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ETIOLOGICAL, CLINICAL AND EEG PROFILE OF NEONATAL SEIZURES- AN INSTITUTIONAL STUDY

Dr. Roshith J. K.*[1] and Dr. K. N. V. Prasad[2]

[1]Post Graduate, Department of Pediatrics, Sri Devaraj Urs Medical College, Tamaka, Kolar, Karnataka.
[2]Professor and HOD of Department of Pediatrics, Sri Devaraj Urs Medical College, Tamaka, Kolar, Karnataka.

***Corresponding Author: Dr. Roshith J. K.**

Post Graduate, Department of Pediatrics, Sri Devaraj Urs Medical College, Tamaka, Kolar, Karnataka.

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ABSTRACT

Background: The occurrence of seizures may be the first indication of a neurological disorder in the neonate. A correct mode of resuscitation soon after birth at first minute of non-vigorous baby, proper monitoring and management of seizure during the post- resuscitation care babies can prevent further occurrence of seizures, deterioration of low agar babies in neonatal care unit and this also helps in better outcome which avoids morbidity, mortality and associated sequel of neonates with seizures. A proper documentation of seizure and clinical profile of seizure can also help with earlier determination of etiology of seizure and help in proper management of the same. Hence this study has been taken up to determine clinical seizure profile, etiology of seizures, bio-chemical and EEG abnormalities in neonatal seizures which would help in early recognition, treatment and better prognosis in neonatal seizures. **Objectives:** 1. To characterize the clinical profile of neonatal seizure. 2. To identify the etiological profile of neonatal seizure. 3. To perform EEG in neonatal seizure and correlate the clinico-etiological profile. **Material and methods:** A total of 68 inborn and outborn neonates with seizure were enrolled in the study from Januray 2015 to January 2016. Data including gestational age, birth weight, antenatal events, perinatal events, apgar score, mode of resuscitation, clinical profile like age of onset of seizures, duration of seizure, episodes of seizure, drug used to abort seizure, type of convulsion, seizure associated autonomic changes, lab investigation reports like complete blood count, C-reactive protein, serum biochemistry, cerebro-spinal fluid study –etiology profile of seizure was evaluated, electro encephalogram was performed as soon as early as possible and all were documented in proforma. **Results:** Of 68 neonates with seizures were included in the study. In the study 91.2% of the neonates were term babies and 8.8%. Male predominance was observed. Out of the neonates enrolled 73.5% of neonates with seizure had low apgar & 77.9% of neonates with seizure who required intermittent positive pressure ventilation were admitted for post -resuscitation care in NICU. In the study 61.7% of neonates had seizures at less than 12 hours of life. Focal clonic type of seizures was more observed (67.2%) as compared to other types. Hypoxic Ischemic Encephalopathy (HIE-II) was a major etiology in the study 42.6%. Among 18 EEG done on neonates with seizures, two had an abnormal EEG pattern. **Limitations**: In my study EEG could not be performed in all neonates who had seizures due to various reasons. **Conclusions:** In the present study of etiology in neonatal seizures showed that Hypoxic ischemic encephalopathy -II was the common etiology in 29 neonates (42.6%) and the second major etiology noted in 13 babies (19.1%) was hyponatremia. Focal clonic type of seizure was seen in 67.2% neonates as a common clinical profile of seizure.

KEYWORDS: Electroencephalography (EEG), Hypoxic ischemic encephalopathy, Perinatal Asphyxia.

INTRODUCTION

Neonatal seizure is defined as a paroxysmal alteration in neurological function i.e. motor, behavior and/or autonomic function.[1] It is one of the first sign of neurological dysfunction and most frequent neurological events in new born infants, reflect a variety of pre-, peri- or postnatal disorders of the central nervous system. These may range from benign, self- limited illness to severe, prolonged or life threatening disorders. They are also a common manifestation of metabolic abnormality in new born.

In 1989 Volpe classified Neonatal seizures as 1. Subtle, 2. Tonic (Generalized, Focal) and 3. Clonic (multifocal, focal), 4.Myoclonic (Focal, Multifocal, Generalized). The international classification of epileptic seizures does not apply to newborn seizures because neonates are unstable to sustain organized discharges and do not manifest generalized tonic clonic seizures. Any abnormal, repetitive and stereotypic behavior in neonates should be evaluated as possible seizure.[1]

Clonic seizures represent the seizure type associated most consistently with time-synchronized EEG seizure

activity. Clonic movements in the newborn are rhythmic and usually rather slow (approximately one to three jerks per second at the onset with the rate progressively declining with the seizure). Focal clonic seizures involve face, upper and or lower extremities on one side of the body, or axial structures (neck or trunk) on one side of the body. However, it is important to recognize that focal clonic seizures may occur with metabolic encephalopathies in the newborn. Multifocal clonic seizures involve several body parts, often in a migrating fashion, although the migration most often "marches" in a non-Jacksonian manner (eg- left arm jerking may be followed by night).[1]

Tonic seizures are clinical episodes, the most common of which are unassociated with time-synchronized EEG discharges. Two categories of tonic seizures should be distinguished i.e., focal and generalized. The latter are much more common than the former (this seizure multi-focal [i.e., migrating, asynchronous] tonic seizures in the newborn were not observed). Focal tonic seizures consist of sustained posturing of a limb or asymmetric posturing of trunk and/or neck, classify horizontal eye deviation as a focal tonic seizure.[1]

Myoclonic seizures (like tonic seizures) are clinical episodes that as a group are most commonly unassociated with time-synchronized EEG discharges. Myoclonic movements are distinguished from clonic movements particularly because of the more rapid speed of the myoclonic jerk and the particular predilection for flexor muscle groups. Three categories of myoclonic seizures should be distinguished, i.e., focal, multifocal, and generalized. Focal myoclonic seizures typically involve flexor muscles of an upper extremity and of 41 focal myoclonic seizures studied by Mizrahi and Kellaway, only three were associated with EEG seizure discharges.[1]

Furthermore, seizures can have immediate and long – term adverse consequences on the immature and developing brain. Irrespective of immediate attempts to suppress the neonatal seizures with anti- epileptic drugs, the risk of subsequent neuro -developmental deficits and early death is substantial. Newborn infants with seizures are at risk for neurological impairment, developmental delay, later epilepsy and cognitive impairment as in WHO guidelines of neonatal seizures.[2]

In 1998 Mizrahi and Kellaway P classified etiology of neonatal seizures into chromosomal anomalies, congenital abnormalities of brain, neuro- degenerative disorders, inborn errors of metabolism, benign neonatal convulsion, benign familial neonatal convulsions and drugs withdrawal or intoxication. This classification elaborates more about the causes of Neonatal seizures including genetic study and pharamaco-kinetic evaluation of anti convulsant drug which gives more focus to genetic causes of Neonatal seizures and compliance of anti-convulsant drug.[3]

Subtle seizures are the most frequent seizure type, which may consist of only horizontal deviation and or jerking of the eyes, repetitive blinking or fluttering of eyelids, drooling, suckling or other oro- buccal movements. Subtle seizures are most common in premature infants. Neonates with seizures are at risk of death, whereas survivors are at risk of neurological sequelae, developmental delay, later epilepsy and cognitive impairment so, we need to initiate an early diagnostic work up to determine the causes to be ruled out.[4] Bio-chemical disturbances occur frequently in neonatal seizures either as underlying causes or as an associated abnormality. In their presence it is difficult to control seizure and there is a risk of further brain damage. Early recognition and treatment of biochemical disturbances are essential for the optimal management and satisfactory outcome.[5]

The overall prognosis for survival in neonatal seizures is 85% with the significant improvement. The range of outcomes after neonatal seizures varies widely with three major predictors:-
1) Underlying Etiology, 2) EEG pattern, 3) Gestational age.[6]

Electroencephalograph (EEG) provides a useful non-invasive diagnostic tool of neonatal seizures and evaluates degree of perinatal brain damage. Its also an interpretation which is influenced by variations is normal maturation process of brain and also not all seizures can be picked by surface recorded EEG and many clinically silent electrographic seizures. The EEG finding, such as background abnormality, area of involvement of the discharges and the type of discharges were seen to categorize the neonatal seizures.[7]

In neonatal seizures, EEG amplitudes are usually low (bilateral cortical damage, wide spread cerebral damage, structural brain damage, mild metabolic disorders etc.) or very low (extensive cortical damage due to any cause). Slow waves in EEG in neonatal seizures signify any type of anoxic, metabolic, toxic encephalopathy etc. It has the potential to assess severity of brain dysfunction and aid in discovering subclinical seizures while at the same time giving us an insight into the various degrees of cerebral maturation.[7]

The occurrence of neonatal seizures may be the first and perhaps the only, clinical sign of a central nervous system disorder in the newborn infant. As such, seizures may indicate the presence of a potentially treatable etiology and should prompt an immediate evaluation to determine cause and to institute etiology-specific therapy. The clinical diagnosis, characterization and classification of neonatal seizures and the use of electroencephalography in clinical management are critical for the neonate`s care.

In addition to classifying seizures based only upon their clinical characteristics, neonatal seizures may also be

classified according to the temporal relationship of clinical events to the occurrence of electrical seizure activity recorded on EEG. Seizures may also be classified according to their pathophysiology - may be epileptic or non-epileptic in origin. These are new concepts about neonatal seizures and its classification.[3]

Hence there is a need for a study which will evaluate the clinical and etiological profile of neonatal seizures and its correlation with EEG. There is a relative dearth of such studies in Indian literature and it is in order to broaden our understanding of this subject that we are undertaking this study.

MATERIAL AND METHODS

Study will be conducted in newborn with clinically identified seizures and admitted in Neonatal Intensive Care unit of RL Jalappa Hospital & Research Centre for period of 1 year starting from January 2015 to January 2016. Inclusion Criteria were all neonates having seizure and admitted to the NICU of R L Jalappa Hospital. Exclusion criteria were neonates with congenital anomalies of the head and critically ill neonates on ventilator. All inborn and outborn neonates who had seizures were admitted to the NICU of the R L Jalappa Hospital with clinically identified seizures will be enrolled in the study. For each recruited baby, the resident NICU doctor will document the accurate description of the clinical seizure as in Clinical profile of neonatal seizures.[2]

Relevant laboratory investigation such asblood counts, C-reactive protein serum biochemistry, cranial ultrasonography, EEG recordings & computerized tonography, magnetic resonance imaging & karyotyping will be performed as per need of the study would be entered into a proforma. All the methods carried out were in accordance with the ethical standards of the institute and approval.

RESULTS

In present study, out of 3606 babies admitted to neonatology unit during the study period 68 babies developed seizures giving a hospital occurrence of seizure was 18.8. Neonatal seizures were more common in term neonates (91.2%) as compared to preterm neonates (8.8%). The highest occurrence of seizures was higher in male (76.4%) as compared to female neonates (23.6%). Neonatal seizures were common in 46 neonates within less than 12 hours of life (61.7%), 18 neonates had seizures during more than 12 to 24 hours of life (18%) and 8 neonates had seizures more than 24 hours to 72 hours of life(11.9%). In my study 50 babies had Low APGAR at 1minute, 30 babies had Low APGAR at 5 minutes and 3 babies had Low APGAR at 10 minutes.77.9% of neonates required intermittent positive pressure ventilation, 25% of neonates required intubation mode of resuscitation.

The major etiology of neonatal seizure was Hypoxic ischemic encepahlopathy- II of 42.6% of neonates and 33.8% of neonates with seizures had hyponatremia. 91.1% of neonates were responded to primary anti-convulsant drugs within less than 5 minutes to abort neonatal seizure. In my study 67.2% of neonates had focal clonic seizure as commonest clinical profile, 14.3% of neonates had focal tonic type of seizures and 8.4% of neonates had generailzed tonic type of seizure. 2.5% of neonates had subtle seizures. 59 neonates with seizures responded to primary anti-convulsant drug (loading dose), 9 neonates with seizures responded to two anti-convulsant drugs(loading and maintenance dose) and 3 neonates with seizures required three anti-convulsant drug intermittently.

In my study 2 neonates had an abnormal EEG profile were observed with perinatal asphyxia where as other 16 neonates with seizures had normal EEG with various etiology of seizure. Hence EEG cannot be a specific tool to evaluate the correlation of EEG with clinical and etiological profile of neonatal seizures.

Table No 1- Distribution of neonates with seizures according to type of seizures

Type of seizure	Number	Percentage
Focal Clonic	**48**	**70.5%**
Focal Tonic	**12**	**17.6%**
Generalised Tonic	**8**	**11.7%**
Myoclonic	**6**	**8.8%**
Spasms	**4**	**5.8%**
Motor Automatism	**3**	**4.4%**

Table No 2- Distribution of neonates with seizures according to etiology of seizures

Etiology of Seizure	Number	Percentage
Hypoglycemia	8	11.7 %
Hyponatremia	13	19.1 %
Hypernatremia	11	16.1 %
Hypocalcemia	1	1.4 %
HIE II	29	42.6%

HIE III	3	4.4 %
Idiopathic	3	4.4 %

Table No 3 - Distribution of neonates with seizures according to age of onset of seizure

Age of Onset of seizures	Etiology of seizures	Percentage
<12 hours of life (n-34)	Hypoglycemia- 8 Hyponatremia- 13 HIE-II -13	23.6% 38.2% 38.2%
>12 hours of life– 24 hours of life (n-20)	Hypocalcemia- Sepsis -1 HIE-II – 10 HYpernatremia- 8	5% 5% 50% 40%
>25 hours of life- 72 hours of life (n-14)	HIE-III-3 Idiopathic – 3 HIE-II-6 Hypernatremia- 2	21.5% 21.5% 42.8% 14.2%

Table No-4 Distribution of neonates with seizures response to primary anti convulsant drug and its correlation to clinic –etiological profile

Anti-convulsant drug	Number of drug	Number	Percentage	Correlation with Etiological & Clinical Profile	
Phenobarbitone (Loading dose and maintenance dose)	1	59	86.7%	Clinical Profile- Duration of seizure	<5 minutes- 50(84.7%) 5 minutes- 30 minutes-9 (15.3%)
				Type of seizures	Focal clonic – 36 (61%) Focal Tonic – 12 (20.3%) Generalised tonic-8 (13.5%) Motor Automatism-1(1.6%) Spasms- 1(1.6%) Cyclonic- 1(1.6%)
				Etiological Profile of Seizure	HIE II – 26(44%) Hyponatremia – 13(22.1%) Hypernatremia -11(18.6%) Hypoglycemia-8(13.6%) Hypocalcemia-1(1.7%)
Anti-convulsant drug	Number of drug	Number	Percentage	Correlation with Etiological &Clinical profile	
Phenytoin Loading dose with Phenobarbitone (maintenance dose)	2	9	13.2%	Clinical Profile- Duration of seizure	<5 minutes-6 (66.7%) 5 minutes – 30 minutes-2 (22.2%) >30 minutes-1(11.1%)
				Type of seizures	Focal clonic-7 (77.7%) Motor Automatism-2 (22.3%) Myoclonic – 5(55.5%) Spasm- 3 (33.3%)
				Etiological Profile	HIE-II – 3 (33.3%) HIE-III – 3 (33.3%) Idiopathic – 3 (33.3%)
				EEG Profile -	1Abnormal EEG –continuous slow waves present
Midazolam intermittent doses with Phenytoin and Phenobarbitone (both maintenance dose)	3	8	11.7%	Clinical Profile- Duration of seizures	< 5 minutes-6(75%) 5 minutes- 30 minutes- 1 (12.5%) >30 minutes- 1(12.5%)
				Type of Seizure	Focal clonic-5 (62.5%)

					Focal Tonic- 3 (37.5%) Spasm-1(12.5%)
				Etiological Profile	Hypernatremia- 5 (62.5%) HIE-III-3 (37.5%)

DISCUSSION

CLINICAL PROFILE

A study done by Shah the incidence of neonatal seizures was 10.3 /1000 live birth.[5] Another study done by Ajay and Sahana showed an incidence of 11.7 /1000 live births[11] and 8.38 /1000 neonates respectively.[12] In my study comparing with the above studies, the occurrence of seizure was 18.9 per 1000 live birth. This could be probably due to more number of cases of birth asphyxia. In a study done by Sahana, the incidence of male neonates with seizure were 52.29% and female were 47.71%.[12] In another study done by Suryavanshi the incidence of male in neonatal seizure were 62.5% and female were 37.5%.[13]

In my study the occurrence of seizure in males neonates were 76.4% and females neonates were 23.6%. As male are better cared for then female due to various cultural beliefs.

The incidence of neonatal seizures as reported by various studies range from 0.1-0.5% in term neonates and 10-22.7% in preterm neonates.[53] In an Indian study done by Ajay, the incidence of neonatal seizures in preterm babies were 6.14% in contrast to 0.69% in term babies.[13] In another study done by Suryavanshi overall incidence of neonatal seizures were 23.75% in preterm and 76.25% in term babies.[15] In my study occurrence of neonatal seizure in term neonates were 91.1% and preterm neonates were 8.8%.

In a study done by Shah on Clinical biochemical profile of neonatal seizures showed that the incidence of neonatal seizure were <2500gms were 66% and >2500gms were 30%.[5]

In another study done by Ajay on Clinico-etiological and EEG profile of neonatal seizures showed that the incidence of neonatal seizure in low birth weight were 14% and 0.59% in normal birth weight.[11] Another study done by Sahana on Clinical profile of neonatal seizures showed that the incidence of neonatal seizure were <2500gms were 45% and >2500gms were 64%.[12]

In Suryavanshi study of clinical profile in neonatal seizures in rural area showed that the incidence of neonatal seizure in <2500gms were 50% and >2500gms were 50%.[13]

In my study the occurrence of neonatal seizure were 79.4% in normal birth weight and 20.6% in low birth weight.

In a study of neonatal seizure done by Saliba on neonatal seizures 91 neonates were Appropriate for gestational age, 24 neonates were Small for gestational age and 7 neonates were Large for gestational age.[53] In my study neonates with seizure the occurrence of seizures in 79.4% neonates were appropriate of gestational age and 26.5% neonates were small for gestational age.

In a study done by Aziz A on clinical and etiological profile of a neonatal seizure which was conducted in a tertiary care hospital of Government Medical College Srinagar. A total of 100 consecutive neonates presented with seizures shows that detailed antenatal history and baseline characteristics of convulsing neonate were recorded at admission and could correlate with the etiology profile, clinical profile of neonatal seizure. 5 neonates with seizure had Pre-eclampsia (5%) and 11 neonates with seizures had Foetal distress (11%).[54]

In my study 6 neonates with seizures had Pre-eclampsia (8.8%), 5 neonates with seizures had Eclampsia (7.3%), 8 neonates with seizures had hypertension (11.7%) and 19 neonates with seizures were drug induced deliveries (27.9%). 18 neonates with seizure had consanguinity and 50 neonates with seizures had non-consanguinity.

In a study done by Aziz A on neonatal seizure 48% of neonates were born by vaginal deliveries, 28% of neonates were born by caesarean, 24% of neonates were born by operated vaginal had neonatal seizures and PROM of 7 neonates (7%).[54] In this study it was observed that the occurrence of seizures in various antenatal events 35.7% of caesarean neonates. 47% of neonates were born by vaginal delivery, 17.6% of neonates were born by assisted vaginal delivery. 23.3% of neonates had <18 hours PROM and 13.9% of neonates had >19 hours PROM.

In a study done by Aziz A on neonatal seizure 44 neonates had Low APGAR at 5 minutes (44%),56 neonates had Low APGAR at 5 minutes (56%.).[54] In this study 73.5% of neonates had Low APGAR at 1minute, 44.1% of neonates had Low APGAR at 5 minutes and 8% of neonates had Low APGAR at 10 minutes.

In a study done by Basil showed that 17 neonates with seizures required intubation mode of resuscitation.[55] In my study 19.1% of neonates required oxygen supportive care, 77.9% of neonates required intermittent pressure positive ventilation, 25% of neonates required intubation, 7.3% of neonates born were induced deliveries.

In a study done by Hafizur shows that 77% of neonates had reactive CTG of which only 11 neonates were associated with foetal distress.[56] In my study of neonatal seizure 47% of neonates had Reactive CTG (Cardiotocography) and 52.9 neonates had Non reactive CTG. 47% of neonates had foetal distress.

In a study done by Basil showed that 286 neonates had meconium stained aminotic fluid (4.8%) and 13 neonates had meconium stained aspiration syndrome (0.21%).[55] In my study 20.3% of neonates with seizures were MSAF (Meconium stained amniotic fluid),3 neonates with seizure had MAS (Meconium Aspiration Syndrome).

A study done by Maya on Neonatal seizure –A profile of the etiology and time of occurrence shows that 59 neonates had seizures less than 24 hours of life (43.7%), 46 neonates had seizures 24 -72 ours of life (34.07%) and 17 neonates had seizures more than 72 hours of life.[9] A study done by Ajay at New Delhi, fifty two neonates developed seizures within 48 hours of life, out of which 20 neonates had seizure in less than 12 hours of life. Autonomic changes (heart rate, respiratory rate of oxygen saturation of blood) were present in 108 neonates with seizure episodes (46.55%) 11.

A study done by Sahana on Clinical profile of neonatal seizures at Level II Government general hospital Bangalore showed the higher percentage of occurrence of seizures less than 24 hours of life was 56 neonates (51.37%), 19 neonates had seizures on second day of life (17.43%), 10 neonates had seizures on third day of life (9.18%) and 24 neonates had seizures more than 4 days of life (22.02%).[12]

In a study done by Suryavanshi on Study of clinical profile in Neonatal seizures in rural area showed that 80 neonates had seizures at first 1-2 days of life to the first week of life.[13]

In a study done by Michael – neonatal seizures in a rural Kenyan district hospital showed that 46 neonates of 142 neonates (32.3%) had seizures within the first 48 hours of life, 30 neonates had seizures on day 3-7 days of life and 66 neonates had seizures after the first week of life (46.5%). In this study it was also observed that 50 of neonates developed seizures within the first week of life and the majority of 79 neonates had seizures (56%) more than one episode of seizures within 24 hours.[57]

In my study 42 neonates had seizures less than <12 hours of life(61.7%),18 neonates had >12-24 hours of life(26.4),8 neonates had seizures >25 hours -72 hours of life (11.9%). 54 neonates had 1 episode of seizure (79.4%),11 babies had 2 episodes of seizure (16.1%) and 3 babies had 3 episode of seizures (4.5%).

Etiological Profile

In a study done by Shah on showed the majority of 40% of neonates with seizure had multi focal clonic seizures, 9% of neonates had tonic seizures, 4% of neonates who had focal clonic, no myoclonic seizures were observed.[5] In Ruma Pravin study on neonatal seizures showed that the majority of seizures were 35.3% of neonates had subtle seizures, 15.7% of neonates had clonic seizures, 3.9% of neonates had mixed type of seizures and 45.35% of neonates had Tonic seizures.[7] In Ajay study on neonatal seizures also showed that 42.24% of neonates with seizures had multifocal clonic seizure, 21.55% of neonates had generalized tonic seizure, 8.19% of neonates had subtle seizure, 6.47% of neonates had focal clonic and 0.86% of neonates had myoclonic.[11] In Shahana study on neonatal seizure majority of 42.2% of neonates had subtle seizures, 33.3% of neonates had focal clonic seizures, 11.1% of neonates had multi focal clonic seizures, 11.1% of neonates had tonic seizure and 2.2% of neonates had myoclonic seizures.[12]

In my study on neonatal seizures 48 neonates had focal clonic seizures (67.2%), 12 neonates had focal tonic seizures (14.3%), 8 neonates had generalized tonic seizures were (8.4%), 6 neonates had myoclonic seizures (5.5%), 4 neonates had spasms (2.5%) and 3 neonates had motor automatism (1.4%).

In Shah study on neonatal seizures showed that the major etiology of seizures were birth asphyxia of 40 neonates (44%), 10 neonates had septicemia (11%), 10 neonates had meningitis (11%), 10 neonates had hypocalcemia (11%), 20 neonates had hypoglycemia (22%).[5]

In a study done by Ruma on neonatal seizures majority of cause of seizure were perinatal asphyxia of 56.86% neonates, followed by 15.67% of neonates had septicemia, 11.76% of neonates had meningitis, 3.92% of neonates had kernicterus, 3.92% of neonates had neurometabolic disorder, 1.96% neonates had TORCH infections, 19.5% of neonates had Hypoglycemia, 15.7% of neonates had Hypocalcemia. There were no hyperglycemia and hypernatremia noted.[8]

In a study done by Maya on neonatal seizures showed that 51 neonates had Hypoxic Ischemic Encephalopathy (37.8%), 26 neonates had hypoglycemia (19.3%), 8 neonates had meningitis (5.9%), 13 neonates had intracranial bleed (9.6%), 3 neonates had hypocalcemia (2.2%), 5 neonates had neonatal stroke (3.7%), 2 neonates had inborn error of metabolism (1.5%), 27 neonates had others causes like seizure disorder, congential malformation and benign neonatal convulsion.[9]

In a study done by Ajay on clinico-etiological and EEG profile of neonatal seizures showed that the major etiology of seizure was perinatal asphyxia of 40% neonates, second major etiology was metabolic causes of 21% neonates which were 10% neonates had

hypoglycemia, 9% neonates had hypocalcemia, 1% of neonate had hypernatremia and 1% of neonate with hypoglycemia with hypocalcemia, 7% of neonates had meningitis. Four neonates with seizure had etiology of intracranial bleed, 2 neonates with seizure had etiology of bilirubin enecephalopathy,1 neonate with seizure had etiology of unexplained encephalopathy, 1 neonate with seizure had etiology of polycythemia, 1 neonate with seizure had etiology of brain malformation. 13 neonates with seizure had undetermined etiology.[11]

In a study done by Sahana on clinical profile of neonatal seizures in which 109 neonates with seizure were enrolled among them 63 neonates with seizure had perinatal asphyxia as the major common etiology (57.80%), second major common etiology were infections for 16 neonates with seizures (14.67%), 10 neonates with seizures had hypoglycemia as etiology,7 neonates with seizures had hypocalcemia as etiology, 6 neonates with seizures had intracranial hemorrhages as etiology (5.5%), 2 neonates with seizure had kernicterus as etiology, 5 neonates with seizure had idiopathic causes of seizures.[12]

In a study done by Suryavanshi on clinical profile of neonatal seizures showed that the major common cause of etiology of seizures were hypoxic ischemic encephalopathy of 37 neonates (46.25%).Acute metabolic disturbances like hypoglycemia, hypocalcemia, infectionsand intracranial hemorrhage were important cause of seizures found in 13 neonates with seizure (16.25%), 4 neonates with seizure (5%), 9 neonates with seizure (11.25%) and 15 neonates with seizure (18.75%) respectively.[13]

In my study Hypoxic Ischemic Encephalopathy II was the major etiology for seizures of 29 neonates with seizure, second common etiology was acute metabolic disturbances like hyponatremia, hypernatremia and hypoglycemia for 13 neonates with seizure (19.1%),11 neonates with seizure(16.1%) and 8 neonates with seizure(11.7%) respectively. 3 neonates with seizure had HIE-III, 3 neonates with seizure had idiopathic cause of seizures and hypocalcemia for 1 neonate (1%). No Chromosomal abnormalities, no congenital abnormalities, no Inborn errors of metabolism neonates were observed.

EEG Profile

In a study done by Ruma on neonatal seizures; correlation between clinic-etiological profile and EEG findings - 43.1% of neonates had abnormal EEG findings. In background of EEG abnormality 12 neonates had low voltage (23.5%), 2 neonates had very low voltage EEG (11.8%). In area of involvement of the discharges it was focal i.e. 31.4. In the type of discharges in EEG 15.7% of neonates with seizure had sharp spikes and 11.8% of neonates with seizures had spikes. There was a significant relation between EEG findings and perinatal asphyxia of neonates. There was significant relation of meningitis with background abnormality of EEG -12 neonates had low voltage EEG recorded, 2 neonates had very low voltage EEG and 2 neonates had slow voltage EEG.[8]

In a study done by Maya on neonatal seizure – a profile of the etiology and time of occurrence shows that 65 neonates had normal EEG and 34% of neonates had abnormal EEG. In neonatal seizures due to HIE, 33.34% of neonates had abnormal EEG. Among them 81% of neonates had predominantly sharp waves EEG and 8.75% of neonates had spike waves. No case of HIE had electro cerebral silence or burst suppression in EEG.[9]

In a study done by Ajay on clinic – etiological and EEG profile of neonatal seizure EEG was performed in 30% of neonates with HIE, among them 8% of neonates had abnormal EEG .Out of these background abnormalities 1 neonate with seizure had low voltage EEG record, 1neonate with seizure had very low voltage EEG record were HIE II and HIE III .Multifocal discharges 2 having sharp waves and spikes were seen in each. In 2 neonates with seizure who had hypoglycemia seizures – had multifocal discharges type of EEG were seen.[11]

In my study EEG was performed in 18 neonates among them 16 neonates had abnormal EEG and 2 neonates had abnormal EEG due to the limitations of my study.

CONCLUSION

Over all incidences of neonatal seizures were 18.9 /1000 live births in our hospital deliveries.

As etiological profile of seizure, hypoxic ischemic encephalopathy was the major common cause, second major etiology was hyponatremia as primary or secondary cause in my study. As clinical profile 67.2% of neonates had focal clonic type of seizures, 61.7% of neonates had seizures less than 12 hours of life, 79.4% of neonates had 1 episode of seizure, 91.2% of neonates with seizures responded within less than 5 minutes to anticonvulsant drug were noted in my study.

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