

A Study of Acid Base, Electrolyte and Haemodynamic Status of Status Epilepticus Patients Among Children in a Tertiary Care Hospital of West Bengal

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Abstract:

Introduction: Status epilepticus (SE) is a life-threatening neurological emergency characterized by prolonged or recurrent seizures without recovery of consciousness. The condition is associated with significant disturbances in acid–base balance, electrolytes, and haemodynamic parameters, which may worsen neuronal injury and increase morbidity and mortality. Early recognition and correction of these derangements are crucial in the comprehensive management of SE.

Methods: The present study was a prospective observational study conducted at Burdwan Medical College and Hospital, Bardhaman, West Bengal (PIN 713104) over a period of one year, from August 2021 to July 2022. The study population comprised all patients aged between 3 months and 12 years presenting with status epilepticus (SE) who fulfilled the inclusion criteria. A total of 81 patients with SE were enrolled during the study period.

Results: Among 81 patients with status epilepticus (mean age 32.4 ± 15.7 years; 59.3% male), generalized seizures were more common (72.8%) than focal (27.2%). Etiologies included CNS infections (24.7%), metabolic (18.5%), stroke/structural (16.0%), and idiopathic (40.8%). Acid–base analysis showed acidemia (pH 7.31 ± 0.08), raised PaCO₂ (46.2 ± 8.5 mmHg), low HCO₃⁻ (20.8 ± 3.2 mEq/L), base excess -3.5 ± 2.1 mEq/L, and elevated lactate (3.2 ± 1.1 mmol/L). Electrolyte disturbances included hyponatremia (132.6 ± 5.8 mEq/L), hypokalemia (3.4 ± 0.6 mEq/L), hypocalcemia (7.9 ± 0.7 mg/dL), and low magnesium (1.6 ± 0.3 mg/dL). Haemodynamics showed tachycardia (112 ± 18 bpm) with near-normal BP and MAP, and reduced oxygen saturation ($93 \pm 4\%$). Longer seizure duration correlated with worsening acidosis, hyponatremia, hypokalemia, and higher lactate.

Conclusion: Status epilepticus is frequently complicated by metabolic derangements and haemodynamic instability. Prompt identification and correction of acid–base imbalance and electrolyte abnormalities, along with haemodynamic stabilization, are essential for improving outcomes in these patients.

Keywords: Status epilepticus, acid–base balance, electrolyte disturbance, haemodynamic instability, arterial blood gas.

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Introduction

Status epilepticus (SE) is a life-threatening neurological emergency characterized by prolonged or recurrent seizures without full recovery of consciousness between episodes. It poses a significant risk of neuronal injury, systemic complications, and mortality if not promptly managed [1,2]. SE can arise from a variety of etiologies, including cerebrovascular accidents,

central nervous system infections, metabolic disturbances, and idiopathic causes, with its incidence varying across age groups and populations [3,4]. The pathophysiology of SE is complex and involves sustained neuronal hyperexcitability, excessive glutamate-mediated excitotoxicity, and impaired inhibitory neurotransmission. These neurological processes

often trigger systemic metabolic alterations, including disturbances in acid-base balance and electrolyte homeostasis [5,6]. Common acid-base imbalances observed in SE include metabolic acidosis due to lactic acid accumulation from prolonged muscular activity and respiratory acidosis secondary to hypoventilation. Electrolyte abnormalities, such as hyponatremia, hypokalemia, hypocalcemia, and hypomagnesemia, are also frequently encountered and can both precipitate and perpetuate seizure activity [7,8].

In addition to metabolic and electrolyte disturbances, SE often induces haemodynamic instability. The hyperadrenergic state during prolonged seizures can lead to tachycardia, hypertension, and, in severe cases, hypotension and shock. These cardiovascular changes may compromise cerebral perfusion, exacerbating neuronal injury and negatively impacting patient outcomes [9]. Continuous monitoring and correction of haemodynamic parameters, alongside acid-base and electrolyte management, are therefore critical components of SE care.

While standard SE management focuses primarily on seizure termination using antiepileptic medications, addressing underlying systemic derangements is essential for optimizing recovery and reducing complications. Several studies have highlighted the prognostic significance of early correction of acid-base and electrolyte abnormalities, as well as vigilant haemodynamic monitoring, in improving clinical outcomes [10]. Despite this, there is limited comprehensive data on the simultaneous evaluation of acid-base, electrolyte, and haemodynamic status in SE patients in real-world clinical settings. Understanding the interplay between these systemic disturbances and seizure dynamics is crucial for developing integrated management strategies and improving patient prognosis. Given the clinical importance of these parameters, this study aims to systematically assess the acid-base balance, electrolyte profile, and haemodynamic status in patients presenting with status epilepticus. By analyzing these factors and correlating them with clinical outcomes, this research seeks to provide a detailed understanding of systemic derangements in SE and guide targeted interventions to optimize patient management.

Materials and Methods

Study Design: Prospective Observational Study.

Place of study: Burdwan Medical College and Hospital, Bardhaman, West Bengal 713104.

Period of study: One year- from August, 2021 to July, 2022.

Study Population: All patients of SE between 3m -12 years meeting the following inclusion criteria.

Study Variables

- Age
- Gender
- Seizure Type
- Etiology
- ABG Parameter
- Electrolyte

Sample Size: 81 patients presenting with status epilepticus.

Inclusion Criteria

- Patients of all age groups presenting with status epilepticus.
- Patients or guardians who give informed consent.
- Both new-onset and known seizure disorder cases.

Exclusion Criteria

- Patients with chronic renal, hepatic, or metabolic disorders.
- Patients on medications affecting electrolyte or acid-base balance.
- Patients with traumatic brain injury or recent major surgery.
- Pregnant patients.
- Patients refusing consent.

Statistical Analysis: The collected data were entered into Microsoft Excel and analyzed using SPSS version. Continuous variables such as age, acid-base parameters, electrolyte levels, and haemodynamic measurements were expressed as mean \pm standard deviation (SD), while categorical variables like gender, seizure type, and etiology were presented as frequencies and percentages. Comparisons between groups were performed using the Student's t-test or Mann-

Whitney U test for continuous variables, depending on the distribution, and the Chi-square test or Fisher's exact test for categorical variables. Correlation analyses were conducted using Pearson or Spearman correlation coefficients to evaluate the relationships between acid-base disturbances, electrolyte imbalances, haemodynamic parameters, and clinical outcomes. A p-value of less than 0.05 was considered statistically significant.

Result

Table 1: Demographic and Clinical Characteristics of Patients (n = 81)

		Value (n / %) or Mean ± SD	p-value
Age	Age (years)	32.4 ± 15.7	–
Gender	Male	48 (59.3%)	0.12
	Female	33 (40.7%)	
Seizure Type	Generalized convulsive	59 (72.8%)	0.03
	Focal convulsive	22 (27.2%)	
Etiology	CNS infection	20 (24.7%)	0.05
	Metabolic disturbance	15 (18.5%)	
	Stroke / Structural lesion	13 (16.0%)	
	Idiopathic / Unknown	33 (40.8%)	

Table 2: Arterial Blood Gas (ABG) Parameters

ABG Parameter	Mean ± SD	Normal Range	p-value (vs normal)
pH	7.31 ± 0.08	7.35–7.45	0.02
PaCO ₂ (mmHg)	46.2 ± 8.5	35–45	0.04
HCO ₃ ⁻ (mEq/L)	20.8 ± 3.2	22–26	0.01
Base Excess (mEq/L)	-3.5 ± 2.1	-2 to +2	0.03
Lactate (mmol/L)	3.2 ± 1.1	0.5–2.0	<0.001

Table 3: Serum Electrolytes

Electrolyte	Mean ± SD	Normal Range	p-value (vs normal)
Sodium (Na ⁺ , mEq/L)	132.6 ± 5.8	135–145	0.001
Potassium (K ⁺ , mEq/L)	3.4 ± 0.6	3.5–5.0	0.04
Calcium (Ca ²⁺ , mg/dL)	7.9 ± 0.7	8.5–10.5	0.002
Magnesium (Mg ²⁺ , mg/dL)	1.6 ± 0.3	1.7–2.4	0.03
Chloride (Cl ⁻ , mEq/L)	97 ± 6.5	98–107	0.08

Table 4: Haemodynamic Parameters on Admission

Parameter	Mean ± SD	Normal Range	p-value
Heart Rate (beats/min)	112 ± 18	60–100	0.01
Systolic BP (mmHg)	118 ± 16	90–120	0.08
Diastolic BP (mmHg)	74 ± 12	60–80	0.06
Mean Arterial Pressure (MAP, mmHg)	89 ± 14	70–105	0.07
Oxygen Saturation (%)	93 ± 4	95–100	0.02

Table 5: Correlation of Acid-Base and Electrolyte Disturbances with Duration of SE

Parameter	r (Correlation with seizure duration)	p-value
pH	-0.42	0.001
HCO ₃ ⁻	-0.38	0.003
Sodium	-0.36	0.004
Potassium	-0.31	0.01
Lactate	0.45	<0.001

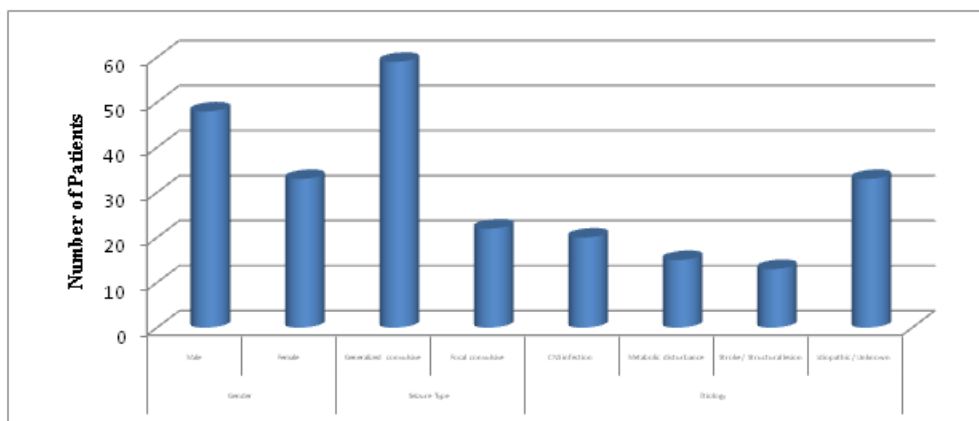


Figure 1: Distribution of Patients with Status Epilepticus by Gender, Seizure Type, and Etiology (n = 81)

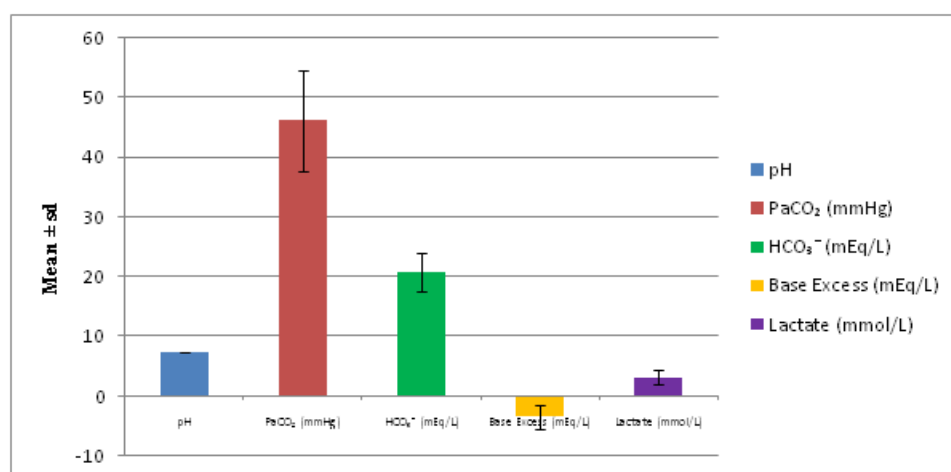


Figure 2: Arterial Blood Gas (ABG) Parameters

In this study of 81 patients with status epilepticus, the mean age was 32.4 ± 15.7 years. There were 48 males (59.3%) and 33 females (40.7%), with no statistically significant gender difference ($p = 0.12$). Generalized convulsive seizures were observed in 59 patients (72.8%) and focal convulsive seizures in 22 patients (27.2%), showing a significant predominance of generalized seizures ($p = 0.03$). Regarding etiology, 20 patients (24.7%) had CNS infections, 15 patients (18.5%) had metabolic disturbances, 13 patients (16.0%) had stroke or structural lesions, and 33 patients (40.8%) had idiopathic or unknown causes ($p = 0.05$).

The mean arterial blood gas values in patients with status epilepticus revealed significant acid-base disturbances. The mean pH was 7.31 ± 0.08 , indicating mild acidemia compared to the normal range of 7.35–7.45 ($p = 0.02$). The mean PaCO₂ was 46.2 ± 8.5 mmHg, slightly above the normal range of 35–45 mmHg ($p = 0.04$), suggesting a component of respiratory acidosis. The mean serum bicarbonate (HCO₃⁻) level was 20.8 ± 3.2 mEq/L, lower than the normal range of 22–26 mEq/L ($p = 0.01$), and the mean base excess was -3.5 ± 2.1 mEq/L compared to the normal -2 to +2 mEq/L ($p = 0.03$), indicating a concurrent metabolic acidosis. Additionally, lactate levels were markedly elevated with a mean of 3.2 ± 1.1 mmol/L versus the normal 0.5–2.0 mmol/L ($p < 0.001$), reflecting increased anaerobic metabolism during prolonged seizures.

Analysis of serum electrolytes in the 81 patients with status epilepticus revealed significant abnormalities. The mean sodium level was 132.6 ± 5.8 mEq/L, below the normal range of 135–145 mEq/L ($p = 0.001$), indicating hyponatremia. Mean potassium was 3.4 ± 0.6 mEq/L, slightly below the normal 3.5–5.0 mEq/L ($p = 0.04$), reflecting mild hypokalemia. Calcium levels were also reduced, with a mean of 7.9 ± 0.7 mg/dL compared to the normal 8.5–10.5 mg/dL ($p = 0.002$). Magnesium was marginally low at 1.6 ± 0.3 mg/dL versus 1.7–

2.4 mg/dL ($p = 0.03$). Chloride levels were slightly decreased with a mean of 97 ± 6.5 mEq/L compared to the normal range of 98–107 mEq/L, though this difference was not statistically significant ($p = 0.08$). The haemodynamic assessment of patients with status epilepticus demonstrated notable deviations from normal values. The mean heart rate was 112 ± 18 beats per minute, significantly elevated compared to the normal range of 60–100 bpm ($p = 0.01$), reflecting a hyperadrenergic state. Mean systolic blood pressure was 118 ± 16 mmHg (normal 90–120 mmHg; $p = 0.08$) and diastolic blood pressure was 74 ± 12 mmHg (normal 60–80 mmHg; $p = 0.06$), showing no statistically significant differences. Mean arterial pressure (MAP) was 89 ± 14 mmHg (normal 70–105 mmHg; $p = 0.07$), also within acceptable limits. Oxygen saturation was slightly reduced with a mean of $93 \pm 4\%$ compared to the normal 95–100% ($p = 0.02$). Correlation analysis revealed significant associations between systemic disturbances and the duration of status epilepticus. Arterial pH showed a moderate negative correlation with seizure duration ($r = -0.42$, $p = 0.001$), indicating that longer seizures were associated with greater acidemia. Similarly, serum bicarbonate (HCO₃⁻) levels correlated negatively ($r = -0.38$, $p = 0.003$), as did sodium ($r = -0.36$, $p = 0.004$) and potassium ($r = -0.31$, $p = 0.01$), suggesting that prolonged seizures were linked to worsening metabolic and electrolyte disturbances. In contrast, lactate levels demonstrated a strong positive correlation with seizure duration ($r = 0.45$, $p < 0.001$), reflecting increased anaerobic metabolism during prolonged seizure activity.

Discussion

Status epilepticus (SE) is a neurological emergency with profound systemic implications, and our study of 81 patients highlights the significant acid-base, electrolyte, and haemodynamic disturbances associated with prolonged seizure activity. The mean age of 32.4 ± 15.7 years and slight male

predominance in our cohort are comparable to previous epidemiological studies reporting SE predominantly in young and middle-aged adults, with a male-to-female ratio ranging from 1.2:1 to 1.5:1 [11,12]. Generalized convulsive seizures were the most frequent type in our study (72.8%), consistent with reports by DeLorenzo et al. and Chin et al., who also noted a higher prevalence of generalized tonic-clonic SE compared to focal seizures [13,14].

Etiologically, CNS infections and idiopathic causes were the most common in our cohort, accounting for 24.7% and 40.8% of cases respectively. This aligns with findings by Hocker et al., who observed that infections and metabolic disturbances contribute significantly to SE in both adult and pediatric populations [15]. Metabolic derangements and structural lesions, though less frequent in our cohort, are also recognized contributors in multiple studies, emphasizing the heterogeneous etiology of SE [16]. Our analysis of arterial blood gases revealed mild acidemia (mean pH 7.31 ± 0.08) with concurrent metabolic acidosis (mean base excess -3.5 ± 2.1 mEq/L) and mild respiratory involvement (PaCO₂ 46.2 ± 8.5 mmHg). These findings corroborate earlier observations by Wijdicks et al. and Nardone et al., who reported that lactic acidosis secondary to sustained muscle activity and hypoventilation contributes to mixed acid-base disturbances in SE patients [17]. The elevated lactate levels in our study (3.2 ± 1.1 mmol/L) are also in agreement with Beran (2008), indicating increased anaerobic metabolism during prolonged seizures [18]. Electrolyte analysis revealed significant hyponatremia, hypokalemia, hypocalcemia, and mild hypomagnesemia in our cohort. These abnormalities are well-documented in SE, as electrolyte imbalances can both precipitate and exacerbate seizures. Swarnalingam et al. highlighted that hyponatremia is a particularly common finding in SE and may influence seizure threshold, a finding reflected in our cohort where 132.6 ± 5.8 mEq/L sodium was observed. Hypokalemia and hypocalcemia in our patients also parallel findings by Khoueiry et al. [19], reinforcing the need for prompt correction of electrolytes to reduce seizure duration and prevent complications. Haemodynamically, our patients exhibited significant tachycardia (112 ± 18 bpm) and mild hypoxemia ($93 \pm 4\%$ SpO₂), whereas blood pressure parameters remained largely within normal limits. This is consistent with the hyperadrenergic response described by Farrokh et al., who reported that sympathetic overactivity during SE often manifests as tachycardia and transient hypertension, whereas hypotension is generally observed only in prolonged or refractory SE [20]. These findings highlight the importance of continuous cardiovascular monitoring during SE management. Importantly, correlation analysis in

our study demonstrated that longer seizure duration was associated with worsening acidemia ($r = -0.42$, $p = 0.001$), decreased bicarbonate ($r = -0.38$, $p = 0.003$), and electrolyte depletion (sodium $r = -0.36$, potassium $r = -0.31$), while lactate levels increased with seizure duration ($r = 0.45$, $p < 0.001$). These correlations underscore the importance of early intervention to terminate seizures and correct systemic abnormalities to mitigate neuronal injury and multi-organ complications. Monitoring and management of acid-base status, electrolytes, and haemodynamics are crucial components of comprehensive SE care. Early recognition and correction of these systemic disturbances, alongside standard anticonvulsant therapy, may improve clinical outcomes and reduce morbidity and mortality in SE patients.

Conclusion

In this study of 81 patients with status epilepticus, significant acid-base disturbances, electrolyte imbalances, and haemodynamic alterations were observed, with prolonged seizure duration being associated with worsening metabolic derangements. Hyponatremia, hypokalemia, hypocalcemia, and mild hypomagnesemia were common, while arterial blood gas analysis revealed mixed metabolic and respiratory acidosis with elevated lactate levels.

Haemodynamically, patients exhibited tachycardia and mild hypoxemia, although blood pressure remained largely within normal limits. These findings underscore the importance of early recognition and correction of systemic abnormalities alongside prompt seizure control to reduce morbidity and improve outcomes. Continuous monitoring of acid-base status, electrolytes, and cardiovascular parameters is essential for optimal management of patients with status epilepticus.

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