

## ELECTROLYTE IMBALANCES IN SEIZURE PATIENTS: PREVALENCE, PATTERNS, AND CLINICAL IMPLICATIONS

### Neurology

**Dr. Prince Kumar Singh**

Assistant Professor Department of Medicine, GMC, Banda

**Dr. Rajat Pandey**

Senior Resident Department of Medicine, MVASMC, Basti

**Dr. Praveen Prasad**

Senior Resident Department of Medicine, MRAMC, Ambedkar Nagar

### ABSTRACT

**Introduction:** Seizures, characterized by abnormal synchronous neuronal discharge in the brain, result in sudden changes in motor, behavior, and sensory perception. Recognizing the type of seizure is crucial for treatment and prognosis. Electrolyte imbalances are considered a potential cause of seizures, impacting clinical outcomes. This study aimed to determine the prevalence of electrolyte imbalances in seizure patients and their relationship with clinical outcomes. **Materials and Methods:** This cross-sectional study was conducted at the Department of Medicine, MLN Medical College, Prayagraj, to investigate the relationship between seizures and electrolyte imbalances in adult patients. The study included 100 participants, with data collected between May 2020 and October 2021. Demographics, seizure diagnoses, hematological and biochemical parameters, as well as serum electrolyte levels, were assessed. The study compared these levels to defined normal ranges, and outcome data, including hospital stay duration, were recorded. **Results:** The study encompassed 100 patients, primarily within the age group of 41 to 70 years (54%) with a mean age of  $54.22 \pm 19.60$  years. Males constituted the majority (72%), resulting in a male-to-female ratio of 2.57. Generalized tonic-clonic seizures (64%) were the most common, followed by focal seizures (27%) and status epilepticus (9%). Various hematological and biochemical parameters exhibited a wide range of values. Among the electrolytes, calcium exhibited the highest degree of derangement (99%), followed by sodium (92%), potassium (63%), and magnesium (33%). **Conclusion:** This study disclosed a high prevalence of electrolyte imbalances in seizure patients, affecting sodium, potassium, magnesium, and calcium levels. However, it did not establish significant associations between these imbalances and clinicodemographic factors or clinical outcomes. It's important to note that the study focused on seizure patients with relatively stable profiles, which may have limited the ability to identify strong correlations between electrolyte imbalances and these factors. To gain deeper insights into these associations, future studies are recommended.

### KEYWORDS

Seizures, Electrolyte Imbalance, Clinical Outcomes, Seizure Types, Neurological Dysfunction, Electrolyte Levels

### INTRODUCTION

Seizure (from Latin word *sacire*, "to take possession of") is a paroxysmal event due to abnormal excessive synchronous neuronal discharge activity in the brain. Although the underlying mechanisms are not fully understood and there are a number of proposed pathophysiological mechanisms yet it is believed that a seizure takes place when there is distortion of the normal balance between excitation (E) and inhibition (I) in the brain. [1] These excitation and inhibition activity in the brain is governed with the help of various neurotransmitters. Glutamate, aspartate, norepinephrine, and acetylcholine are the neurotransmitters associated with excitation whereas gamma-aminobutyric acid (GABA) and glycine play an inhibitory role. Both excitatory as well as inhibitory neurotransmitters perform their action through the release of electric charge resulting into an impulse transmission. In rest state, the neuron membrane has some electric potential and is slightly polarized. A change in the polarity of neuron membrane triggered by an action potential activates all the cells to release an electric charge. [2]

The change in status of neuron membrane could be brought about by a number of factors. Neuron membrane is permeable to electrolyte ions like calcium and sodium and permits them to pass from extracellular to intracellular region. Entry of these ions into intracellular environment affects the polarization status of neuron membrane resulting into release of excessive, irregular, and controlled electric charge. When this activity takes place at a large level in multiple neurons then it may result in seizures. [3] Thus, electrolytes seem to have a dominant role in the causation of seizures.

Electrolytes play an important role in maintenance of human health and wellbeing including functioning of brain. In this respect, calcium is an important electrolyte that plays an important role in regulation of cell events, such as signaling hormone secretion, glycogen metabolism, and cell mitosis. Extracellular calcium is not only a source for intracellular calcium but also has an important role in the maintenance and stability of cell wall and clot formation. [3] Calcium is also a universal messenger for extracellular signals in a great variety of cells; it regulates several neuronal functions, such as neurotransmitter synthesis and release, neuronal excitability, phosphorylation and so on. [4] Calcium along with phosphorous and magnesium is also responsible for bone mineralization and formation of skeletal system. Magnesium and phosphorous [5,6] are some of the

other electrolytes that play an active role in intracellular and extracellular mechanisms in various metabolic activities.

Electrolyte homeostasis in the central nervous system (CNS) is a characteristic essential feature behind brain functioning. "Regulation of ionic balance is a critical process involving a complex array of molecules for moving ions into and out of the brain and involving blood-brain barrier function as well as mechanism in the membranes of both neurons and glia. Alterations in ion gradients across cellular membranes can have direct and indirect effects on neuronal discharge and may facilitate epileptiform activities". [7]

A number of clinical studies have also shown a relationship between electrolyte imbalance and seizures. In clinical practice, hyponatremia and hypokalemia have frequently been seen in seizure patients while hypernatremia, hypercalcemia, hypermagnesemia and hyperkalemia have occasionally been seen in the seizures. [8] Hypocalcemia is relatively less encountered in seizures patients, however, patients having severe and acute hypocalcemia are at an increased risk of seizures. [8]

Given the understanding that electrolyte imbalance could be an important reason for seizure. It is important to recognize it and make appropriate interventions. Most of the electrolytes have multiple functions apart from affecting the CNS homeostasis and hence they could have a detrimental effect on the clinical outcomes too. Hence the present study was planned to determine the prevalence of electrolyte imbalance in patients of seizure and to study its relationship with clinical outcome.

### MATERIAL AND METHODS

This cross-sectional study, conducted at the Department of Medicine, MLN Medical College, Prayagraj, explored the relationship between seizures and electrolyte imbalances in adult patients. A sample of 100 participants was included based on a calculated flip-coin probability, with data collected between May 2020 and October 2021. Demographics, seizure diagnoses, and hematological/biochemical parameters were assessed, with serum electrolyte levels compared to defined normal ranges. Outcome data, including duration of hospital stay, was recorded. Statistical analysis was performed using SPSS, with significance defined as  $p < 0.05$ . The study provides insights into the prevalence and implications of electrolyte imbalances in seizure

patients.

## RESULTS

In this study, a total of 100 seizure patients were enrolled, and their characteristics were analysed. The patient population had an age range of 18 to 92 years, with the majority falling within the 41 to 70 years age group (54%). Notably, two patients were above 90 years of age. The mean age of the patients was  $54.22 \pm 19.60$  years. The study predominantly included male patients (72%), resulting in a male-to-female (M: F) ratio of 2.57. The most common seizure type among the patients was generalized tonic-clonic seizures (64%), followed by focal seizures (27%) and status epilepticus (9%). Hemoglobin levels ranged from 4.10 to 15.20 g/dl, with an average of  $10.76 \pm 1.91$  g/dl. The total leucocyte count exhibited a wide range of values, from 2.2 to 22.2 thousands/mm<sup>3</sup>.

Random blood sugar levels ranged from 85 to 456 mg/dl, with a mean of  $161.58 \pm 53.66$  mg/dl. Serum bilirubin levels ranged from 0.2 to 5.89 mg/dl, with a mean of  $1.15 \pm 0.84$  mg/dl. Serum AST levels ranged from 15 to 626.70 IU/L, with a mean of  $80.94 \pm 90.11$  IU/L. Serum ALP levels had a range of 48.20 to 1162.20 IU/L, with a mean of  $182.68 \pm 149.86$  IU/L.

Serum urea levels ranged from 28.90 to 125.00 mg/dl, with an average of  $61.58 \pm 22.57$  mg/dl, while serum creatinine levels had a range of 0.08 to 12.00 mg/dl, with a mean of  $1.41 \pm 1.15$  mg/dl. The study also assessed various electrolyte levels, with serum sodium ranging from 100.2 to 185.2 mEq/L (mean  $119.97 \pm 19.73$  mEq/L), serum potassium ranging from 1.04 to 5.60 mEq/L (mean  $3.53 \pm 0.78$  mEq/L), serum magnesium ranging from 0.40 to 2.10 mEq/L (mean  $1.69 \pm 0.32$ ), and serum calcium ranging from 0.80 to 5.20 mEq/L (mean  $4.37 \pm 0.80$  mEq/L).

Among the electrolytes, calcium exhibited the highest degree of derangement (99%), followed by sodium (92%), potassium (63%), and magnesium (33%). Hyponatremia, normonatremia, and hypernatremia were observed in 82%, 8%, and 10% of cases, respectively. Hypokalemia, normokalemia, and hyperkalemia were observed in 54%, 37%, and 9% of cases, respectively. Hypomagnesemia, normomagnesemia, and hypermagnesemia were observed in 33%, 67%, and 0% of cases, respectively. Hypocalcemia, normocalcemia, and hypercalcemia were observed in 5%, 1%, and 94% of cases, respectively.

The study also conducted investigations such as CT, MRI, and EEG in a subset of patients. Abnormal findings were observed in 4.1%, 3.3%, and 20% of cases, respectively. The duration of hospital stay ranged from 1 to 10 days, with an average of 4.46 days. Most patients (76) had a hospital stay of less than 5 days, while the remaining 24 had a stay of more than 5 days. A total of 85 patients were discharged after recovery, and 15 were referred to another facility. Importantly, there were no in-hospital mortalities observed.

Regarding serum sodium levels, no significant associations were found with patient age, sex, hemoglobin levels, or various biochemical parameters. However, a significant relationship was identified between serum sodium levels and the type of seizures, with hyponatremia being more common in generalized tonic-clonic seizures and hypernatremia more common in status epilepticus ( $p < 0.001$ ). Serum sodium levels were also significantly associated with total leukocyte count, with higher values seen in both hyponatremia and hypernatremia compared to normonatremia ( $p = 0.011$ ).

Serum potassium levels did not exhibit significant associations with clinicodemographic variables or outcomes, except for serum ALP levels, which were significantly higher in hyperkalemia compared to hypokalemia and normokalemia ( $p = 0.010$ ). Similarly, serum magnesium levels did not show significant associations with clinicodemographic variables, except for random blood sugar levels, which were significantly higher in hypomagnesemia compared to normomagnesemia ( $p = 0.043$ ). Serum calcium status did not exhibit significant associations with any clinicodemographic variables.

## DISCUSSION

The present study was carried out in an adult population (aged >18 years) and had majority of patients in age range 41-70 years (54%). Mean age of patients was 54.22 years. Most of the previous studies assessing electrolyte balance in children below 6 years of age [9,14,17,18,19,21] There are only few studies conducted among

adults. In one such study Halawa et al. [10] reported the age range of patients as 36 to 88 years and mean age of patients as 62 years. In their study they had dominance of women (63%). In a study conducted in Indian population that included patients aged 20 to 60 years, mean age of patients was  $41.6 \pm 10.22$  years and similar to present study majority of cases (74%) were males. In their study by Mohanta et al. [21] found 73% patients in age range 26-45 years thus showing the age profile of patients to be younger than the present study, however, similar to present study they also found a dominance of males (69%).

Generalised tonic-clonic seizures were observed in 64% of the patients in the current study, followed by focal seizures (27%), and status epilepticus (9%). Similar to the findings of the current investigation, Mohanta et al. [21] identified a preponderance of generalised tonic-clonic seizures (73%) among adult studies. The remaining seizure type observed in their research was focal (30%), and no case of status epilepticus was documented. In a separate study involving adults, Dhadke et al. [23] discovered that generalised seizures predominated over focal seizures by 76% to 24%; however, they did not specify the proportion of patients with status epilepticus. Antony and Nathan in a recent study also reported generalized seizures to be dominant type (65.4%) followed by focal seizures (14.0%) and reported 20.6% cases as unclear epileptic spasms. Sambyal and Chandail [24] in their study that included a total of 1680 seizure patients reported 86.2% as GCTCS, 3.9% as partial seizures with secondary generalization, 4.8% complex partial seizures and 3.9% partial seizures evolving to secondarily generalised seizures. In general most of the studies similar to present study have reported a dominance of generalized seizures as the underlying seizure type.

The hematological profile of patients showed mean hemoglobin and total leucocyte count in normal range. Although iron deficiency anemia is associated with an increased febrile seizure risk among children yet tenability of this relationship in adults is not established. Moreover, the present study ruled out inclusion of febrile seizure patients. As far as association of anemia with nonfebrile seizures is concerned, there is no established evidence to support a relationship. Although a study reports higher leukocyte count in patients with generalized seizures as compared to those having focal seizures 85, however, there is no convincing evidence to establish the role of TLC in non-febrile seizures. A normal hematological profile in terms of hemoglobin levels and total leukocyte count in the present study also ruled out any particular role of these parameters in non-febrile seizure cases.

In this study, although mean random blood sugar levels were within the normal range, there were higher levels of liver enzymes (AST/ALT) and serum urea. However, it's noteworthy that liver enzymes and renal functions were less affected in non-febrile seizures compared to febrile seizures. While an association between liver dysfunction and the use of antiepileptic drugs has been reported in the literature, the broad spectrum of exclusions in this study, such as cerebrovascular accidents, subarachnoid hemorrhage, and other conditions, makes it challenging to definitively attribute seizures to hepatic or renal causes. The study focused on four key electrolytes – sodium, potassium, calcium, and magnesium. Notably, the mean serum concentrations for these electrolytes deviated from the normal ranges, with sodium levels falling below, potassium levels nearing the lower limit, magnesium remaining within, and calcium levels exceeding the normal range. Hyponatremia and hypokalemia were prevalent, reflecting significant electrolyte imbalances in this cohort. While serum calcium and sodium imbalances were the most frequent, magnesium showed the least pronounced imbalance, with the majority of cases falling within the normal range. This emphasizes the need for further comprehensive investigations into electrolyte imbalances in seizures.

In relation to sodium and calcium concentrations, it has been documented that both of these elements play a role in the initiation of seizure activity (34, 46, 47). In contrast to the findings of the current investigation, Namakian et al. [15], who examined the four electrolytes (serum sodium, potassium, magnesium, and calcium), documented the following mean values for children experiencing febrile seizures:  $136.2 \pm 3.3$ ,  $4.38 \pm 0.41$ ,  $8.79 \pm 0.47$ , and  $1.9 \pm 0.32$ , respectively. Patients with epilepsy in that study had significantly elevated levels of serum sodium, potassium, and magnesium in comparison to the present investigation, whereas serum calcium levels were considerably decreased. Despite this, it is important to note that their research was conducted on minors and included patients with febrile seizures. Febrile illness has its own impact on the electrolyte

levels and in non-febrile cases such as ours need not necessarily replicate those findings. In another study that included children with febrile seizure Usha Kiran and Suresh [11] reported the mean serum sodium, magnesium and calcium levels as  $138.18 \pm 5.23$ ,  $1.85 \pm 0.26$  and  $8.88 \pm 0.56$  mEq/L respectively thus showing trends for serum sodium levels to be quite different from the present study but that of serum magnesium and calcium in agreement with the present study. Most of the other studies, primarily conducted in children with febrile seizures do not show trends similar to the present study, especially for serum sodium levels which were much lower in the present study as compared to these studies. [12,18] Among children, only one study highlights serum sodium levels  $<130$  mmol/l in 31% children. In the present study, we found these levels to be  $<135$  mmol/l in as many as 82% of cases thus reflecting sodium deficiency as the dominant underlying electrolyte imbalance condition.

Among adults too, Mohanta et al. [75] in their study found hypocalcemia and hypomagnesemia to be highly prevalent in seizure patients. In the present study, though we found prevalence of hypomagnesemia to be 37%, however, only 5 patients with hypocalcemia and hypercalcemia was the dominant issue (94%). Association of low magnesium levels with seizure activity in adults was also established by Abdulallahi et al. [17] in their study. In another study among adults, Sharma et al. [29] indicated the possibility that calcium deficiency could be an electrolyte imbalance leading to seizures, however, in the present study there is high prevalence of hypercalcemia, thus ruling out such association. Normal calcium levels or a low prevalence of hypocalcemia in seizure patients as observed in the present study was also witnessed by Rama Krishna et al. [14] who found calcium levels within normal range in 82% adult seizure patients. However, in their study, prevalence of potassium deficiency was also much lower (10%) which is in contrast with the findings of the present study where majority (54%) of seizure patients had potassium deficiency.

In the present study, there was a high prevalence of hyponatremia (82%). Hyponatremia could be one of the most common factors leading to seizure activity as also shown by Halawa et al. [10] who found that with decreasing serum sodium levels the odds of seizure increase substantially. The high prevalence of hyponatremia in the present study also endorsed this relationship. In a recent study by Sambyal et al. [23], hyponatremia, hypocalcemia and hypomagnesemia were reported in 46%, 28.6% and 25.4% of adult seizure patients respectively. In effect, most of the studies whether in children as well as in adults have shown low sodium levels to be associated with seizure activity which is also established in the present study with as high as 82% of the patients having low sodium levels.

This study, in comparing findings with prior research in adults and children, underscores the common occurrence of electrolyte imbalances in seizure cases, particularly a deficiency in cations. However, pinpointing a single responsible electrolyte for seizure activity remains challenging, as the nature and direction of relationships between different cations can vary due to distinct pathophysiological pathways. Seizure activity involves the release of electrical charges, but the specific electrolyte responsible is difficult to identify and may vary with different underlying conditions. Notably, this study differs from previous ones by focusing on adult non-febrile seizure cases and excluding various chronic and acute conditions that typically lead to electrolyte imbalances. Within its defined scope, the study highlights a high prevalence of hyponatremia and hypercalcemia as prominent electrolyte imbalances in non-febrile adult seizure patients, free from underlying chronic or acute confounding conditions. One of the limitations of most of the previous studies, as well as the present study, was the ignorance of anion imbalance. Interestingly, some workers have focused on this direction too and

have found a possible role of anions imbalance in causation and differentiation of seizure and its types. It must be noted that a cation can bind with different anions and vice versa, and thus a disturbance in equilibrium of this anion-cation relationship could be responsible for release of electrical charge responsible for seizure activity and thus study of only a few electrolytes could give indication of only a possible but not established relationship between electrolyte imbalance and seizure activity. Having deduced this, the practical implications and feasibility of comprehensive electrolyte evaluation in context with seizure activity remains to be examined.

In the present study, the outcomes were generally favourable. There was no mortality and duration of hospital stay was  $<5$  days in 76% cases. None of the patients stayed in hospital beyond 10 days. The favorable outcomes in the present study could be primarily attributable to the eligibility criteria of the study that ruled out a number of serious conditions and restrained the study population having a relatively stable condition. Non-febrile seizures without a serious underlying conditions generally have a favourable outcome. In different studies that included an assessment of electrolyte imbalance in adult seizure patients too no mortality has been reported. [15,23] In the study by Antony and Nathan [18] recovery rate as high as 98% was reported. The present study also showed a high recovery rate (85%) and referral in 15% patients only.

In the present study, we did not find a significant association of electrolyte imbalance of any type with final outcome and duration of hospital stay. This is despite a high prevalence of hyponatremia and hypokalemia which are often associated with critical illness. [31,32,33] One of the reasons for this could be the fact that hyponatremia and hypokalemia were generally of transient nature and were effectively managed by appropriate measure in-hospital interventions.

In the present study, we found an association of serum sodium levels with type of seizures with hyponatremia being more common in GTCS and hypernatremia being more common in status epilepticus. In their study, Mohanta et al. [21] found higher serum magnesium and calcium levels in GTCS as compared to focal seizure cases. Most of the other studies did not report any such association. The findings thus suggest that it is one of the less explored relationships that warrants further evaluation.

In the present study, we also found some physiological and metabolic relationships of electrolyte imbalances, viz. a significant relationship between serum sodium status and total leukocyte count, serum ALP levels with serum potassium status and random blood sugar levels with serum magnesium status. These significant electrolyte relationships tend to elaborate the role of these electrolytes in different metabolic and physiological activities which eventually could be related with pathogenesis of seizure activity.

One of the limitations of the study was absence of a control group. A number of previous studies evaluating the relationship of electrolyte imbalance with seizure have been done using a case-control model. [9,29] Inclusion of a control group could have helped to specify a possible cause-effect relationship in a better manner.

With its limitations, the present study found a high prevalence of electrolyte imbalances in seizure patients. The study highlighted that electrolyte imbalance needs a more elaborative investigation focusing on the imbalance of an electrolyte equilibrium instead of focusing only on some selected electrolytes. An imbalance of both cations and anions is essential to be studied. Further studies in this direction are needed with focus on long-term outcomes and recurrence too.

**Table-1 Association Of Serum Sodium Status With Different Clinicodemographic Variables And Outcome.**

| SN | Variable                  | Hyponatremia (n=82) |    | Normal (n=8)      |    | Hypernatremia (n=10) |    | Statistical significance |   |
|----|---------------------------|---------------------|----|-------------------|----|----------------------|----|--------------------------|---|
| 1. | Mean age $\pm$ SD (years) | 53.65 $\pm$ 19.79   |    | 61.88 $\pm$ 20.75 |    | 52.80 $\pm$ 17.52    |    | F=0.667; p=0.516         |   |
| 2. | Sex                       |                     |    |                   |    |                      |    |                          |   |
|    | Male                      | 59 (72.0%)          |    | 6 (75.0%)         |    | 7 (70.0%)            |    | $\chi^2=0.056$ ;         |   |
|    | Female                    | 23 (28.0%)          |    | 2 (25.0%)         |    | 3 (30.0%)            |    | p=0.973                  |   |
| 3. | Type seizures of          |                     |    |                   |    |                      |    |                          |   |
|    | Focal                     | 21 (25.6%)          |    | 4 (50.0%)         |    | 2 (20.0%)            |    | $\chi^2=37.878$ ;        |   |
|    | GTCS                      | 58 (70.7%)          |    | 4 (50.0%)         |    | 2 (20.0%)            |    | p<0.001                  |   |
|    | Status epilepticus        | 3 (3.7%)            |    | 0                 |    | 6 (60.0%)            |    |                          |   |
|    |                           | Mean                | SD | Mean              | SD | Mean                 | SD | F                        | p |

|     |                            |              |        |             |       |             |        |                   |       |
|-----|----------------------------|--------------|--------|-------------|-------|-------------|--------|-------------------|-------|
| 4.  | Hb (g/dl)                  | 12.60        | 16.03  | 9.28        | 2.85  | 21.21       | 33.11  | 1.253             | 0.290 |
| 5.  | TLC(thou-sands/ cumm)      | 9.65         | 4.04   | 5.36        | 2.38  | 8.82        | 1.89   | 4.743             | 0.011 |
| 6.  | RBS (mg/dl)                | 162.80       | 55.80  | 157.00      | 27.02 | 155.20      | 54.82  | 0.119             | 0.888 |
| 7.  | S. Bil. (mg/dl)            | 1.11         | 0.71   | 0.88        | 0.24  | 1.72        | 1.65   | 2.919             | 0.059 |
| 8.  | S. AST (IU/L)              | 84.24        | 96.36  | 55.71       | 24.96 | 74.04       | 67.44  | 0.393             | 0.676 |
| 9.  | S. ALP (IU/L)              | 180.58       | 119.67 | 140.41      | 71.82 | 232.71      | 331.23 | 0.881             | 0.417 |
| 10. | S. Urea (mg/dl)            | 62.85        | 23.31  | 61.95       | 21.57 | 50.84       | 14.45  | 1.270             | 0.285 |
| 11. | S. creatinine (mg/dl)      | 1.42         | 1.26   | 1.40        | 0.37  | 1.28        | 0.44   | 0.068             | 0.935 |
| 12. | Abnormal CT                | 3/79 (3.8%)  |        | 0/8 (0%)    |       | 1/10 (10%)  |        | □2=1.239; p=0.538 |       |
| 13. | Abnormal MRI               | 1/50 (2%)    |        | 0/5 (0%)    |       | 1/6 (16.7%) |        | □2=3.819; p=0.148 |       |
| 14. | Abnormal EEG               | 6/35 (17.1%) |        | 1/5 (20%)   |       | 2/4 (50%)   |        | □2=2.383; p=0.304 |       |
| 15. | Hospital stay >5 days      | 18 (22%)     |        | 3/8 (37.5%) |       | 3/10 (30%)  |        | □2=1.185; p=0.553 |       |
| 16. | Referred to other facility | 13 (15.9%)   |        | 1 (12.5%)   |       | 1 (10.0%)   |        | □2=0.282; p=0.868 |       |

**Table: - 2 Association Of Serum Potassium Status With Different Clinicodemographic Variables And Outcome**

| SN  | Variable                   | Hypokalemia (n=54) |        | Normal (n=37) |       | Hyperkalemia (n=9) |        | Statistical signi-ficance |       |
|-----|----------------------------|--------------------|--------|---------------|-------|--------------------|--------|---------------------------|-------|
| 1.  | Mean age±SD(years)         | 51.43±18.26        |        | 58.54±19.85   |       | 53.22±25.07        |        | F=1.474; p=0.234          |       |
| 2.  | Sex                        |                    |        |               |       |                    |        |                           |       |
|     | Male                       | 42 (77.8%)         |        | 23 (62.2%)    |       | 7 (77.7%)          |        | □2=2.819;<br>p=0.244      |       |
|     | Female                     | 12 (22.2%)         |        | 14 (37.8%)    |       | 2 (22.2%)          |        |                           |       |
| 3.  | Type of seizures           |                    |        |               |       |                    |        |                           |       |
|     | Focal                      | 14 (25.9%)         |        | 10 (27.0%)    |       | 3 (33.3%)          |        | □2=8.322;<br>p=0.080      |       |
|     | GTCS                       | 37 (68.5%)         |        | 34 (64.9%)    |       | 3 (33.3%)          |        |                           |       |
|     | Status epilepticus         | 3 (5.6%)           |        | 3 (8.1%)      |       | 3 (33.3%)          |        |                           |       |
|     |                            | Mean               | SD     | Mean          | SD    | Mean               | SD     | F                         | p     |
| 4.  | Hb (g/dl)                  | 13.66              | 19.69  | 13.17         | 17.38 | 10.51              | 1.88   | 0.118                     | 0.889 |
| 5.  | TLC(thousands/cumm)        | 8.88               | 3.60   | 9.26          | 4.27  | 11.20              | 4.28   | 1.360                     | 0.262 |
| 6.  | RBS (mg/dl)                | 157.70             | 62.94  | 170.59        | 40.67 | 147.78             | 35.39  | 0.960                     | 0.387 |
| 7.  | S. Bil. (mg/dl)            | 1.16               | 0.67   | 1.02          | 0.59  | 1.61               | 1.98   | 1.864                     | 0.161 |
| 8.  | S. AST (IU/L)              | 86.50              | 111.40 | 67.18         | 39.77 | 104.08             | 97.21  | 0.828                     | 0.440 |
| 9.  | S. ALP (IU/L)              | 187.82             | 126.34 | 143.91        | 87.53 | 310.07             | 335.10 | 4.876                     | 0.010 |
| 10. | S. Urea (mg/dl)            | 60.58              | 23.34  | 63.94         | 23.15 | 57.80              | 15.36  | 0.377                     | 0.687 |
| 11. | S. creatinine (mg/dl)      | 1.55               | 1.51   | 1.23          | 0.35  | 1.29               | 0.69   | 0.868                     | 0.423 |
| 12. | Abnormal CT                | 4/54 (7.4%)        |        | 0/34 (0%)     |       | 0/9(0%)            |        | □2=3.322; p=0.190         |       |
| 13. | Abnormal MRI               | 1/33 (3.0%)        |        | 0/22 (0%)     |       | 1/6 (16.7%)        |        | □2=4.143; p=0.126         |       |
| 14. | Abnormal EEG               | 5/28 (17.9%)       |        | 3/12 (25%)    |       | 1/4 (25%)          |        | □2=0.319; p=0.852         |       |
| 15. | Hospital stay >5 days      | 9 (16.7%)          |        | 11 (29.7%)    |       | 4 (44.4%)          |        | □2=4.320; p=0.115         |       |
| 16. | Referred to other facility | 7 (13%)            |        | 6 (16.2%)     |       | 2 (22.2%)          |        | □2=0.587; p=0.746         |       |

**Table-3: Association Of Serum Magnesium Status With Different Clinicodemographic Variables And Outcome**

| SN  | Variable                   | Hypomagnesemia (n=33) |       | Normal (n=67) |        | Statistical significance |       |
|-----|----------------------------|-----------------------|-------|---------------|--------|--------------------------|-------|
| 1.  | Mean age±SD (years)        | 54.85±20.95           |       | 53.91±19.05   |        | t=0.224; p=0.823         |       |
| 2.  | Sex                        |                       |       |               |        |                          |       |
|     | Male                       | 20 (60.6%)            |       | 52 (77.6%)    |        | □2=3.172; p=0.075        |       |
|     | Female                     | 13 (39.4%)            |       | 15 (22.4%)    |        |                          |       |
| 3.  | Type of seizures           |                       |       |               |        |                          |       |
|     | Focal                      | 7 (21.2%)             |       | 20 (29.9%)    |        | □2=3.625; p=0.163        |       |
|     | GTCS                       | 25 (75.8%)            |       | 39 (58.2%)    |        |                          |       |
|     | Status epilepticus         | 1 (3.0%)              |       | 8 (11.9%)     |        |                          |       |
|     |                            | Mean                  | SD    | Mean          | SD     | t                        | p     |
| 4.  | Hb (g/dl)                  | 10.38                 | 1.61  | 14.58         | 21.70  | -1.109                   | 0.270 |
| 5.  | TLC (thousands/ cumm)      | 8.63                  | 3.97  | 9.52          | 3.91   | -1.067                   | 0.289 |
| 6.  | RBS (mg/dl)                | 177.03                | 72.18 | 153.97        | 40.18  | 2.053                    | 0.043 |
| 7.  | S. Bil. (mg/dl)            | 0.99                  | 0.56  | 1.23          | 0.94   | -1.337                   | 0.184 |
| 8.  | S. AST (IU/L)              | 62.05                 | 47.85 | 90.24         | 103.94 | -1.480                   | 0.142 |
| 9.  | S. ALP (IU/L)              | 160.86                | 88.59 | 193.27        | 171.84 | -1.017                   | 0.312 |
| 10. | S. Urea (mg/dl)            | 65.12                 | 25.07 | 59.83         | 21.22  | 1.104                    | 0.272 |
| 11. | S. creatinine (mg/dl)      | 1.36                  | 0.47  | 1.43          | 1.37   | -0.319                   | 0.750 |
| 12. | Abnormal CT                | 1/32 (3.1%)           |       | 3/65 (4.6%)   |        | □2=0.120; p=0.729        |       |
| 13. | Abnormal MRI               | 0/21 (0%)             |       | 2/40 (5%)     |        | □2=1.086; p=0.297        |       |
| 14. | Abnormal EEG               | 2/13 (15.4%)          |       | 7/31 (22.6%)  |        | □2=0.291; p=0.589        |       |
| 15. | Hospital stay >5 days      | 8 (24.2%)             |       | 16 (23.9%)    |        | □2=0.002; p=0.968        |       |
| 16. | Referred to other facility | 4 (12.1%)             |       | 11 (16.4%)    |        | □2=0.320; p=0.572        |       |

**Table-4 Association Of Serum Calcium Status With Different Clinicodemographic Variables And Outcome**

| SN | Variable            | Hypocalcemia (n=5) | Normal (n=1) | Hypercalcemia (n=95) | Statistical significance |
|----|---------------------|--------------------|--------------|----------------------|--------------------------|
| 1. | Mean age±SD (years) | 53.0±14.98         | 60.00        | 54.22±19.97          | F=0.052; p=0.949         |
| 2. | Sex                 |                    |              |                      |                          |

|    |                    |         |          |            |                   |
|----|--------------------|---------|----------|------------|-------------------|
|    | Male               | 4 (80%) | 1 (100%) | 67 (71.3%) | □2=0.572; p=0.751 |
|    | Female             | 1 (20%) | -        | 27 (28.7%) |                   |
| 3. | Type seizures      |         |          |            |                   |
|    | Focal              | 3 (60%) | 0        | 24 (25.5%) | □2=3.600; p=0.463 |
|    | GTCS               | 2 (40%) | 1 (100%) | 61 (64.9%) |                   |
|    | Status epilepticus | 0       | 0        | 9 (9.6%)   |                   |

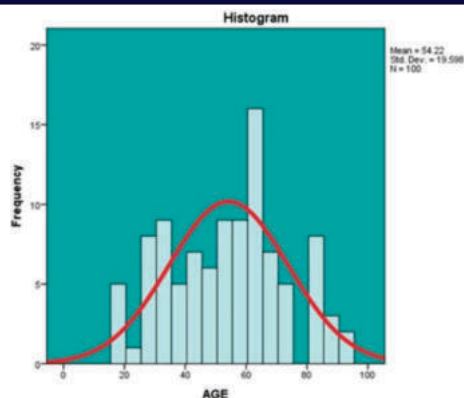


Fig. 1: Histogram Showing Age Dispersion Of Study Population

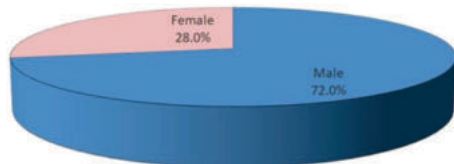


Fig. 2: Pie Diagram Showing Gender Distribution Of Study Population

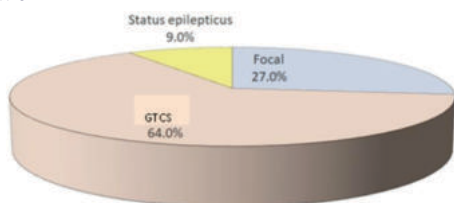


Fig. 3: Distribution Of Cases According To Seizure Type

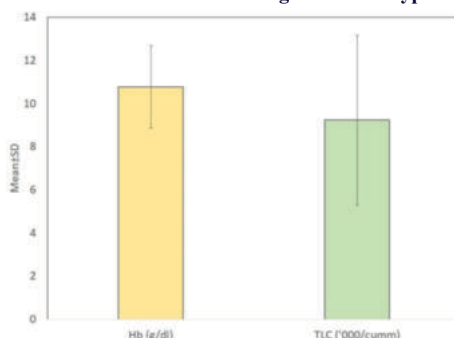


Fig. 4: Hemoglobin And TLC Profile In Study Population (Mean±SD)

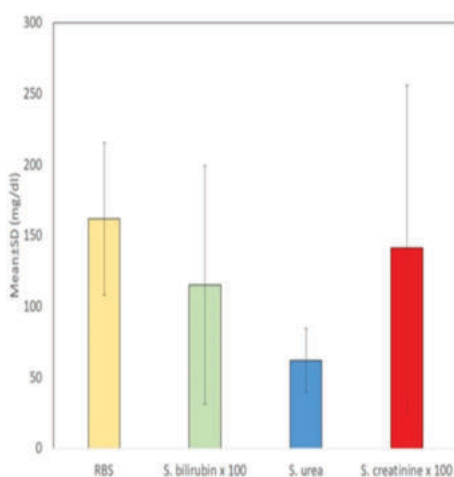


Fig. 5: Random Blood Sugar, Bilirubin, Urea And Creatinine Levels In Study Population

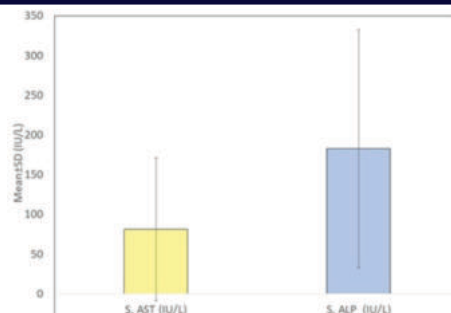


Fig. 6: Serum AST And ALP Levels In Study Population

## CONCLUSION

In conclusion, this study revealed a high prevalence of electrolyte imbalances among seizure patients, with a substantial number exhibiting variations in sodium, potassium, magnesium, and calcium levels. However, the research did not establish significant associations between these electrolyte imbalances and various clinicodemographic factors or clinical outcomes. It's important to note that the study focused on seizure patients with a relatively stable profile, which may have limited the ability to identify strong correlations between electrolyte imbalances and these factors. To gain deeper insights into these associations, future studies with larger and more diverse patient populations encompassing various seizure types are recommended.

## REFERENCES

1. Stafstrom CE. Pathophysiological mechanisms of seizures and epilepsy: A primer. In *Epilepsy: Mechanisms, models, and translational perspectives* (ed. Rho JM, Sankar R, Stafstrom CE), 2010, CRC, Boca Raton, FL, pp. 3–19.
2. Haurer Stephen L. In: Harrison's Neurology In clinical Medicine. 2013, USA: Mc Graw Hill Education.
3. Calvi LM, Bushinsky DA. When is it appropriate to order an ionized calcium? *J Am Soc Nephrol*. 2008;19(7):1257–60.
4. Gareri P, Mattace R, Nava F, De Sarro G. Role of calcium in brain aging. *Gen Pharmacol*. 1995 Dec;26(8):1651–7.
5. Swaminathan R. Magnesium Metabolism and its Disorders. *The Clinical Biochemist Reviews*. 2003;24(2):47–66.
6. Shaikh A, Berndt T, Kumar R. Regulation of phosphate homeostasis by the phosphatonins and other novel mediators. *Paediatr Nephrol* 2008; 23: 1203–10.
7. Schwartzkroin PA, Baraban SC, Hochman DW. Osmolarity, ion flux, and changes in brain excitability. *Epilepsy Research* 1998; 32:275–285.
8. Castilla-Guerra L, del Carmen Fernández-Moreno M, López-Chozas JM, Fernández-Bolaños R. Electrolytes disturbances and seizures. *Epilepsia*. 2006 Dec;47(12):1990–8. Link
9. Nadkarni J, Binaykiya I, Sharma U, Dwivedi R. Role of serum sodium levels in prediction of seizure recurrence within the same febrile illness. *Neurology Asia* 2011; 16(3) : 195 – 197. Link
10. Halawa I, Andersson T, Tomson T. Hyponatremia and risk of seizures: a retrospective cross-sectional study. *Epilepsia*. 2011 Feb;52(2):410–3. Link
11. Usha Kiran CB, Suresh R. Reduced serum calcium is a risk factor for febrile seizures. *Int J Contemp Pediatr* 2017;4:1506–8. Link
12. Charan J, Biswas T. How to calculate sample size for different study designs in medical research? *Indian J Psychol Med*. 2013 Apr;35(2):121–6.
13. Kwak BO, Kim K, Kim SN, Lee R. Relationship between iron deficiency anemia and febrile seizures in children: A systematic review and metaanalysis. *Seizure*. 2017 Nov; 52:27–34. Link
14. Rama Krishna C, Basha SJ, Venkateswara Rao B, Preethi B. Serum Magnesium Levels in Seizure Disorders. *Journal of Evolution of Medical and Dental Sciences* 2014; 3(35): 9313-9319. Link
15. Namakian K, Zardast M, Sharifzadeh G, Bidar T, Zargarian S. Serum Trace Elements in Febrile Seizure: A Case-Control Study. *Iran J Child Neurol*. 2016;10(3):57–60. Link
16. Chen BB, Prasad C, Kobrzyński M, Campbell C, Filler G. Seizures Related to Hypomagnesemia: A Case Series and Review of the Literature. *Child Neurol Open*. 2016;3:2329048X16674834. Link
17. Abdullahi I, Watila M M, Shahi N, Nyandaiti Y W, Bwala S A. Serum magnesium in adult patients with idiopathic and symptomatic epilepsy in Maiduguri, Northeast Nigeria. *Niger J Clin Pract* 2019;22:186-93. Link
18. Antony A, Nathan J. Clinical Profile of Seizure Disorder in Patients Hospitalised in Kanyakumari Government Medical College. *JMSCR* 2019; 7(3): 545-548. Link
19. Sarkis RA, Jehi L, Silveira D, Janigro D, Najm I. Patients with generalised epilepsy have a higher white blood cell count than patients with focal epilepsy. *Epileptic Disord*. 2012 Mar;14(1):57–63. Link
20. Prabhakar S, Bhatia R. Management of agitation and convulsions in hepatic encephalopathy. *Indian J Gastroenterol*. 2003 Dec;22 Suppl 2:S54-Link
21. Mohanta SK, Naik M, Murmu M. Study of Serum Magnesium and Calcium Levels in Patients with new onset Seizures. *JMSCR* 2019; 7(3): 1320-1326. Link
22. Dhadke VN, Dhadke SV, Dawar A. Clinical profile of seizure disorder in hospitalized patients. *Int J Adv Med* 2016;3:275–81. Link
23. Sambyal V, Chandail VS. A study of clinical profile of seizure disorder at tertiary care centre a retrospective cross sectional study. *International Journal of Current Research* 2019; 11(5): 2541-2543. Link
24. Ahmed SN, Siddiqi ZA. Antiepileptic drugs and liver disease. *Seizure*. 2006 Apr 1;15(3):156–64.
25. Medeiros FC, Moraes AC, Bicalho AL, Pinto TV, Dahy FE. Cerebral venous sinus thrombosis presenting with subarachnoid hemorrhage: a series of 11 cases. *Acta Neurologica Belgica*. 2023 Jun;123(3):911–6.
26. Stafstrom CE. Persistent sodium current and its role in epilepsy. *Epilepsy Curr*. 2007;7(1):15–22. Link
27. Han P, Trinidad BJ, Shi J. Hypocalcemia-induced seizure: demystifying the calcium paradox. *ASN Neuro*. 2015;7(2):1759091415578050. Link
28. Rajakulendran S, Hanna MG. The Role of Calcium Channels in Epilepsy. *Cold Spring*

- Harb Perspect Med. 2016;6(1):a022723. Link
29. Sharma S, Fiza B, Tiwari SK, Sinha M. Evaluation of Serum Calcium Levels in Patients with Epileptic Seizure. J Mahatma Gandhi Univ Med Sci Tech 2020;5(2):35-37. Link
30. Janoff A. Elastases and emphysema: current assessment of the protease-antiprotease hypothesis. American Review of Respiratory Disease. 1985 Aug;132(2):417-33.
31. Vachharajani TJ, Zaman F, Abreo KD. Hyponatremia in critically ill patients. J Intensive Care Med. 2003 Jan-Feb;18(1):3-8. Link
32. Padhi R, Panda BN, Jagati S, Patra SC. Hyponatremia in critically ill patients. Indian J Crit Care Med. 2014;18(2):83-87. Link
33. Tan WW, Chan DWS, Lee JH, Thomas T, Menon AP, Chan YH. Use of Magnesium Sulfate Infusion for the Management of Febrile Illness-Related Epilepsy Syndrome: A Case Series. Child Neurol Open. 2015;2(1):2329048X14550067. Link