An Overview of Neonatal Seizures in the Neonatal Intensive Care Unit of Assiut University Children's Hospital: A Cross-sectional Study

Ahmed Mohamed Salama, Emad El-Deen Mahmoud Hammad,
Safwat Mohammed Abdel-Aziz
Pediatrics Department, Faculty of Medicine, Assiut University, Assiut, Egypt
Corresponding author: Ahmed Mohamed Salama
E-mail: *ahmed01162@med.aun.edu.eg*

Abstract:

Seizures are critical indicators of neurological dysfunction in newborns. They are characterized by a temporary manifestation of signs and/or symptoms resulting from abnormally excessive or synchronous neuronal activity in the brain. Seizures in neonates can be categorized into several types: clinical-only seizures, electro-clinical seizures, electrographic-only seizures, epilepsy, and status epilepticus..[1]

Patients and Methods

This hospital-based cross-sectional study was conducted in the Neonatal Intensive Care Unit of Assiut University Children's Hospital from July 2022 to March 2023, lasting about 9 months without interruptions.

Results and Conclusion

The findings of this study contribute significantly to the understanding of neonatal seizures. The study demonstrates that 19.5% of neonates experienced clinical seizures, primarily subtle in nature, alongside the identification of hypoxic ischemic encephalopathy as the predominant etiological factor. The study's EEG evaluations revealed various patterns, including burst-suppression and generalized discharge among the participant neonates showing convulsive phenomena. Importantly, there is a statistically significant difference in the maturity and birth weight of convulsing versus non-convulsing neonates, in contrast to the lack of significant differences regarding mean gestational age, sex, and mode of delivery, which points to specific risk factors associated with neonatal seizures. These results underline the necessity for thorough and multi-dimensional assessments in diagnosing and managing neonatal seizures, providing valuable insights for clinical practices and future research.

Keywords: Neonatal seizures; Electroencephalography (EEG); Hypoxic ischemic encephalopathy; Intracranial hemorrhage; Antiepileptic drugs; Phenobarbital; Neurodevelopmental outcome.

Introduction:

Background

Seizures are critical indicators of neurological dysfunction in newborns. They are characterized by a temporary manifestation of signs and/or symptoms resulting from abnormally excessive or synchronous neuronal activity in the brain. Seizures in neonates can be categorized into several types: clinical-only seizures, electro-clinical seizures, electrographic-only seizures, epilepsy, and status epilepticus. [1]

According to clinical observation, neonatal seizures are classified into subtle, tonic, clonic, and myoclonic.[2] The incriminated causes of acute symptomatic seizures in neonates include: hypoxic ischemic encephalopathy, ischemic stroke, neonatal epilepsy, and intracranial hemorrhage,

transient metabolic derangements, and central nervous system (CNS) infection, among other rare causes.[3] Neonatal seizures are diagnosed based on observational clinical findings, laboratory tests, and neuroimaging studies. Conventional electroencephalogram (EEG) remains the gold standard for detecting seizure phenomena in neonates.[4] Neonatal seizures are known to be associated with generally poor neurodevelopmental outcomes and future epileptic sequelae at a rate of approximately 20%. However, the risk of death or specific neurodevelopmental disability is still difficult to estimate.[5]

Many anti-epileptic drugs have been used to treat neonatal seizures with great caution to avoid adversely affecting neurodevelopmental outcomes. Phenobarbital is the most frequently used anti-epileptic agent as a first line for seizure control; however, concerns are rising regarding its effect on the long-term neurodevelopment of neonates.[6]

The prognosis of neonatal seizures is a critical issue for all caregivers. The two primary factors in predicting infant outcomes are the infant's underlying neuropathological etiology and the recorded EEG pattern.[7] The infant's gestational age must reinforce discussions of probable outcomes with caregivers, the underlying neuropathology causing seizures, how easily the seizures are controlled with antiseizure methods, and the infant's EEG patterns supplemented by neuroimaging data.[8]

Aim of the Study

To evaluate the clinical seizures during the neonatal period, their frequency among the neonatal population admitted to the neonatal intensive care unit (NICU) of Assiut University Children's Hospital (AUCH), clinical types, etiological factors, and short-term outcomes (e.g., mortality and response to treatment).

Patients and Methods

- **Study design**: A hospital-based cross-sectional study carried out in the Neonatal Intensive Care Unit of Assiut University Children's Hospital from July 2022 to March 2023, lasting about 9 months without interruptions.
- **Study participants**: All neonates (preterm and full-term infants) aged 0 to 28 days admitted to the neonatal intensive care unit of Assiut University Children's Hospital during the study period.
- **Exclusion criterion**: Neonates with multiple congenital anomalies that are incompatible with life.
- **Sample size calculation**: Sample size was calculated using EPI Info 2000 statistical package based on an expected prevalence of seizures among neonates attending NICU of Assiut University Children's hospital of 2.7% with a difference of 1.35% and a confidence interval of 95%, the minimum sample size was calculated to be **554** neonates.
- The study was approved by the ethical committee of Assiut University, Faculty of Medicine (Institutional Review Board local approval number: 17101539)
- A chronological algorithm for the methodology of this study is shown below in **Figure (1).**

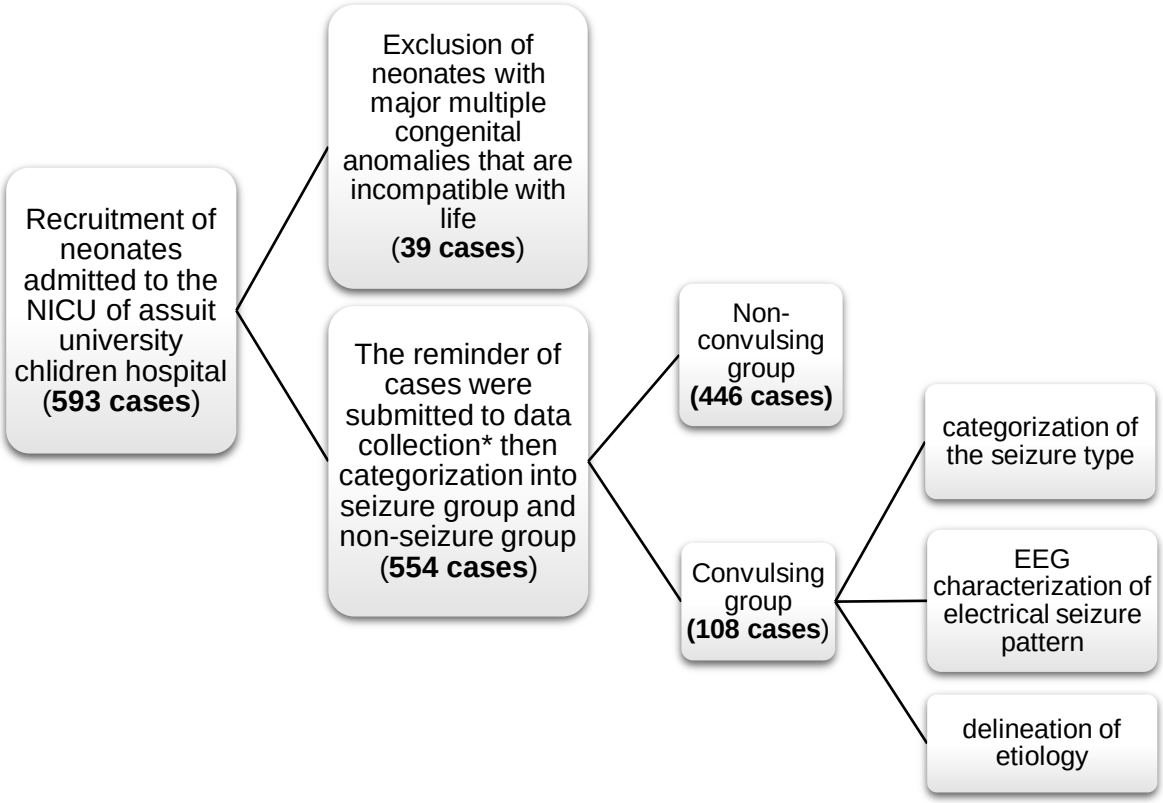

Figure (1): chronological algorithm of the methodology of the study.

The data collected includes:

1.<u>Demographics of participant neonates</u>:

a) **Maturity status and gestational age**: The included neonates are classified according to prenatal ultrasonographic examination in combination with the date of last menstrual period, supported by postnatal age assessment via the new Ballard scoring into:
- Preterm <37 weeks.
- Full term ≥37weeks

b) **Sex**: The included neonates are classified into males or females.

c) **Mode of delivery**: Cesarean section or normal vaginal delivery.

2.<u>Laboratory Investigations</u>:

a) **Complete blood count** (hemoglobin level, total leucocytic count, platelet count)

b) **Serum electrolytes**: calcium, sodium, and Magnesium.

c) **Mean random blood sugar.**

d) **Liver function tests**: Alanine transaminase, total serum bilirubin, total serum proteins, and serum albumin.

e) **Kidney function tests**: serum creatinine and blood urea nitrogen.

f) **Serum ammonia and lactate levels.**

g) **TORCH screening.**

3.<u>Neuroimaging Studies</u>:

a) **Transcranial Ultrasonography**: to assess for major anatomical brain anomalies, hydrocephalus, interventricular hemorrhage, etc.

b) **CT / MRI studies** (when there is a strong transcranial ultrasonographic evidence of the necessity of further neuroimaging evaluation, in addition, health status of the neonate is feasible for transfer to radiology unit): detect congenital abnormalities of the brain such as lissencephaly, pachygyria, and polymicrogyria, along with IVH with infarct, calcifications, white matter

abnormalities, and Hypoxic ischemic encephalopathy.

4. **EEG Characterization of Electrical Seizure Pattern**
5. **Categorization of the Seizure Type,** as evidenced by clinical observation, is supported by EEG findings.
6. **Delineation of the most probable underlying etiological factor** causing the seizure phenomenon.

Results

- According to **maturity status**, there were 359 (65%) preterm neonates and 195 (35%) full-term neonates.
- **Gestational age** ranged from 26 to 40 (mean 34.14).
- **Maturity status** ranged from 700 g to 4.5kg (mean 2.6).
- According to **sex**, there were 316 (57%) males and 238 (43%) females.
- According to the **mode of delivery,** there were 420 cesarean sections and 134 normal vaginal deliveries.
- A statistically significant difference was observed between the convulsing and non-convulsing groups concerning maturity status ($p = 0.027$), with neonates in the convulsing group being more frequently preterm.
- The two groups' birth weight comparison showed a borderline statistical difference ($p = 0.062$), with the convulsing group demonstrating a slightly higher mean birth weight than the non-convulsing group.
- There was NO statistically significant difference between the Convulsing and Non-Convulsing groups regarding gestational age, sex, and mode of delivery.
- According to the **occurrence of convulsive phenomena**, 19.5% of participant neonates experienced clinical or electrical convulsive phenomena. *This is 19.5%, which is the frequency of neonatal seizure occurrence in the NICU of Assiut University Children's Hospital.*
- Regarding the **etiological factors** for neonatal seizures there were 56 neonates (52%) with hypoxic ischemic encephalopathy, 17 neonates (15.7%) with intracranial hemorrhage, 12 neonates (11%) with CNS anomalies, 10 neonates (9.3%) with metabolic etiology, nine neonates (8.3%) with CNS infection, diagnosed in the context of neonatal sepsis and supported by elevated total leukocyte count and C-reactive protein (CRP), and 4 (3.7%) neonates with Neonatal Epilepsy Syndromes, Figure (2).
- Regarding the **clinical type of seizures,** 49 neonates, 45% of the convulsing group, experienced subtle seizure phenomena. Generalized forms that could be distinguished are generalized tonic (12 neonates, 11,1%), generalized tonic clonic (11 neonates, 10.2%), generalized clonic (5 neonates, 4,6%), and generalized myoclonic (3 neonates, 2.8%). Focal forms identified are focal tonic clonic (7 neonates, 6.5%), focal tonic (only 1 case, 0.9%), focal clonic (6 neonates, 5.6%), focal myoclonic (5 neonates, 4.6%), and focal with secondary generalization (3 neonates, 2.8%). Only two neonates experienced atonic forms, constituting 1.9% of the total convulsing neonates.
- According to **EEG evaluations**, out of 108 neonates in the convulsing group, there were 19 neonate (17.6%) who represented clinical only seizure type with no associated electrical correlates, 44 neonates (40.7%) with Burst suppression, 20 neonates (18.5%) with Generalized discharge, 17 neonates (15.7%) with Multifocal discharge, three neonates (2.8%) with Focal Centro-Tempro-Parietal discharge, and five neonates (4.7%) with Trace alternant, Figure (3).
- According to **Trans-Cranial Ultra/Sound** evaluation, results were normal for 324 neonates (58.5%), while there was 1st degree intraventricular hemorrhage in 54 neonates (9.7%), 2nd degree intraventricular hemorrhage in 101 neonates (18.2%), and 3rd degree intraventricular hemorrhage in 15 neonates (2.7%). Hydrocephalus represented 24 neonates (4.3%), ventriculitis eight neonates (1.4%), space-occupying lesion two neonates (0.6%), brain atrophy 13 neonates (2.4%), and periventricular leukomalacia 13 neonates (2.4%).

- A total of 84 neonates were investigated via **CT/MRI brain imaging** due to a suggestive clinical picture of central neurologic etiology. Twenty-five neonates (29.8%) showed normal CT/MRI studies. Further data are summarized in Table (1).

Table (1): Distribution of the studied neonates according to CT/MRI Brain imaging (No. = 84).

Brain CT/MRI conclusive imaging reports	No.	%
Normal study	25	29.8%
Abnormal CT/MRI findings:	59	70.2%
- Definite Hypoxic ischemic encephalopathy	22	26.2%
- Diffuse white matter hypodensity (HIE??)	10	11.9%
- Agenesis of the corpus callosum	2	2.4%
- Aqueductal stenosis with supratentorial Hydrocephalus	3	3.6%
- Holoprosencephaly	1	1.2%
- Communicating Hydrocephalus	6	7%
- Hydrocephalus with brain atrophy	3	3.6%
- Hydrocephalus with Dandy-Walker variant	1	1.2%
- Lissencephaly	2	2.4%
- Massive Hydrocephalus	3	3.6%
- Posterior occipital meningocele with Dandy-Walker variant	1	1.2%
- Subdural hematoma	2	2.4%
- Mild Hydrocephalic changes	3	3.6%

According to **laboratory investigations,** findings are summarized in Table (2).

Table (2): Distribution of the studied neonates according to laboratory investigations.

	Characteristics	No. = 554
Hemoglobin (mg/dL)	Mean ± SD	14.07 ± 2.88
	Range	5.7 – 20.7
TLC (cell/mcL)	Mean ± SD	12.39 ± 9.71
	Range	1.4 – 123
Plt (thrombocyte/mcL)	Median (IQR)	251.5 (170 – 347)
	Range	18 – 642
Glucose (mg/dL)	Mean ± SD	132.69 ± 77.45
	Range	39 – 421
Calcium (mg/dL)	Mean ± SD	8.35 ± 0.74
	Range	6.1 – 10
Sodium (mEq/L)	Mean ± SD	139 ± 69.6
	Range	118-179
Magnesium (mg/dL)	Mean ± SD	1.8 ± 0.69
	Range	0.8 – 3.3
ALT (U/L)	Median (IQR)	16 (12 – 23)
	Range	4 – 144

Table (2): Distribution of the studied neonates according to laboratory investigations. ***(Cont).***

	Characteristics	**No. = 554**
Mean Total Serum Bilirubin (mg/dL)	Median (IQR)	5 (3.7 – 7)
	Range	0.2 – 24
Total Protein (g/dL)	Mean ± SD	4.95 ± 0.98
	Range	3.2 – 6.7
Serum Albumin (g/dL)	Mean ± SD	3.35 ± 0.53
	Range	1.9 – 4.4
Serum Creatinine (mg/dL)	Median (IQR)	0.60 (0.40 – 0.80)
	Range	0.1 – 3.6
Blood Urea (mg/dL)	Median (IQR)	8.5 (5.1 – 13.1)
	Range	1.5 – 44
pH	Mean ± SD	7.36 ± 0.07
	Range	6.9 – 7.5

The median of platelet count was statistically significant (p-value = 0.032), Figure (4). The mean of Sodium of the convulsing group is 139 ± 19.6, and it is 135 ± 7.5 for neonates of the non-convulsing group, which is of *high statistical significance* (p-value = 0.001). The mean of Magnesium of the convulsing group is 1.4 ± 0.59, and it is 1.8 ± 0.69 for neonates of the non-convulsing group, which is of *high statistical significance* (p-value = 0.001). The mean of Serum Albumin for neonates of the convulsing group was 3.32 ± 0.42, and it was 3.18 ± 0.41 for neonates of the non-convulsing group, which was statistically *insignificant*.

Almost all non-convulsing groups have normal lactate levels (61 out of 62 neonates, 98.4%). The convulsing group has an equal split between normal and high lactate levels (6 neonates, 50% each). This was statistically significant (p-value = 0.001), Figure (5).

There was NO statistically significant difference between Convulsing and Non-Convulsing groups regarding Hemoglobin, TLC, Glucose, Calcium, ALT, mean Total Serum Bilirubin, Total Protein, Serum Albumin, Serum Creatinine, Blood Urea and PH, serum Ammonia, and TORCH Screening.

According to the **short-term outcome** of the neonates of the convulsing group, 12 neonates (11.1%) were discharged without anti-convulsant agents. Thirty-four neonates (31.5%) were discharged on a single anti-convulsant agent. Only nine neonates (8.3%) were discharged on ≥ 2 anti-convulsant agents. More than 50 cases died, 11 of them (10.2%) died due to the underlying disease that caused seizures, while 42 neonates (38.9%) died due to another disease unrelated to seizures. **Figure (6)**

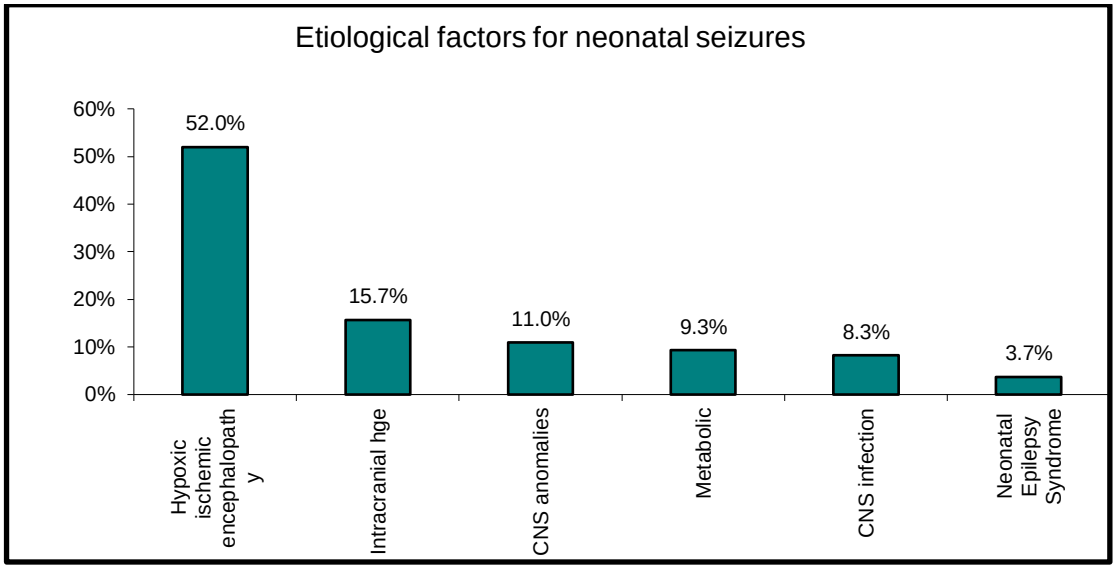

Figure (2): Distribution of the studied neonates according to Etiological factors for neonatal seizures.

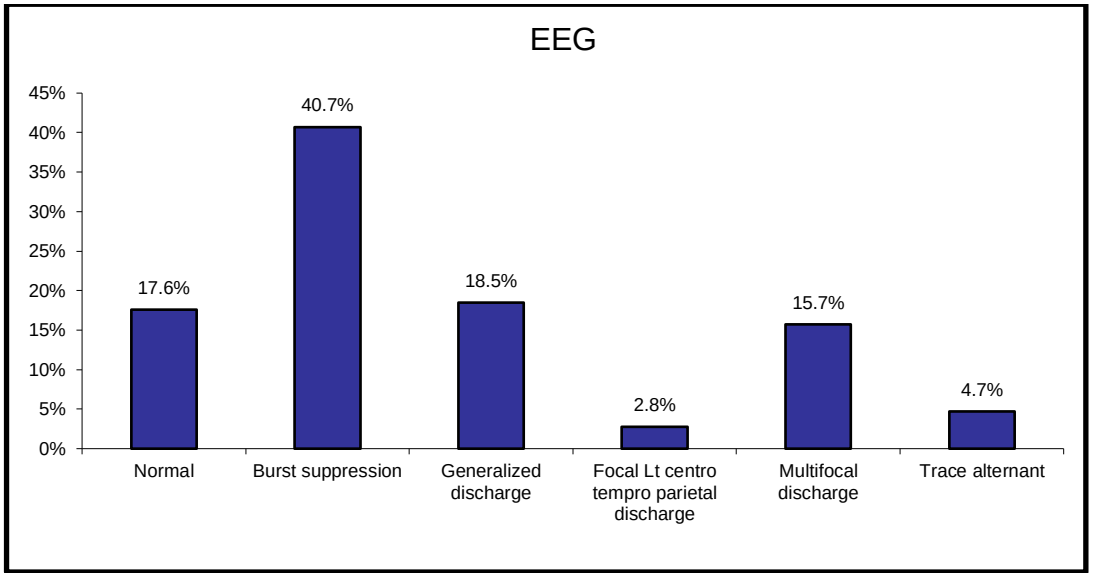

Figure (3): The studied convulsing neonates were distributed according to EEG evaluation.

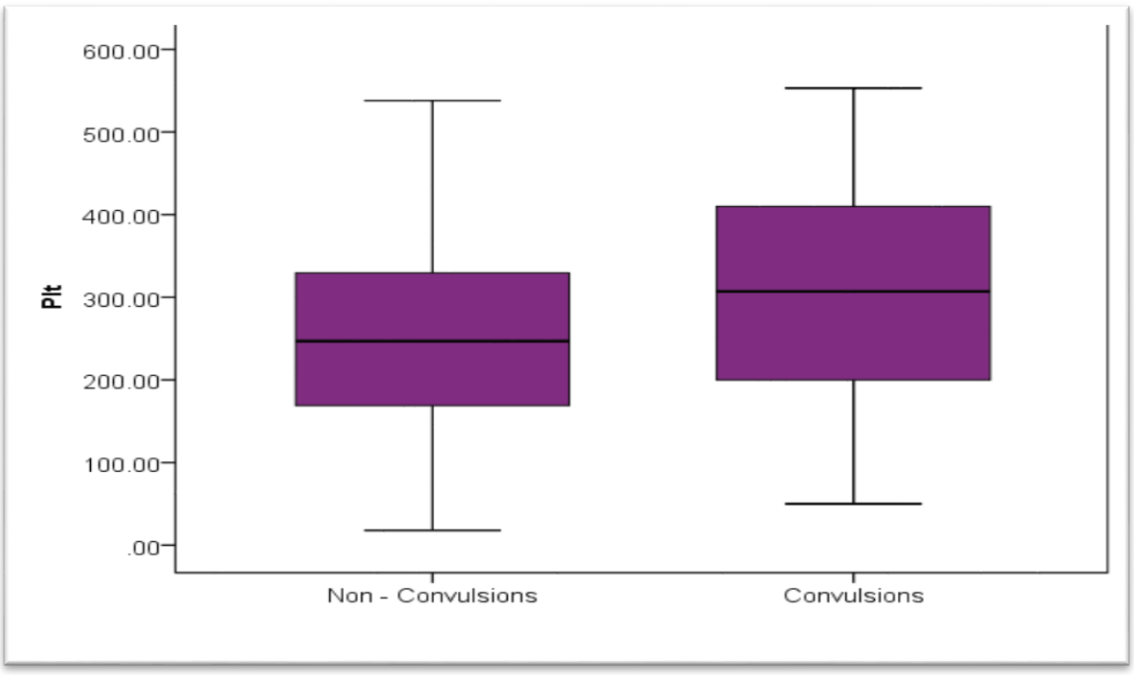

Figure (4): The difference between Convulsing and Non-Convulsing groups regarding platelet count.

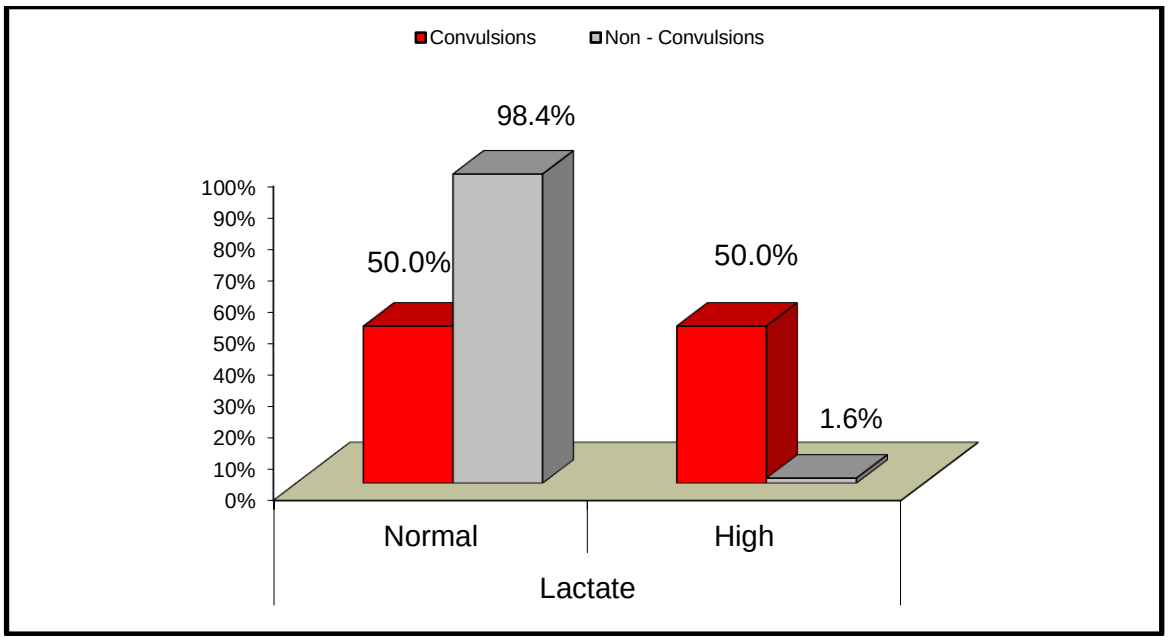

Figure (5): The difference between convulsing and non-convulsing groups regarding serum lactate levels.

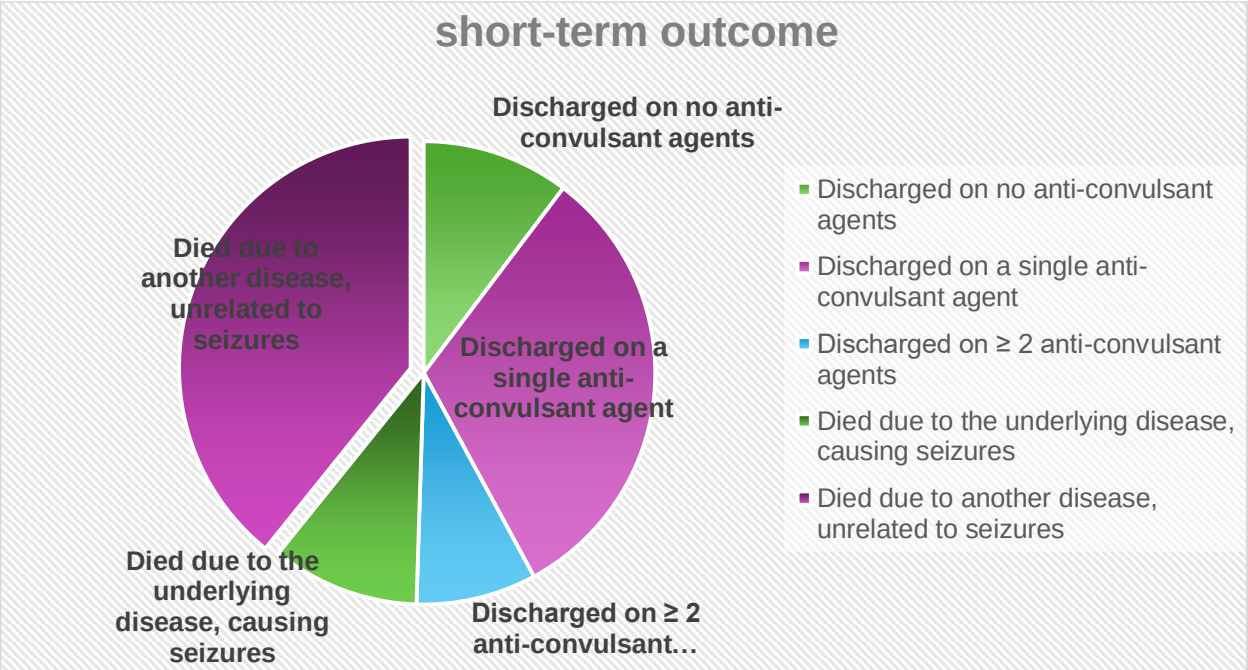

Figure (6): Distribution of the studied convulsing neonates according to short-term outcome

Discussion

Regarding demographics, in our study, 65% of neonates were preterm neonates and 35% were full-term neonates. Gestational age ranged from 26 to 40 (mean 34.14)**.** Birth weight ranged from 700 g to 4.5kg (mean 2.6). Males were 57% and females were 43%. As regards Mode of delivery, 76% delivered by C-section and 24% delivered by Normal vaginal delivery.

Lawgali et al. (2019), who aimed to determine frequency, natural history, etiological determinants, clinical variants, and the short-term outcomes of neonatal seizures, reported that 131 (87%) were term and 15 (10%) preterm. This differed from our study's 65% preterm neonates and 35% full-term neonates.[9] **Eghbalian et al. (2015**) reported that female infants comprised the majority of patients in this study. 76% of patients had more than 2.5 kilograms of weight, which agrees with our study.[10] **Dehdashtian et al. (2009**) reported that male infants comprised most patients experiencing seizures.[11] This agrees with our study results.

Regarding the clinical type of seizures, in our study, the frequency of neonatal seizure occurrence among participant neonates is 19.5% who experienced clinical or electrical convulsive phenomena. Subtle convulsions were the most common, occurring in 49 neonates (45.3%), indicating a high frequency of less obvious seizure activity.

Amare and Amare (2019), who aimed to assess the clinical pattern, etiological factors, and short-term outcomes of neonatal seizures in the neonatal intensive care unit, reported that subtle seizures were the most common variant of seizures in newborns, accounting for 60.7%.[12] This goes along with our study results. In agreement with **Abdelhaie et al. (2021),** who reported that subtle seizures were the most common clinical type of seizures noted in 29 (58.0%)

infants, followed by clonic type in 11 (22.0%) infants, and tonic 9 (18.0%) infants in their study.[13] In contrast to these results, **Fiaz et al. (2009)** studied 101 neonates experiencing seizures, of whom 39.6% had both tonic and clonic seizures as the predominant clinical seizure pattern.[14] Also, our results differed from studies in which clonic seizures were the most common. [15,16]

Regarding the etiological factors for neonatal seizures, in our study, 52% of convulsing neonates showed hypoxic ischemic encephalopathy as their primary etiology for their seizure phenomena, 15.7% with Intracranial hemorrhage, 11% with CNS anomalies, 9.3% with Metabolic etiology, 8.3% neonates with CNS infection, and 3.7% with Neonatal Epilepsy Syndromes.

Ghanshyambhai et al. (2016) found that the most common etiological factor of neonatal seizure was hypoxic ischemic encephalopathy (HIE). [17] This was in agreement with **Baudou et al. (2019**), who found the frequencies of etiologies of neonatal seizures were: 37% hypoxic ischemic encephalopathy, 12% ischemic parenchymal infarcts, 15% intracranial hemorrhage, 8% intracranial infections, 3% metabolic derangements, 2% inborn errors of metabolism, 5% congenital CNS malformations, and 11% neonatal epileptic syndromes.[18] **Abdelhaie et al. (2021)** reported that hypoxic ischemic encephalopathy was the most common etiological factor incriminated in neonatal seizures causality and was identified in 14 (28.0%) infants. The second most common cause was intracranial hemorrhage in 11 (22.0%) infants, followed by intracranial infection in 9 (18.0%) infants.[13] **Moayedi et al. (2007)** reported that sepsis was the main etiological factor in 85 neonates, accounting for about 60% of the convulsing cases, followed by neonatal encephalopathy in 30 neonates (21%).[15] **Marzoki (2010)** reported that metabolic derangements were the most common cause of seizures in neonates, as observed in 42 neonates (47.7%) in this study. Birth asphyxia was identified as the second most common cause, affecting 14 neonates (15.9%).[19]

Regarding EEG evaluations, in our study, burst suppression was the most common EEG pattern among convulsing neonates.

Zupanc et al. (2004) reported that EEG has been reported to be abnormal in 100% of cases experiencing clonic, partial tonic, and spasmic seizures; 60% of generalized myoclonic patterns; 7% of focal and multifocal myoclonic patterns, and 10% of generalized tonic seizures. However, subtle patterns of seizures did not show a specific correlation with EEG findings.[20] Studies on EEGs show that EEG-only seizures have no significant correlation with pathological EEG changes, whereas generalized seizure patterns (tonic, clonic, myoclonic) have almost always abnormal EEG findings. However, the value of these results in neonatal seizure management remains largely unclear.[21]

Regarding CT/MRI brain imaging evaluation, in our study, 26.2% a definite radiographic picture of hypoxic ischemic encephalopathy, and 11.9% had diffuse white matter hypodensity (query hypoxic ischemic insult), making HIE the most common radiological finding.

In **Taghdiri et al. (2005)**, 65% of neonates had abnormal CT scan results, with intracranial hemorrhage being the most common radiological finding, appearing in 23% of the studied neonates.**[22]** **Eghbalian et al. (2015**) reported that 33.3% neonates showed abnormal CT scan results, with hypoxemic-ischemic encephalopathy being the most common radiological finding, appearing in 47% of cases.[10] **Almuqbil et al. (2023)** reported that HIE was the most common etiological factor in neonatal seizures among late preterm and term neonates (42.6%), whereas intracranial hemorrhage was more common among extremely preterm neonates (19.7%).[23]

Large-scale studies are needed to validate the generalizability of these findings to diverse populations and healthcare settings. Future studies should investigate the long-term

neurodevelopmental outcomes of neonates with neonatal seizures, correlating the outcome to the early EEG patterns of these neonates, the controllability of their seizure phenomena by anti-convulsants, and the potential benefits (or risks) of prolonged versus shortened anti-convulsant medication courses in neonates with various types of seizures.

Conflicts of Interest

No conflicts of interest.

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