to the lactate and pyruvate accumulation in the cells disrupting the oxidative phosphorylation leading to the increased release of lactate into circulation[10].

Hyperlactataemia can occur with or without the metabolic acidosis. Hyperlactatemia arise when the conditions are still in good tissue perfusion, for instance the release of catecholamine, alkalosis or increased metabolic activity due to the sepsis or burns, then the buffer mechanism can compensate for any drops in pH. When the lactate levels increase due to the poor tissue perfusion, the buffer system is unable to handle so the acidosis happens. The ICU patients often experience a variety of the mechanisms behind the causes of hyperlactatemia arising from two types of the main mechanisms mentioned above[11].

The organ failure and sepsis arise very often in the second and the third day care in the ICU. At least, the improvement of the organ failure when treating is associated with the increased mortality and morbidity that are usually measured by the SOFA score. However, the specific mechanism associated with the organ failure that can lead to the death is not known yet certainly[10].

Base excess and blood lactic acid levels at the beginning of the measurement have better prognostic value. The best predictive value of early base excess is when the base excess is less than -4 mmol L^{-1} and in accordance with the blood lactic acid levels that is greater than 1.5 mmol L^{-1}. The combination of both markers at the beginning of ICU admission have a sensitivity of 80.3% and a specificity of 58.7% for determining the mortality. The combination of these two markers is associated with the increased mortality, aging, increased need for inotropic support and an increase in the organ failure scores, both during the first time ICU or the next 24 h[10].

This is consistent with our study that showed a significant relationship ($p<0.05$) between the base excess for 24 h after treatment and the SOFA score for 48 h after the treatment. If the value of the base excess was normal, the potency to not experience the organ dysfunction was big enough for 75% and the rest 25% potentially experienced the organ dysfunction. Meanwhile, when the value of base excess was acid, the potential to not experience the organ dysfunction was very small (14.3%) or 85.7% had the first degree organ dysfunction.

CONCLUSION AND RECOMMENDATIONS

The studyers concluded that there was a significant relationship between the base excess value and the SOFA score and there was a significant difference from the dynamics of lactate levels and the value of base excess to the post trepanation patients with severe traumatic brain injury admitted to the ICU between the groups experiencing and not experiencing the dysfunctional organ and between groups with normal and abnormal base excess. The dynamics of changes in the value of base excess and blood lactate levels were in line with the dynamics of the SOFA score. When the value of the base excess and blood lactate levels were normal, the organ dysfunction is not potentially occurred. The studyers suggest that further study on the relationship of the changes dynamic in the base excess value and the blood lactate levels on the incidence of each organ system dysfunction were assessed on the SOFA score.

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