

Base deficit and serum lactate concentration in patients with post traumatic convulsion

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ABSTRACT

Introduction: Traumatic brain injury is a major cause of morbidity and mortality worldwide, and has been reported to be one of the risk factors for epileptic seizures. Abnormal blood lactate (LAC) and base deficit (BD) reflects hypoperfusion and could be used as metabolic markers to predict the outcome. The aim of this study is to assess the prognostic value of BD and LAC levels for post traumatic convulsion (PTC) in head injury patients.

Materials and Methods: All head injury patients with PTC were studied for the demographics profile, mechanism of injury, initial vital signs, and injury severity score (ISS), respiratory rates, CT scan findings, and other laboratory investigations. The data were obtained from the trauma registry and medical records. Statistical analysis was done using SPSS software.

Results: Amongst 3082 trauma patients, 1584 were admitted to the hospital. Of them, 401 patients had head injury. PTC was observed in 5.4% (22/401) patients. Out of the 22 head injury patients, 10 were presented with the head injury alone, whereas 12 patients had other associated injuries. The average age of the patients was 25 years, comprising predominantly of male patients (77%). Neither glasgow coma scale nor ISS had correlation with BD or LAC in the study groups. The mean level of BD and LAC was not statistically different in PTC group compared to controls. However, BD was significantly higher in patients with associated injuries than the isolated head injury group. Furthermore, there was no significant correlation amongst the two groups as far as LAC levels are concerned.

Conclusion: Base deficit but not lactic acid concentration was significantly higher in head injury patients with associated injuries. Early resuscitation by pre-hospital personnel and in the trauma room might have impact in minimizing the effect of post traumatic convulsion on BD and LAC.

Key words: Base deficit, convulsion, head injury, lactic acid, trauma

Background

Studies performed over the past two to three decades have reported that the incidence of clinical seizure activity after brain injury ranges from 5 to 15%. Focal and generalized clinical seizures occur with greater frequency in patients, who are more severely injured, with a frequency of 8 to

10%.^[1-3] Early post traumatic seizures are thought to be due to mechanical damage to the neurons, caused by extravasated blood. Head trauma initiates a sequence of responses that includes altered blood flow and vasoregulation, disruption of blood brain barrier, increase in intracranial pressure, focal or diffuse ischemic hemorrhage, inflammation, necrosis, and disruption of fiber tract and blood vessels.^[4] The reports have also suggested that iron (hemorrhage) induced neuronal lipid peroxidation and excitotoxicity could be a probable mechanisms, involved in the post traumatic epilepsy.^[5]

Generalized seizure can produce rhabdomyolysis, apnea, hypoxia, metabolic acidosis, and neural damage. Extreme muscle activity can lead to lactic acidosis; while catecholamine excess can cause hyperglycemia which can further exacerbate acidosis through anaerobic metabolism.^[6] Lactic acidosis and rhabdomyolysis are reported, although under diagnosed, accompaniments of generalized convulsions.^[7-9] In trauma patients, the admission value of arterial base deficit stratifies injury severity predicts complications, and is correlated with arterial lactate

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concentration.^[7-9] Herein, the present study evaluate the effect of post traumatic convulsions of head injured patients on the sensitivity of base deficit (BD) and lactic acid concentration (LAC).

Materials and Methods

All post-traumatic convulsion patients admitted to the Trauma Intensive Care Unit (TICU) at Hamad General Hospital, Doha-Qatar (Level 1 trauma center) from January 2008 through January 2010 were included in the study. A control group of the same injury severity score (ISS) were also selected randomly for comparison. Patients identified as having clonic activity of an extremity or hemi body and tonic convulsion were selected for the study. Other types of convulsions as described by EMS such as eyelid fluttering, lip smacking, and episodic staring associated with motor automatisms were excluded from the study. All patients were studied for the demographics, mechanism of injury or heart rate (HR), blood pressure (BP), respiratory rate (RR), body temperature, oxygen saturation (SaO₂); Glasgow Coma Score (GCS); description of post traumatic convulsion in the prehospital course and upon admission to trauma resuscitation room; ISS and detailed injuries based on computed tomographic (CT) scan findings. Laboratory investigations such as Hg, platelet, creatinine, INR, glucose, pH, BD, LAC (initial and follow up); fluid resuscitation; blood and ventilatory requirement were also assessed. All data were collected from the trauma registry and medical records. The study was approved by the medical research center, HMC (IRB#10052/10).

Descriptive statistics i.e., mean and standard deviation, for continuous variables and frequency distribution for categorical variables were calculated. Statistical techniques like Chi-square test and correlation coefficient were applied. $P \leq 0.05$ was considered as statistical significance. SPSS 18.0 statistical package was used for the analysis.

Results

Of the 3082 trauma patients presented to our trauma center, 1584 patients were admitted; of them, 401 patients had head injuries. Amongst the head injury patients, 70.8% (284/401) had GCS > 8 and the remaining 29.2% (117/401) patients had GCS < 8. During the study period, 22 of 401 (5.4%) patients were identified with post traumatic convulsion. Post traumatic convulsion occurred at scene in 12 patients and in trauma room in 13 patients (three patients had their second attack in the trauma room).

Average age of the patients was 25 years, and majority was male patients (77%). In thirteen patients, mechanism of injury was motor vehicle crash (MVC), seven patients were pedestrian struck by a car, and two had fallen from height. Seventeen patients (77%) were intubated for low GCS or to control convulsions and protect airway. ISS was <14 in 7 (32%) patients and >14 in 15 (68%) patients.

Convulsions were described at scene by EMS personnel in 12 patients, where all of them intubated to control convulsions associated with low GCS, whereas convulsions described in 15 patients in trauma room (2 in CT scan room) and 5 patients intubated after drop of the initial GCS.

All patients were hemodynamically stable with no blood transfusion requirement. Only two patients required an operation for depressed skull fractures and two patients were operated for evacuation of hematomas with midline shift. Head injuries were concussion in seven patients with facial bone fractures in two of them, intracranial hemorrhages and contusions in thirteen patients, and diffuse axonal injury was present in five patients [Table 1].

Associated injuries were long bone fractures in four patients, blunt chest trauma in five patients (one with significant injury), C-spine injury in two patients with no neurologic deficits, and one patient with GI splenic injury. There was no significant statistical correlation observed between GCS and ISS, neither between GCS or ISS with BD or LAC in the study groups [Figures 1 and 2].

Statistically non-significant difference was observed amongst the mean ISS ($P = 0.08$), BD ($P = 0.14$) and LAC ($P = 0.27$) values in post-traumatic convulsion group compared to controls [Figure 3]. However, BD was significantly higher in patients with associated injuries compared to the isolated head injured group [$P = 0.03$; Table 2; Figure 4]. On the other hand, mean LAC values were not statistically different between the two groups [$P = 0.21$; Table 2].

Discussion

Traumatic brain injury is a significant cause of morbidity and mortality worldwide and has been reported to be one of the major risk factors for epileptic seizures. Posttraumatic epilepsy following severe head injury characterized by recurrent

Table 1: Diagnostic profile of 22 head injury patients, based on CT scan

Diagnosis	Number of lesions*	Operations
Concussion	7	
Skull fracture	7 (depressed in 3 patients)	2 bone elevation
Skull base fracture	3	
Fracture facial bones	5	
Hematomas		
EDH	3	1 craniotomy with evacuation
SDH	5	1 craniotomy with evacuation
SAH	3	
Diffuse axonal injury	4	
Hemorrhagic contusions	10	
Total	22	

EDH – Epidural hematoma; SDH – Subdural hematoma; SAH – Subarachnoidal hematoma, *One patient may have more than one lesion

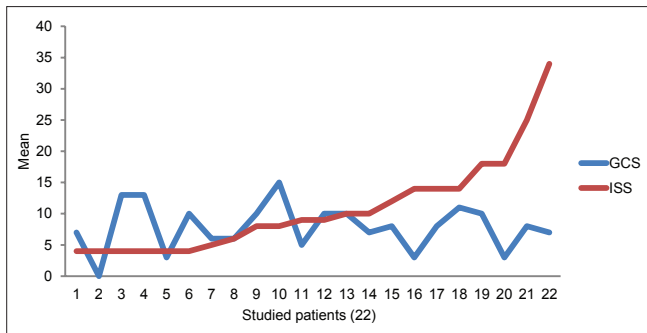


Figure 1: The correlation between GCS and ISS amongst head injury patients

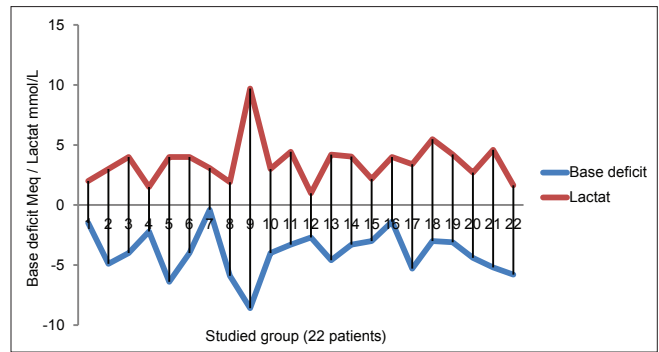


Figure 2: The correlation between BD and LAC levels in head injury patients

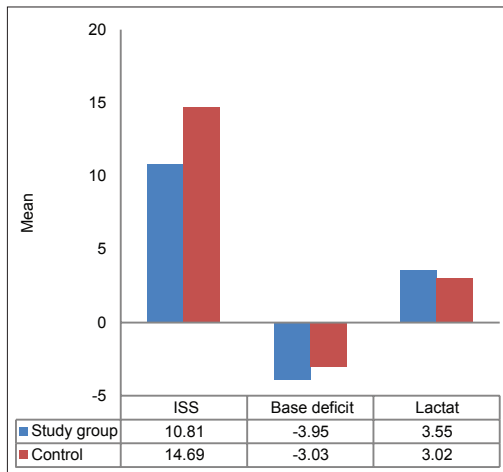


Figure 3: Comparison of mean ISS, BD and LAC values in post-traumatic convulsion group compared to controls

Table 2: Comparison of mean BD and LAC values among patients with isolated head injury and with associated injuries

Parameters	Group 1* n=12	Group 2# n=10	P value
BD (meq)	-4.62	-2.98	0.03
LAC (mmol/L)	3.75	2.67	0.21

*Group 1 – Mean BD values in patients with associated injuries; #Group 2 – Mean BD values in isolated head injury patients

epileptic seizures that complicates the management of head injuries by increasing the intracranial pressure and altering the level of unconsciousness.^[10,11]

Depending on the time interval following head trauma, the epilepsy can be classified into three major types; impact, early and late seizures. Impact seizures are sometimes considered as subgroup of early seizures and occur immediately after or within 24 hours post injury. Early seizures are acute symptomatic seizures and appear within one week after injury. Late seizures might have a chronic course and occur after the recovery from the acute effects of the injury.^[2,3,11,12]

The identification of seizure activity in patients with moderate-to-severe brain injury who are in coma is more

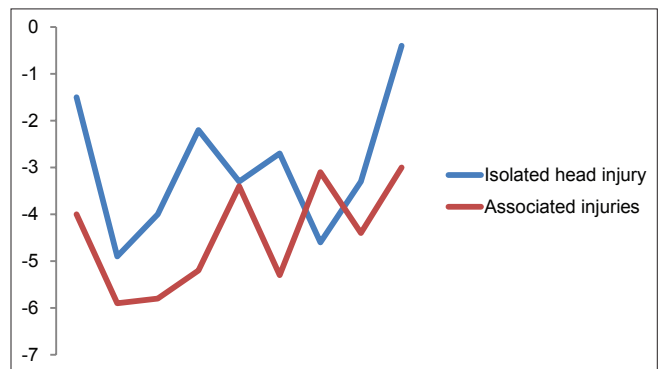


Figure 4: Correlation of BD and LAC values among patients with isolated head injury and with associated injuries

difficult. To date, most reports of seizure frequency in this group have been based on documenting clinical events. However, a few reports of subclinical or non-convulsive seizures suggest that the incidence of seizures may be underestimated.^[13]

Several risk factors complicate the posttraumatic epilepsy, for example neural location, agent of injury, severity, missile injuries, loss of consciousness, intra-cerebral hemorrhage, diffuse cerebral contusions, presence of focal neurological deficits and prolonged (>3 days) post traumatic amnesia.^[11] The main clinical risk factors for seizures are younger age, greater severity of brain injury and subdural hematoma and penetrating wounds.^[13]

Early post traumatic seizure is explained by the increase in the extracellular levels of potassium and a consequent increase in glucose utilization that fuels ionic pumps to re-establish homeostasis, called “hyper glycolysis”, which is accompanied by a concurrent reduction of cerebral blood flow (CBF) which creates a state of vulnerability to secondary insults.^[1,14-20]

The mechanism of lactic acidosis following seizures is thought to be the consequence of local muscle hypoxia and increased production of pyruvic acid, which is normally in equilibrium with lactic acid. Resolution is thought to occur predominantly due to aerobic metabolism leading to reduced production of lactic acid and enhanced gluconeogenesis, although renal clearance may

also be a factor. In this regard, the elevation of plasma creatinine and creatine kinase may reflect transient rhabdomyolysis-induced renal impairment which could have delayed the return of normal serum lactic acid levels to >2 days.^[8,21]

The degree of acidosis and muscle damage associated with post traumatic convulsion might have led to inappropriate therapy with bicarbonate, since the acidosis resolved following supportive therapy with oxygen, intravenous saline and anticonvulsants.^[7-9]

Abnormal blood lactate and BD reflect hypoperfusion, and several studies have documented the ability of these metabolic markers to predict outcome in trauma. It has been reported earlier that abnormal blood lactate concentrations correlate with injury severity, poor cardiac performance, increased mortality, multiple system organ failure, respiratory complications, and hospital length of stay in trauma.^[22] Furthermore, normalization of abnormal blood lactate concentration within 24 hours has been associated with improved survival after trauma.^[23] The initial base deficit has also been documented as a predictor of outcome in trauma, including mortality, ICU and hospital LOS and the need for blood product transfusions in trauma. In addition, BD has been shown to predict the presence of intra-abdominal injury, ongoing hemorrhage, the volume of fluid required for resuscitation and the development of multiple organ failure.^[24-28]

In our study, no significant correlation of post traumatic convulsion group with the control group was observed with respect to BD and LAC. However, we found BD to be significantly higher in head injured patients with associated injuries.

Winocour and his associates reported low potassium as important hallmark of metabolic acidosis secondary to lactic acidosis. Authors concluded that severe lactic acidosis and extensive muscle damage can accompany status epilepticus; these disturbances may be self-limiting and do not require specific corrective therapy.^[29]

Furthermore, Orringer and his colleges reported a severe metabolic acidosis following a single grand-mal seizure of 30 to 60 seconds duration, which might be the consequence of suppression of the respiratory center after the convulsion.^[7]

The present study found that the BD can be a good indicator for diagnosis and treatment of shock in head injury patients and could be a marker of impaired oxygen utilization which is consistent with earlier studies.^[30,31] In a recent study, Herbert *et al.*,^[32] lactate was not shown to be an independent predictor of mortality, which is similar to our observation.

Conclusion

BD and LAC are still accurate indicators of injury severity in trauma patients. In our study, population with post-traumatic

convulsions BD, but not LAC, was significantly higher in head injured patients with associated injuries. Early resuscitation started from the scene by pre-hospital skilled personal and in the trauma room might have impact in minimizing the effect of post traumatic convulsion on BD and LAC.

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