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Research Article

A Clinical-Electrographic Profile of Neonatal Seizures and Their Etiologies in a Single Level III Neonatal Intensive Care Unit

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Abstract

Background: Seizures are the most frequent neurological emergency in neonatal period and can be associated with significant mortality and subsequent neuro-developmental disability if not recognized and managed early. Repaid identification of neonatal seizures and evaluation is critically required to identify and treat the underlying etiology. Clinicians face a major challenge in diagnosing and treating neonatal seizure because of inconspicuous clinical presentation, variable electro-clinical correlation. Assessing the combination of clinical presentation, etiological factors, different diagnostic tools, and treatments, can broaden the knowledge and understanding towards neonatal seizure providing better management and outcome.

Objective: To study the characteristics of clinical and electrographic profile of neonatal seizure and the relations to their etiologies in neonatal intensive care unit.

Methods: This is a descriptive cross sectional retrospective study of electronic health records with ICD- 10 diagnosis of neonatal seizure from birth to 28 days, who were admitted to neonatal intensive care unit at Sheikh Shakhbout Medical City between January 2020 to December 2021. Clinical data obtained including basic demographics: gender, gestational age, mode of delivery, birth weight, and Apgar score. Onset and type seizure, type of antiepileptic drugs administered, EEG findings, brain imaging, results of relevant laboratory tests and etiology of seizure were collected.

Results: Total 31 neonates included in the study. Majority were male (71%), term (87%), and birth weight >2,5 kg (90%). Seizure within 24 hours was 58% and semiology was focal clonic in 32% of patient. No events in EEG in 81% and 6% had electrographic seizure (n=2). 94 % were treated with anti-convulsant, 86% of them before EEG. The majority (86%) received phenobarbitone as 1st line treatment and 83% received monotherapy. Leading causes of seizure were HIE (48%) followed by Vascular causes (16%). Seizure within 24 hours caused mainly by HIE (61%) and 60% of neonates with vascular and all infectious and genetic causes had seizure after 24 hours. In vascular causes, (60%) presented with focal clonic seizure. 60% of Multifocal epileptiform activity seen in HIE whereas focal presented more in vascular patient (36%). In HIE cases, 53% had normal brain MRI and 47% were abnormal. All cases with vascular causes had diagnostic abnormal MRI. The majority of HIE cases (87%) and all vascular cases received monotherapy.

Conclusion: In this descriptive study of neonatal seizure, certain demographic data, clinical and electrographic characteristics had established links to their etiologies. Clinical events captured during EEG with no electrographic seizure are classified as non-epileptic clinical phenomenon. Diagnostic utility of EEG is required in diagnosis of involuntary movements and avoid unnecessary anti-seizure medications. Understanding the clinical-electrographic profile of neonatal seizure helps in the early recognition of specific etiologies, allowing an early diagnosis by organized diagnostic approach and management.

Keywords: *Neonatal seizures, Electroencephalogram, Neonatal convulsion, HIE*

Introduction

Seizures are the most frequent neurological emergency in neonatal period with highest incidence of seizure in first month of life compared to any other period of life. (1,2,3). The incidence of neonatal seizures is reportedly 1.5–5.5 per 1000 live births among term infants but tends to be higher in preterm or very low birth weight infants. (4, 5) Its incidence in neonatal intensive care unit (NICU) can be as high as 10-25%, and they are a common cause for admission to the unit (6, 7). Because of their brain immaturity and high risk of injury, neonates are more susceptible to seizures which have unique pathophysiology and electrographic findings resulting in clinical manifestations that can be different and more problematic to identify when compared to older age groups. (4,5,8). They are defined as the occurrence of sudden, paroxysmal, abnormal alteration of electrographic activity with or without clinical signs at any point from birth to the end of the neonatal period excluding clinical events without an electrographic correlation. (1,4,8)

Most neonatal seizures occur as acute reactive events which may be the first or only manifestation of underlying serious neurological dysfunction in response to identifiable etiologic factors, these can include Neonatal encephalopathy and hypoxic-ischemic encephalopathy, acquired structural brain lesions, including ischemic and hemorrhagic stroke, metabolic disturbances and CNS or systemic infections which are the most common factors. A minor percentage occurs in the context of an epilepsy syndrome such as KCNQ2 or KCNT1- related encephalopathies. (9, 10, 11)

Neonatal seizure can be associated with significant mortality and subsequent neuro - developmental disability if not recognized and managed early. (7, 12)

Thus, repaid identification of neonatal seizures and evaluation is critically required to identify and treat the underlying etiology, prevent further brain injury, and extinguish the seizure activity, as any delay in therapy is often result in poor neurological outcome. (2, 8 ,13)

Clinicians face a major challenge in diagnosing and treating neonatal seizure because of inconspicuous clinical presentation, variable electro-clinical correlation. Many seizures present as electrographic-only events without clinical signs or any abnormal movement that can be due to seizure but are difficult to distinguish from other neonatal behaviors. (4, 12 ,14)

The increasing awareness and concerns of the dangers of neonatal seizures has driven a liberal approach to the use of anticonvulsants in the neonatal intensive care unit (NICU), As many neonatal units initiate the anticonvulsants on the basis of clinical diagnosis, but this policy carries risk and may cause additional injury to the neonatal brain. (6,15,16)

Video-Electroencephalogram (Video- EEG) remains the gold standard for their diagnosis and classification, to properly identify electrographic discharges and recognize nonepileptic events, also amplitude integrated EEG (aEEG) has been widely adopted in NICU are suited to continuous use and bedside interpretation, they are extremely important for the accurate assessment of response to therapy and seizure burden. Thus, clinician should be aware of the limitations of the clinical assessment in over and under-diagnosing seizures, and aEEG or cEEG confirmation of clinically diagnosed seizures should be sought whenever possible (11, 12, 15)

Because of the significant short and long outcomes of neonatal seizure and their underlying etiologies, Assessing the combination of clinical presentation, etiological factors, different diagnostic tools, and treatments, can broaden the physician's knowledge and understanding towards neonatal seizure providing better management and outcome. Therefor the objective is to study the characteristics of clinical and electrographic profile of neonatal seizure and the relations to their etiologies in neonates admitted in a tertiary care neonatal intensive unit.

Methodology

Study design: This is a descriptive cross sectional retrospective study of electronic health records with ICD- 10 diagnosis of neonatal seizure among neonates from birth to 28 days, who were admitted to tertiary neonatal intensive care unit at Sheikh Shakhbout Medical City between the periods of January 2020 to December 2021.

Inclusion criteria: The neonates who developed clinical seizure documented in nurses and medical records before 28 days and neonates who required EEG for clinical concerns (neonatal encephalopathy, unusual movements, HIE and intracranial infection) were included in the study.

Exclusion Criteria: Neonates who had no EEG report or were admitted from a different hospital beyond 3 weeks of age and were already on anti-convulsant therapy, excluded from the study.

Sample size: Number of neonates involved in study were 31.

Data collection: Clinical data were obtained from patient medical record including basic demographic information in terms of gender, gestational age, mode of delivery, birth weight, head circumference, Apgar score and labor records. Onset and type seizure, type of antiepileptic drugs administered, Electroencephalogram findings, brain imaging studies, results of relevant laboratory tests and etiology of seizure were all collected from medical records.

Statistical analysis: Data entry, tabulation and analysis was done by using Microsoft excel.

Results

Total 31 neonates who met the inclusion and exclusion criteria of neonatal seizure and admitted in NICU of SSMC, were included in this study. Table 1 shows demographics characteristics. Among them, 71% were male (n=22) and 29% were female (n=9). Majority were born term 87% and only 10% were born preterm (GA < 37 weeks). Out of 31 neonates, 55% (n=17) delivered by Emergency c section, 32 % by normal vaginal delivery. Average recorded Apgar score at 5 mins was 7.7 for all patients with 71% were ≥7 and 29% < 7. Average birth weight is 3 kg with 90% were above 2.5 kg.

Table 1. Demographic characteristics of neonates with seizure.

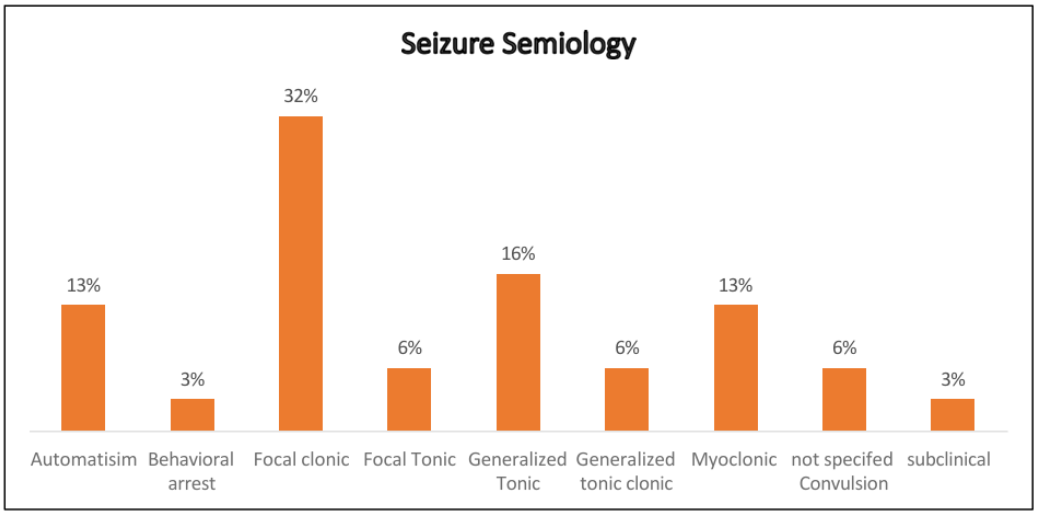
Characteristics		No. of neonates	Percentage
Gender	Male	22	71
	Female	9	29
Gestational age	Term	27	87
	Preterm	3	10
	Post term	1	3
Mode of delivery	Emergency c-section	17	55
	normal vaginal delivery	10	32
	Assisted vaginal delivery	3	10
	Elective c-section	1	3
Apgar score at 5 mins	< 7	9	29
Average 7.3	7 and above	22	71
Birth weight in kg	< 2.5 kg	3	10
	>2.5 kg	28	90

18 out 31 neonates (58%) had onset of seizure within first 24 hours of life and 13 (42%) after24 hours of life.

Table 2. The time of onset of seizures.

Seizure onset	No. of neonates	Percentage
Within 24 hours	18	58
After 24 hours	13	42

Regarding the types of clinical seizures, 32% had focal clonic, 16% generalized tonic, 13% myoclonic, and 13% automatism. ***Graph 1***



Graph 1. Seizure semiology.

Almost all EEG done were abnormal epileptiform 97% and 3% (n=1) was Abnormal Non- epileptiform. The epileptiform activity reported in EEG was multifocal in 20 neonates (65%) and focal in 11 neonates (35%). Table 3

81 % of neonate (n=25) didn't develop any event during EEG monitoring, 4 neonates (13%) had clinical seizure with no electrographic in EEG, 1 neonate had both clinical and electrographic activity in EEG, and 1 neonate had only electrographic seizure activity during EEG monitoring. Table 3

Table 3. EEG findings.

EEG Findings		**No. of neonates**	**Percentage**
Classification of EEG	Abnormal epileptiform	30	97
	Abnormal Non-epileptiform	1	3
Epileptiform activity	Focal	11	35
	Multifocal	20	65
Events during EEG			
	Clinical	4	13
	Electro-clinical	1	3
	Electrographic	1	3
	None	25	81

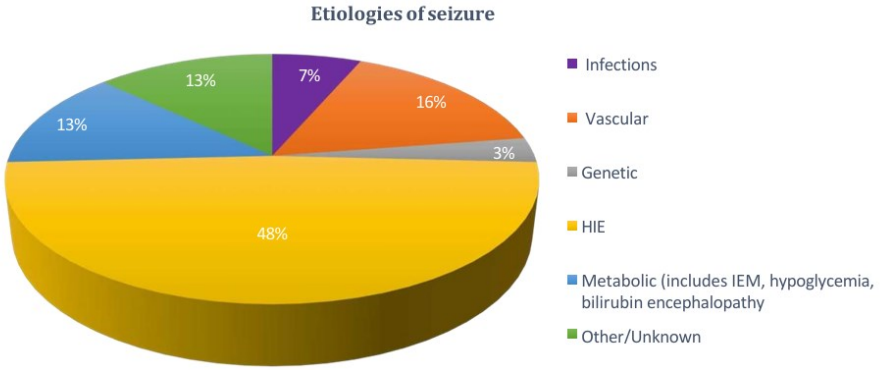
29 out 31 neonates (94 %) were treated with anti-epileptic medications, 86% of them (n=25) the AEDs were started prior to EEG monitoring and 13% (n=4) were started after EEG. 24 out of 29 neonates were treated with monotherapy AEDs (83%) and followed by 14% were treated with double therapy and only 1 neonate required more than 3 AEDs. Majority (25/29, 86%) received phenobarbitone as their first-line treatment, with the remaining 4 receiving midazolam (n=2), levetiracetam (n=1) and lorazepam as rescue (n=1). ***Table 4***

Table 4. Anti-epileptic medications (AEDs).

Anti-epileptic medications (AEDs)		No. of neonates Total = 29	Percentage
Number of AEDs	One	24	83
	Two	4	14
	Three and more	1	3
1st line medication			
	Phenobarbitone	25	86
	Midazolam	2	7
	Levetiracetam	1	3
	Lorazepam	1	3
2nd line medication			
	Phenobarbitone	4	14
	Phenytoin	2	7
	Oxcarbazepine	1	3
	Levetiracetam	1	3
	Clonazepam	1	3

Regarding the etiologies of neonatal seizure, 48% were due to hypoxic-ischemic encephalopathy (n=15): 9 neonates had mild HIE, 5 neonates with moderate HIE and one with severe HIE. 16% were due to Vascular (infarction/hemorrhage): 3 neonates had intracranial hemorrhage, and 2 neonates had stroke.

13 % due to metabolic causes including 2 cases of hypoglycemia, 1 case of bilirubin encephalopathy, 1 case of Inborn error of metabolism (non- ketotic hyperglycinemia). Rest was due to infectious causes (2 cases of meningitis), 1 case was due to genetic disorder (early infantile epileptic encephalopathy KCNQ2 gene) and remaining cases were either unknown aetiology (n=1), Benign sleep myoclonus of infancy (n=1) and Neonatal Epileptic Encephalopathy with unknown cause (n=1). ***Graph 2, Table 5***



Graph 2. Etiology of neonatal seizure

Table 5. Etiology of neonatal seizure.

Seizure Etiology		No. of neonates Total = 31	Percentage
HIE		**15**	**48**
	Mild HIE	9	29
	Moderate HIE	5	16
	Severe HIE	1	3
Vascular (infarction/ hemorrhage)		**5**	**16**
	Intra-cranial hemorrhage	3	10
	Neonatal stroke	2	6
Metabolic (includes IEM, hypoglycaemia, bilirubin encephalopathy		**4**	**13**
	Hypoglycemia	2	6
	Bilirubin Encephalopathy	1	3
	Inborn error of metabolism (non-ketotic hyperglycinemia)	1	3
Infections		**2**	**6**
	Meningitis	2	6
Others/unknown		**4**	**13**
	Unknown	1	3
	Neonatal Epileptic Encephalopathy with unknown cause	1	3
	Suspected seizures—unconfirmed	1	3
	Benign sleep myoclonus of infancy		

The relation between the onset of seizure and etiological disorders showed leading cause of seizure within 24 hours, were HIE (61%), others/unknown 22% and vascular 11%. All neonates with infectious and genetic causes had onset of seizure after 24 hours. 75% of neonates with metabolic causes had seizure after 24 hours compared to 25% within 24 hours and 60% of neonates with vascular causes had seizure after 24 hours compared to 40% within 24 hours. ***Table 6 and Table 7***

Table 6. Causes of seizure within 24 hours.

Causes of seizure within 24 hours	No. of neonates Total = 18	Percentage
HIE	11	61
Other/Unknown	4	22
Vascular (infarction/hemorrhage)	2	11
Metabolic (includes IEM, hypoglycemia, bilirubin encephalopathy	1	6

Table 7. The relation of seizures onset time to etiological disorders.

	Within 24 hrs.		After 24 hours		Total	
	Percentage	No.	Percentage	No.	%	No.
Infections	0	0	100%	2	100%	2
Vascular (infarction/ hemorrhage)	40%	2	60%	3	100%	5
Genetic	0	0	100%	1	100%	1
HIE	73%	11	27%	4	100%	15
Metabolic (includes IEM, hypoglycemia, bilirubin encephalopathy	25%	1	75%	3	100%	4
Other/Unknown	100%	4	0%	0	100%	4

Table 8. Causes of focal clonic seizure.

Causes of Focal clonic seizure		
	No.	Percentage
Infections	1	10%
Vascular (infarction/hemorrhage)	3	30%
HIE	5	50%
Other/Unknown	1	10%
Grand Total	10	100%

Table 9. The relation of seizure semiology to etiological disorder.

	Infections		Vascular		Genetic		HIE		Metabolic		Other/Unknown	
	%	**No.**	**%**	**No.**	**%**	**No.**	**%**	**No.**	**%**	**No.**	**%**	**No.**
Automatism	0.00%	0	20.00%	1	0.00%	0	6.67%	1	50.00%	2	0.00%	0
Behavioral arrest	0.00%	0	0.00%	0	0.00%	0	6.67%	1	0.00%	0	0.00%	0
Focal clonic	50.00%	1	60.00%	3	0.00%	0	33.3%	5	0.00%	0	25.00%	1
Focal Tonic	0.00%	0	0.00%	0	0.00%	0	0.00%	0	25.00%	1	25.00%	1
Generalized Tonic	0.00%	0	0.00%	0	100.0 0%	1	20.00%	3	0.00%	0	25.00%	1
Generalized tonic clonic	50.00%	1	20.00%	1	0.00%	0	0.00%	0	0.00%	0	0.00%	0
Myoclonic	0.00%	0	0.00%	0	0.00%	0	13.33%	2	25.00%	1	25.00%	1
Not specified	0.00%	0	0.00%	0	0.00%	0	13.33%	2	0.00%	0	0.00%	0
subclinical	0.00%	0	0.00%	0	0.00%	0	6.67%	1	0.00%	0	0.00%	0

The relation of EEG findings to etiological disorder showed the 2 neonates who developed either Electro- clinical or Electrographic during EEG, had metabolic causes. 4 neonates who had clinical seizure with no electrographic changes in EEG, 50% had HIE and 25% metabolic. ***Table10.***

Table 10. Relation of EEG findings to etiological disorders.

Relation of EEG findings to etiological disorders								
	Clinical Total=4		Electro-clinical Total=1		Electrographic Total=1		None To-tal=25	
	%	No.	%	No.	%	No.	%	No.
Infections	0%	0	0%	0	0%	0	8%	2
Vascular (infarction/hemorrhage)	0%	0	0%	0	0%	0	20%	5
Genetic	0%	0	0%	0	0%	0	4%	1
HIE	50%	2	0%	0	0%	0	52%	13
Metabolic (includes IEM, hypoglycemia, bilirubin encephalopathy	25%	1	100%	1	100%	1	4%	1
Other/Unknown	25%	1	0%	0	0%	0	12%	3
Grand Total	100%	4	100%	1	100%	1	100%	25

The main etiology of seizure in neonates with multifocal epileptiform activity in EEG, was HIE (60% n=12/20), followed by metabolic (15% n=3/20). In focal epileptiform activity, 36% (n=4/11) were due to vascular causes followed by HIE (27% n= 3/11). ***Table 11***

The relation of epileptiform activity to etiological disorder				
	Multifocal Total=20		Focal Total=11	
	No.	%	No,	**%**
Infections	1	5%	1	9%
Vascular (infarction/hemorrhage)	1	5%	4	36%
Genetic	1	5%	0	0%
HIE	12	60%	3	27%
Metabolic (includes IEM, hypoglycemia, bilirubin encephalopathy	3	15%	1	9%
Other/Unknown	2	10%	2	18%

50% of neonates had abnormal MRI brain findings (15/30). The relation of MRI findings with etiology of seizure showed, neonates who had HIE, 53% of them (n=8/15) had normal brain MRI and 47% (n =7/15) had abnormal MRI brain. All neonates with vascular causes were diagnosed by abnormal MRI and the 2 neonates with meningitis had abnormal MRI brain as well. In neonates with metabolic causes 75% (n=3/4) had normal MRI and the remaining case had abnormal MRI which was consistent with findings of non- ketotic hyperglycinemia. Unknown and other causes all had normal brain MRI findings in which some went further metabolic and genetic investigation and were normal. ***Table 12***

The relation of MRI findings with aetiology of seizure				
	Abnormal MRI Total=15		Normal MRI Total=15	
	No.	%	No.	%
Infections	2	100%	0	0%
Vascular (infarction/hemorrhage)	5	100%	0	0%
HIE	7	47%	8	53%
Metabolic (includes IEM, hypoglycemia, bilirubin encephalopathy	1	25%	3	75%
Other/Unknown	0	0%	4	100%

The relation of Number of AEDs with etiology of seizure showed, 2 Neonates with meningitis received either one or two AEDs (50% each), in vascular aetiologias all neonate received one AEDs ,87% and 75% of neonates with HIE and metabolic cases respectively, received one AEDs. Neonate who had genetic disorder received two AEDs given to nature of genetic disease. ***Table 13***

Etiology of seizure' and anti-seizure medications	**Numbers of Anti-seizure medications**			
Etiology of seizure	**4**	**2**	**1**	**0**
Infections	0%	50.00%	50.00%	0%
Vascular (infarction/hemorrhage)	0%	0%	100.00 %	0%
Genetic	0%	100.00 %	0%	0%
HIE	0%	6.67%	87%	6.67%
Metabolic (includes IEM, hypoglycemia, bilirubin encephalopathy	0%	25.00%	75.00%	0%
Other/Unknown	25.00 %	0%	50.00%	25.00 %

Discussion

The results of this study, showed the following demographic characteristics including, male, term and birth weight above 2.5 kg were dominant in neonates with seizure which is comparable with several studies done in USA, Iran, India and Egypt (5,7,10,14,17)

Most patients were delivered by Emergency C section 55%, followed by normal vaginal delivery 32%, with similar to study done by Nemati et al. (17) but in study done by Mohanram et al, 50% were delivered by normal vaginal delivery followed by 40% c-section. (5)

Regarding the onset of seizure, more than half of our patients developed seizures within 24 hours, which was less in a study done Agrawal et al, showed 38% within 24 hours and within 72 hours 85% and other similar studies seizures within 1st day were less compared to our study (5, 7, 10)

Most common type of clinical seizure in our patient was focal clonic seizure, similar to a study by santarone et al in patients with motor seizure type only (11). 2 studies showed subtle seizure was the most common (5,7). multifocal clonic seizure was most common type in other 2 studies. (10, 17).

All patients had abnormal EEG findings with epileptiform activity, and more than half (65%) were multifocal, and the rest were focal. Surprisingly majority didn't develop any events during EEG except for 2 patients who had electrographic and electro-clinical seizure, keeping in mind that majority had been started on AEDs before EEG. On the other hand, study done by Glass et al showed 82% of patients had electrographic detected by continuous EEG monitoring which could explain the high incidence of electrographic seizure in their study. (14)

HIE was the most common cause of seizure in our patient which is well known and documented worldwide. Second most common were Vascular (infarction/hemorrhage) causes which was consistent with 3 studies Santarone et al, Abdelhaie et al and Glass et al. (3, 11,13, 14) Whereas 2 other studies reported metabolic causes as 2nd most common cause in their patients. (7, 17)

Seizures presenting within 24 hours of life, were mainly caused by HIE and more than half of patients with metabolic causes and vascular causes presented after 24 hours of life. All patients with meningitis and genetic disorder presented after 24 hours. In Santarone et al study they found, HIE have an earlier onset in comparison both to stroke and infections (11). Agrawal et al stated early onset seizures are usually associated with HIE, intracranial hemorrhage and metabolic abnormality while late onset seizures are associated with sepsis, meningitis, developmental defects, and metabolic abnormality. (7)

Most neonatal seizures occur very early in life with nearly a third occurring within the first day, and another third occurring within the first week. (3)

60% of patients with vascular causes presented with focal clonic but still HIE was the most common cause of focal clonic in this study followed by vascular, compared to study by Santarone et al, stroke followed by infective causes were the leading causes of focal clonic. (11)

Multifocal epileptiform activity noticed in EEG, presented more commonly in HIE patients whereas focal presented more in patients with vascular. In Santarone et al study showed unilateral spikes in EEG was seen in stroke patients, but bilateral spikes were mainly seen in encephalitis and HIE. (11)

MRI brain is crucial in diagnosing patients with vascular causes and its important diagnostic step in evaluating neonatal seizures. techniques such as diffusion-weighted magnetic resonance imaging (MRI) have enhanced the diagnosis of focal ischemic lesions. (3)

In this study majority of patients received one AEDs which was before starting EEG, phenobarbitone was the first line of treatment in almost all patients compared to other study similar result except 42 % received AED before EEG. (16)

The current study didn't not include the short- and long-term outcomes of neonatal seizure. Thus, a long-term study is needed to evaluate the neurodevelopmental outcome in these patients.

Conclusion

In this descriptive study of neonatal seizure, certain demographic data, clinical and electrographic characteristics had established links to their etiologies.

Most patients were male and full term, presented with early onset seizure with focal clonic semiology and with no electrographic activity who were started on phenobarbitone prior to EEG monitoring.

Most common causes were HIE and vascular in which HIE presenting as early onset, whereas vascular and infectious causes presented in late onset seizure.

Clinical events captured during EEG with no electrographic seizure are classified as non- epileptic clinical phenomenon. Diagnostic utility of EEG is required in diagnosis of involuntary movements and avoid unnecessary anti-seizure medications.

Different seizure semiology depends on the abnormal discharge extent and its location in the brain and are linked to specific etiologies.

Understanding the clinical-electrographic profile of neonatal seizure help physicians in the early recognition of specific etiologies, allowing an early diagnosis by organized diagnostic approach and management.

Conflict of Interest

The authors declare that they have no competing interests.

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