

## Hematological and biochemical parameters in various Clinical and Pharmacological types of Childhood Epilepsy

Asmaa A. Abdelfatah<sup>a\*</sup>, Ahmed El-Abd Ahmed<sup>a</sup>, Mohammed H. Hassan<sup>b</sup>, Ali H. Bakry<sup>a</sup>

<sup>a</sup>Department of Pediatrics, Faculty of Medicine, South Valley University, Qena, Egypt.

<sup>b</sup>Department of Medical Biochemistry, Faculty of Medicine, South Valley University, Qena, Egypt.

### Abstract

**Background:** Pediatric epilepsy, treated with antiepileptic drugs (AEDs), can lead to significant metabolic disturbances, impacting hematological and biochemical parameters vital for child health management.

**Objectives:** This study evaluates the effect of epilepsy types on hematological and biochemical markers in children, aiming to understand the metabolic side effects of AEDs.

**Patients and methods:** Conducted at Qena University Hospital, this cross sectional study involved 50 children categorized into 5 subgroups pharmacoresponsive cases vs. pharmacoresistant cases, cases with focal onset seizures vs cases with generalized onset seizures, cases managed with single antiepileptic drug vs cases managed with multiple antiepileptic drugs, cases with normal EEG vs cases with abnormal EEG and cases with epileptic discharge vs cases without epileptic discharge. Parameters including CBC, serum ionized calcium, serum sodium, serum potassium, serum creatinine, AST, and ALT levels were measured.

**Results:** Focal onset seizures were associated with lower hemoglobin and higher ionized calcium level compared to generalized onset seizures. No significant differences were noted in the majority of parameters between pharmacosensitive and -resistant groups, cases with single antiepileptic drug compared to cases managed with multiple antiepileptic drugs, cases with normal EEG and cases with abnormal EEG or cases with epileptic discharge vs cases without epileptic discharge.

**Conclusion:** The study reveals that epilepsy and its treatment with AEDs have an influence on the assessed hematological and biochemical parameters in children, with specific attention to the type of epilepsy and seizure onset. These findings support a tailored approach to monitoring and managing children with epilepsy, ensuring both seizure control and overall health preservation.

**Keywords:** Pediatric Epilepsy; Metabolic effects; Hematological parameters; Biochemical parameters.

\*Correspondence: asma.asma9902@gmail.com

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## Introduction

Epilepsy is one of the most common neurological diseases that affects people of all ages, races, social classes, and geographical locations, with a significant portion being children.(**Beghi, 2020**). In epilepsy, monitoring hematological and biochemical parameters is vital for assessing the impact of seizures on the body and guiding treatment. Hemoglobin levels (Hgb) are crucial for evaluating oxygen supply, particularly during and after seizures when oxygen levels may be affected. Serum creatinine (S. Creat) levels help assess kidney function, which can be affected by seizure-related dehydration or medication side effects. Electrolytes like sodium (Na+) and potassium (K+) are essential for nerve function and are closely monitored as seizures can disrupt their balance, leading to neurological complications. Additionally, ionized calcium (Ca+) levels are significant for muscle and nerve signaling and may be altered during seizure activity. Liver enzymes such as alanineaminotransferase (ALT) and aspartateaminotransferase (AST) are also monitored to detect any medication-induced liver damage, as certain antiepileptic drugs can affect liver function. Regular monitoring of these parameters helps clinicians understand the impact of seizures on the body and adjust treatment plans accordingly to optimize seizure control and minimize potential adverse effects(**Bhosale et al., 2014; Aydın et al., 2022**).

The liver significantly metabolizes many of the antiepileptic drugs (AEDs), raising concerns about hepatotoxicity during therapy initiation, compounded by the potential impact of liver disease on drug biotransformation. While drug-induced liver injury linked to AEDs is recognized, its frequency is low; however, severe consequences like acute liver failure necessitating liver transplantation or leading

to death underscore the importance of caution in managing potential liver-related complications(**Vidaurre et al., 2017; Kamitaki et al., 2021**).

The main aim of the study was to assess the influence of different types of epilepsy in children on hematological and biochemical markers such as hemoglobin, serum creatinine, ionized calcium, serum sodium, serum potassium, AST and ALT.

## Patients and methods

**Study design:** Cross sectional study.

**Study setting:** Pediatric Department and Neuropediatric Outpatient Clinic of Pediatric Department-Qena university hospital.

**Study subjects:** This study was carried on 50 subjects (fifty pediatric patients with epilepsy).

**Inclusion criteria:** All pediatric patients aged > 1 month to 18 years old presented with epilepsy and attending pediatric department and neuropediatric outpatient clinic of pediatric department at Qena University Hospital.

**Exclusion criteria:** Patients who had non-epileptic disorders, including electrolyte disturbances, metabolic disorders, acute brain disease or trauma and epilepsy in adults.

These subjects are subdivided clinically, pharmacologically (according to response to AEDs) and by imaging based on EEG findings into 5 subgroups (pharmacoresponsive cases vs. pharmaco-resistant cases, cases with focal onset seizures vs. cases with generalized onset seizures, cases managed with single antiepileptic drug vs. cases managed with multiple antiepileptic drugs, cases with normal EEG vs. cases with abnormal EEG and cases with epileptic discharge vs. cases without epileptic discharge.

All patients underwent a comprehensive assessment, encompassing detailed history-taking and physical

examinations. The personal history included demographic details such as age and sex, while the present history covered seizure-related information, post-ictal symptoms, seizure frequency, and duration. Additionally, past history of status epilepticus and febrile seizures and family history of epilepsy were documented. The medical history focused on antiepileptic drug usage. BMI and neurological examination was conducted.

### Investigations

1-Hematological and biochemical investigations: CBC, ionized calcium, serum sodium, serum potassium, serum creatinine, AST & ALT levels were measured.

2-Neuroimaging: EEG and CT or MRI brain scans were used to rule out other neurological problems.

**Ethical considerations:** Study was approved by the local Ethical Committee of Faculty Of Medicine, South Valley University. Informed and written consents were taken from patient's parents or caregivers.

### Statistical analysis

Data were checked, entered and analyzed using SPSS version 20.0 for data processing. The following statistical methods were used for analysis of results of the present study, data were expressed as mean + standard deviation (SD) for quantitative one, then data were summarized using the

arithmetic mean ( $\bar{X}$ ) as an average describing the central tendency of observations & the standard deviation (SD) as a measure of dispersion of the results around the mean. Finally, the comparison were done using the student "t" test for comparison of means of two independent groups, mann Whitney test was used to calculate difference between quantitative variables in not normally distributed data in two groups, Chi-square test ( $X^2$ ) used to find the association between row and column variables, Z-test for percentage to compare percentage of outcome between the two groups, odds ratio (OR): Compares the odds or the risk that a disease will occur among individuals who have a particular characteristic or who have been exposed to a risk factor to the Odds that the disease will occur in individuals who lack the characteristic or have not been exposed.

### Results

In the comparison of pharmaco-responsive (N = 25) and -resistant (N = 25) cases, laboratory data showed no significant differences in hemoglobin levels, serum creatinine, sodium, potassium, ionized calcium, ALT, AST between pharmaco-responsive and -resistant cases ( $p > 0.05$ ). There was a borderline significance in ALT levels ( $p = 0.0506$  [MWU]) in responsive cases ( $p = 0.065$  [MWU]), (**Table .1**).

**Table 1. Comparison between pharmaco responsive and resistant cases regarding laboratory data**

Variables	Responsive (N = 25)	Resistant (N = 25)	P. value
Hgb (gm/dl)	11.03 ± 1.48	10.76 ± 0.94	0.4583 <sup>[w,t]</sup>
	11.4 (9.6 - 12.1)	11 (10.3 - 11.3)	
S. Creat (mg/dl)	0.58 ± 0.11	0.62 ± 0.12	0.1684 <sup>[MWU]</sup>
	0.6 (0.49 - 0.7)	0.62 (0.48 - 0.72)	
Na+(mmol/l)	139.88 ± 2.5	139.24 ± 3.4	0.40299 <sup>[t]</sup>
	140 (138 - 141)	140 (137 - 141)	
K+(mmol/l)	4.18 ± 0.32	4.21 ± 0.44	0.7596 <sup>[t]</sup>
	4.2 (3.9 - 4.4)	4.2 (3.9 - 4.4)	
Ionized Ca+ (mmol/l)	1.12 ± 0.12	1.11 ± 0.08	0.7552 <sup>[MWU]</sup>

	1.1 (1 - 1.24)	1.1 (1.05 - 1.18)	
ALT(U/L)	35.28 ± 5.55	38.72 ± 9.36	0.0506 <sup>[MWU]</sup>
	37 (29 - 39)	42 (36 - 46)	
AST(U/L)	35.36 ± 9.38	33.28 ± 10.18	0.5187 <sup>[t]</sup>
	38 (29 - 42)	32 (25 - 40)	

In comparing cases with focal onset seizures (N = 8) and generalized onset seizures (N = 42), laboratory data demonstrated a significant decrease in hemoglobin levels in focal onset seizures (9.75 ± 1.05 gm/dl) compared to generalized onset seizures (11.12 ± 1.15 gm/dl, p = 0.0084 [MWU]). Ionized calcium levels

were significantly higher in focal onset seizures (1.2 ± 0.12 mmol/l) compared to generalized onset seizures (1.1 ± 0.09 mmol/l, p = 0.04299 [MWU]). Other parameters showed no significant differences between the groups (p > 0.05), (Table .2).

**Table 2. Comparison between cases with focal and generalized onset seizures regarding laboratory data**

Variables	Focal onset seizure	Generalized onset seizure	P. value
	(N = 8)	(N = 42)	
Hgb (gm/dl)	9.75 ± 1.05	11.12 ± 1.15	0.0084 <sup>[MWU]</sup>
	9.4 (9.225 - 9.9)	11.2 (10.325 - 11.8)	
S. Creat (mg/dl)	0.65 ± 0.06	0.59 ± 0.13	0.3113 <sup>[MWU]</sup>
	0.65 (0.595 - 0.7)	0.6 (0.465 - 0.7)	
Na+(mmol/l)	140.75 ± 2.77	139.33 ± 2.99	0.2452 <sup>[s.t]</sup>
	140 (139 - 144)	140 (137 - 141)	
K+(mmol/l)	4.24 ± 0.23	4.19 ± 0.41	0.6899 <sup>[s.t]</sup>
	4.3 (4.05 - 4.425)	4.2 (3.9 - 4.4)	
Ionized Ca+ (mmol/l)	1.2 ± 0.12	1.1 ± 0.09	0.04299 <sup>[MWU]</sup>
	1.27 (1.05 - 1.3)	1.1 (1.02 - 1.18)	
ALT(U/L)	37.5 ± 8.46	36.9 ± 7.77	0.8656 <sup>[s.t]</sup>
	39 (28 - 46)	37 (32.25 - 42)	
AST(U/L)	34.63 ± 12.07	34.26 ± 9.35	0.9413 <sup>[s.t]</sup>
	38 (28 - 44.5)	35.5 (28 - 40)	

Comparing cases managed with a single drug (N = 23) to those with multiple drugs (N = 27), no significant difference in

lab parameters showed between the two groups (p > 0.05) , (Table .3).

**Table 3. Comparison between cases with single or multiple drugs regarding laboratory data**

Variables	Cases managed with Single Drug (N = 23)	Cases managed with multiple Drugs (N = 27)	P. value
<b>Hgb (gm/dl)</b>	11.17 ± 1.46	10.83 ± 1.16	0.16999 <sup>[w.t]</sup>
	11.5 (10 - 12.1)	10.9 (10.11 - 11.3)	
<b>S. Creat (mg/dl)</b>	0.57 ± 0.11	0.61 ± 0.12	0.0586 <sup>[MWU]</sup>
	0.6 (0.465 - 0.68)	0.62 (0.54 - 0.71)	
<b>Na+(mmol/l)</b>	139.87 ± 2.61	139.59 ± 3.21	0.5032 <sup>[s.t]</sup>
	140 (138 - 141)	140 (137 - 141)	
<b>K+(mmol/l)</b>	4.16 ± 0.32	4.21 ± 0.4	0.4672 <sup>[s.t]</sup>
	4.2 (3.9 - 4.4)	4.2 (3.9 - 4.5)	
<b>Ionized Ca+ (mmol/l)</b>	1.1 ± 0.12	1.12 ± 0.1	0.2912 <sup>[MWU]</sup>
	1.07 (1 - 1.2)	1.1 (1.05 - 1.19)	
<b>ALT(U/L)</b>	35.91 ± 5.33	37.22 ± 8.32	0.2082 <sup>[MWU]</sup>
	37 (32 - 39)	39 (32 - 46)	
<b>AST(U/L)</b>	37.04 ± 7.75	33.85 ± 10.32	0.0666 <sup>[s.t]</sup>
	38 (30 - 43)	32 (23 - 40)	

Comparing cases with normal EEG (N = 5) to those with abnormal EEG (N = 45), no significant differences were observed in hemoglobin, serum creatinine, sodium or AST levels (p > 0.05). However,

a significant increase in potassium levels were noted in the abnormal EEG group (4.24 ± 0.38 mmol/l) compared to the normal EEG group (3.86 ± 0.14 mmol/l, p = 0.0282 [MWU]), (Table .4).

**Table 4. Comparison between cases with normal and abnormal EEG regarding laboratory data**

Variables	Cases with Normal EEG (N = 5)	Cases with abnormal EEG (N = 45)	P. value
<b>Hgb (gm/dl)</b>	11.44 ± 0.72	10.84 ± 1.27	0.1866 <sup>[s.t]</sup>
	11.8 (10.9 - 12.1)	11 (9.9 - 11.5)	
<b>S. Creat (mg/dl)</b>	0.54 ± 0.14	0.61 ± 0.12	0.3368 <sup>[MWU]</sup>
	0.46 (0.4 - 0.7)	0.6 (0.5 - 0.7)	
<b>Na+(mmol/l)</b>	139.6 ± 2.87	139.56 ± 3.02	0.9776 <sup>[s.t]</sup>
	138 (138 - 140)	140 (137 - 141)	
<b>K+(mmol/l)</b>	3.86 ± 0.14	4.24 ± 0.38	0.0282 <sup>[MWU]</sup>

	3.9 (3.9 - 3.9)	4.2 (3.9 - 4.5)	
<b>Ionized Ca+ (mmol/l)</b>	1.07 ± 0.08	1.12 ± 0.1	0.31499 <sup>[MWU]</sup>
	1.05 (1 - 1.11)	1.1 (1.03 - 1.2)	
<b>ALT(U/L)</b>	30.6 ± 10.37	37.71 ± 7.22	0.245 <sup>[s.t]</sup>
	29 (29 - 35)	38 (33 - 44)	
<b>AST(U/L)</b>	33.6 ± 9.18	34.4 ± 9.91	0.875 <sup>[s.t]</sup>
	30 (30 - 39)	36 (28 - 42)	

No significant differences were observed in hemoglobin levels (11.39 ± 1.53 vs. 10.72 ± 1.07 gm/dl, p = 0.1801), sodium concentration (139.62 ± 3.63 vs. 139.54 ± 2.75 mmol/l, p = 0.9486), potassium levels (4.12 ± 0.29 vs. 4.23 ± 0.41 mmol/l, p = 0.3), ionized calcium (1.06 ± 0.07 vs. 1.13 ± 0.11 mmol/l, p = 0.1618), alanine

aminotransferase (ALT) levels (36.62 ± 8.96 vs. 37.14 ± 7.47 U/L, p = 0.9205), and aspartate aminotransferase (AST) levels (35.38 ± 8.91 vs. 33.95 ± 10.12 U/L, p = 0.6445). Moreover, no significant differences were observed in serum creatinine levels (0.55 ± 0.11 vs. 0.62 ± 0.12 mg/dl, p = 0.1244), (**Table .5**).

**Table 5. Comparison between cases with and without epileptic discharge regarding lab data**

Variables	Cases with epileptic discharge (N = 13)	Cases without epileptic discharge (N = 37)	P. value
<b>Hgb (gm/dl)</b>	11.39 ± 1.53	10.72 ± 1.07	0.1801[s.t]
	12 (10.3 - 12.1)	10.8 (10.1 - 11.5)	
<b>S. Creat (mg/dl)</b>	0.55 ± 0.11	0.62 ± 0.12	0.1244[MWU]
	0.6 (0.44 - 0.65)	0.6 (0.5 - 0.7)	
<b>Na+(mmol/l)</b>	139.62 ± 3.63	139.54 ± 2.75	0.9486[s.t]
	138 (137 - 141)	140 (138 - 141)	
<b>K+(mmol/l)</b>	4.12 ± 0.29	4.23 ± 0.41	0.3[s.t]
	4.2 (3.9 - 4.4)	4.2 (3.9 - 4.5)	
<b>Ionized Ca+ (mmol/l)</b>	1.06 ± 0.07	1.13 ± 0.11	0.1618[MWU]
	1.07 (1.02 - 1.11)	1.1 (1.03 - 1.2)	
<b>ALT(U/L)</b>	36.62 ± 8.96	37.14 ± 7.47	0.9205[MWU]
	37 (35 - 40)	38 (32 - 44)	
<b>AST(U/L)</b>	35.38 ± 8.91	33.95 ± 10.12	0.6445[s.t]
	35 (30 - 39)	36 (27 - 42)	

**Discussion**

In terms of laboratory data, hemoglobin levels exhibited a significant reduction in cases with focal onset seizures (9.75 ± 1.05

gm/dl) compared to those with generalized onset seizures (11.12 ± 1.15 gm/dl, p = 0.0084). However, no significant differences were observed in hemoglobin levels when

comparing pharmacoresponsive cases ( $11.03 \pm 1.48$  gm/dl) with pharmacoresistant cases ( $10.76 \pm 0.94$  gm/dl,  $p = 0.4583$ ). Similarly, no significant variances in hemoglobin levels were reported between cases managed with a single drug ( $11.17 \pm 1.46$  gm/dl) compared to cases managed with multiple drugs ( $10.83 \pm 1.16$  gm/dl,  $p = 0.16999$ ). Furthermore, no significant differences in hemoglobin levels between cases with a normal EEG ( $11.44 \pm 0.72$  gm/dl) and cases with an abnormal EEG ( $10.84 \pm 1.27$  gm/dl,  $p = 0.1866$ ). Finally, no significant differences in hemoglobin levels were observed between cases with epileptic discharge ( $11.39 \pm 1.53$  gm/dl) and cases without epileptic discharge ( $10.72 \pm 1.07$  gm/dl,  $p = 0.180$ ).

In line with our finding, the study conducted by **Fallah et al. (2014)** involving 150 children distributed equally into three groups (febrile seizures, afebrile seizures, and control), the analysis of hemoglobin levels revealed that both the febrile seizures group and the afebrile seizures group exhibited lower hemoglobin levels compared to the control group. This suggests a potential association between seizures and decreased hemoglobin levels in children.

In our study, in this comparison between pharmacoresponsive and pharmacoresistant epilepsy cases (25 each), no significant differences were observed in serum creatinine levels ( $0.58 \pm 0.11$  vs  $0.62 \pm 0.12$  mg/dl respectively,  $p = 0.1684$ ). In the comparison between cases with focal and generalized onset seizures (8 and 42 cases, respectively), no significant differences were found in serum creatinine levels ( $0.65 \pm 0.06$  vs  $0.59 \pm 0.13$  mg/dl respectively,  $p = 0.3113$ ).

In the comparison between cases managed with single versus multiple drugs (23 and 27 cases, respectively), no statistically significant differences were

observed in serum creatinine levels ( $0.57 \pm 0.11$  vs  $0.61 \pm 0.12$  mg/dl respectively,  $p = 0.0586$ ). In the comparison between cases with normal and abnormal EEG findings (5 and 45 cases, respectively), no significant differences were observed in serum creatinine levels ( $0.54 \pm 0.14$  vs  $0.61 \pm 0.12$  mg/dl respectively,  $p = 0.3368$ ). In the comparison between cases with epileptic discharge and those without (13 and 37 cases, respectively), no statistically significant differences were observed in serum creatinine levels ( $0.55 \pm 0.11$  vs  $0.62 \pm 0.12$  mg/dl respectively,  $p = 0.1244$ ).

However, **Hamed et al. (2018)** study, aimed to assess clinical and subclinical laboratory evidence of renal proximal tubular dysfunction in 60 epilepsy patients as an adverse effect linked to certain antiepileptic drugs (AEDs). Their findings indicate that individuals with epilepsy demonstrated elevated serum creatinine (sCr) levels compared to the control group.

In our study, in the comparison between cases with normal and abnormal EEG findings (5 and 45 cases, respectively), there was a significant difference in potassium levels ( $p = 0.0282$ ), with cases having abnormal EEG results showing higher potassium levels ( $4.24 \pm 0.38$  mmol/l) compared to those with normal EEG results ( $3.86 \pm 0.14$  mmol/l). However, in the comparison between pharmacoresponsive and pharmacoresistant epilepsy cases (25 each), no significant differences were observed in potassium levels ( $4.18 \pm 0.32$  vs  $4.21 \pm 0.44$  mmol/l respectively,  $p = 0.7596$ ). In the comparison between cases with focal and generalized onset seizures (8 and 42 cases, respectively), no significant differences were found in potassium levels ( $4.24 \pm 0.23$  vs  $4.19 \pm 0.41$  mmol/l respectively,  $p = 0.6899$ ). In the comparison between cases managed with single versus multiple drugs (23 and 27 cases, respectively), no statistically

significant differences were observed in potassium levels ( $4.16 \pm 0.32$  vs  $4.21 \pm 0.4$  mmol/l respectively,  $p = 0.4672$ ). In the comparison between cases with epileptic discharge and those without (13 and 37 cases, respectively), no statistically significant differences were observed in potassium levels ( $3.86 \pm 0.14$  vs  $4.24 \pm 0.38$  mmol/l respectively,  $p = 0.3$ ).

The significant difference in potassium levels between cases with normal and abnormal EEG findings may be attributed to various factors. These include potential fluctuations induced by seizure activity, as seizures can disrupt electrolyte balance, including potassium levels, through increased neuronal firing and medication effects. Additionally, individuals with abnormal EEG results may have more severe epilepsy or underlying neurological pathology, which could further impact potassium regulation. Moreover, the secondary effects of seizures, such as metabolic disturbances, could contribute to potassium level variations (Liu et al., 2020; Gao et al., 2022).

However, Tolou-Ghamari et al. (2013) assessed the impact of antiepileptic drugs, including Phenytoin, Valproate, and Carbamazepine on biochemical parameters in children aged 4 to 13 years. They found no significant differences in serum levels of potassium between children on antiepileptic drugs and controls.

In contrast, Ristić et al. (2014) conducted research on electrolyte levels in patients with drug-resistant mesial temporal lobe epilepsy compared to controls. They observed significantly decreased potassium levels in the epilepsy group.

In our study, ionized calcium levels displayed a significant increase in focal onset seizures ( $1.2 \pm 0.12$  mmol/l) compared to generalized onset seizures ( $1.1 \pm 0.09$  mmol/l,  $p = 0.04299$ ). However, no significant differences were reported in

ionized calcium levels when comparing pharmacoresponsive cases ( $1.12 \pm 0.12$  mmol/l) with pharmacoresistant cases ( $1.11 \pm 0.08$  mmol/l,  $p = 0.7552$ ). Additionally, no significant differences were noted in ionized calcium levels when comparing cases managed with a single drug ( $1.1 \pm 0.12$  mmol/l) to those managed with multiple drugs ( $1.12 \pm 0.1$  mmol/l,  $p = 0.2912$ ). Furthermore, no significant differences in ionized calcium levels between cases with a normal EEG ( $1.07 \pm 0.08$  mmol/l) and cases with an abnormal EEG ( $1.12 \pm 0.1$  mmol/l,  $p = 0.31499$ ). Finally, no significant differences were detected in ionized calcium levels between cases with epileptic discharge ( $1.06 \pm 0.07$  mmol/l) and cases without abnormal epileptic discharge ( $1.13 \pm 0.11$  mmol/l,  $p = 0.1618$ ).

In line with our results, the study conducted by Tombini et al. (2018) the findings confirm an increase in calcium levels in patients with epilepsy. It's worth noting that their analysis did not encompass a comparison of different seizure types. This highlights the relevance of our study's contribution in potentially addressing this gap and providing a more comprehensive understanding of the relationship between calcium metabolism and seizure types.

Tolou-Ghamari et al. (2013) investigated the effects of antiepileptic drugs such as Phenytoin, Valproate, and Carbamazepine on biochemical markers in children aged 4 to 13 years. Their study revealed no notable variances in calcium serum levels between children undergoing antiepileptic treatment and the control group.

In our study, no significant differences were observed in sodium levels when comparing pharmacoresponsive cases ( $139.88 \pm 2.5$  mmol/l) with pharmacoresistant cases ( $139.24 \pm 3.4$  mmol/l,  $p = 0.40299$ ). Furthermore, sodium levels showed no significant variances between focal onset



seizures ( $140.75 \pm 2.77$  mmol/l) and generalized onset seizures ( $139.33 \pm 2.99$  mmol/l,  $p = 0.2452$ ). Additionally, no significant differences were noted in sodium levels between cases managed with a single drug ( $139.87 \pm 2.61$  mmol/l) and cases managed with multiple drugs ( $139.59 \pm 2.61$  mmol/l,  $p = 0.5032$ ). Moreover, no significant differences in sodium levels between cases with a normal EEG ( $139.6 \pm 2.87$  mmol/l) and cases with an abnormal EEG ( $139.56 \pm 3.02$  mmol/l,  $p = 0.9776$ ). Finally, no significant differences in sodium levels between cases with epileptic discharge ( $139.62 \pm 3.63$  mmol/l) and cases without abnormal epileptic discharge ( $139.54 \pm 2.75$  mmol/l,  $p = 0.9486$ ).

In line with our results, **Tolou-Ghamari et al. (2013)** assessed the impact of antiepileptic drugs, including Phenytoin, Valproate, and Carbamazepine, on biochemical parameters in children aged 4 to 13 years. They found no significant differences in serum levels of sodium between children on antiepileptic drugs and controls.

**Kaplan et al. (2016)** reported that both increases and decreases in sodium levels can influence neuronal firing thresholds, contributing to the hyperexcitability seen in epilepsy.

In contrast, **Ristić et al. (2014)** conducted research on electrolyte levels in patients with drug-resistant epilepsy compared to controls. They observed significantly increased sodium levels in the epilepsy group.

In our study, no significant differences were observed in AST levels when comparing pharmacoresponsive cases ( $35.36 \pm 9.38$  U/L) with pharmacoresistant cases ( $33.28 \pm 10.18$  U/L,  $p = 0.5187$ ). Similarly, AST levels showed no significant differences between focal onset seizures ( $34.63 \pm 12.07$  U/L) and generalized onset seizures ( $34.26 \pm 9.35$  U/L,  $p = 0.9413$ ).

Additionally, no significant differences were noted in AST levels between cases managed with a single drug ( $37.04 \pm 7.75$  U/L) and cases managed with multiple drugs ( $33.85 \pm 10.32$  U/L,  $p = 0.0666$ ). Furthermore, no significant differences in AST levels between cases with a normal EEG ( $33.6 \pm 9.18$  U/L) and cases with an abnormal EEG ( $34.4 \pm 9.91$  U/L,  $p = 0.875$ ). Finally, no significant differences in AST levels between cases with epileptic discharge ( $35.38 \pm 8.91$  U/L) and cases without abnormal epileptic discharge ( $33.95 \pm 10.12$  U/L,  $p = 0.6445$ ).

Regarding ALT, no significant differences were observed in ALT levels when comparing pharmacoresponsive cases ( $35.28 \pm 5.55$  U/L) with pharmacoresistant cases ( $38.72 \pm 9.36$  U/L,  $p = 0.0506$ ). Similarly, ALT levels showed no significant differences between focal onset seizures ( $37.5 \pm 8.46$  U/L) and generalized onset seizures ( $36.9 \pm 7.77$  U/L,  $p = 0.8656$ ).

Additionally, no significant differences were noted in ALT levels between cases managed with a single drug ( $35.91 \pm 5.33$  U/L) and cases managed with multiple drugs ( $37.22 \pm 8.32$  U/L,  $p = 0.2028$ ). Furthermore, no significant differences in ALT levels between cases with a normal EEG ( $30.6 \pm 10.37$  U/L) and cases with an abnormal EEG ( $37.71 \pm 7.22$  U/L,  $p = 0.245$ ). Finally, no significant differences in AST levels between cases with epileptic discharge ( $36.62 \pm 8.96$  U/L) and cases without abnormal epileptic discharge ( $37.14 \pm 7.47$  U/L,  $p = 0.6445$ ).

In contrast, the study by **Khubchandani et al. (2014)** in adults indicates that serum levels of aminotransferase enzymes (both AST & ALT) in epilepsy cases were significantly higher ( $p < 0.05$ ) compared to the levels observed in controls.

### Conclusion

Epileptic types among children can significantly affect various hematological

and biochemical parameters so monitoring those patients by CBC, liver function test, kidney function test and electrolytes (serum sodium, potassium and ionized calcium) is an important issue in their management to avoid significant health problems.

#### References

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