

Original Research Article

**Clinico-etiological profile of neonatal seizures in a tertiary care hospital of Northern India: a prospective cohort study**

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**Abstract**

**Background:** Neonates are at higher risk for the development of seizures due to various reasons, however diagnosis of neonatal seizures is difficult to establish because of varied aetiologies involved. Seizures are one of the most common risk factor for neonatal mortality and subsequent long term neurological sequelae. The incidence of neonatal seizure (NS) varies from 1.8-3.5/1000 live birth; whereas in NICU it could be as high as 5/1000 live births. **Objectives:** The present study was undertaken to evaluate the incidence, etiological factor, biochemical abnormalities, days of onset and clinical types in cases having neonatal seizures. **Methods:** This was a hospital based descriptive type of observational study done in a Neonatal Intensive Care Unit (NICU) of Department of Pediatrics, Nalanda Medical College & Hospital, Patna, Bihar, India. The study was conducted over a period of 1 year from 1<sup>st</sup> April 2019 to 31<sup>st</sup> March 2020. 214 neonates fulfilling the inclusion criteria of being < 28 days of life and either presenting with history of seizures or developing seizure during their course of treatment in NICU were included in the study, after consent from parents. They were evaluated with necessary investigations, ultrasound head and CT scan for incidence, etiological factor, clinical types and biochemical abnormalities found in neonatal seizure. **Results:** Out of 1488 NICU admissions during study period, seizures occurred in 214 neonates. Incidence of NS was 14.38% of total NICU admission. Neonatal seizure occurred more commonly among male (65.4%) than female admissions. 88.3% of NS occurred in babies delivered through spontaneous vaginal delivery. 97.7% neonates with seizure were hospital delivered. Most (68.7%) of these cases had birth weight > 2.5 kg. 90.2% of these case were term babies and only 7.9% were pre-term deliveries. 80.8% were appropriate for gestation age (AGA). 54.7% babies developed seizure within 24 hours of life and total 72% developed seizure within 72 hours of life. Subtle seizure was most common (45.3%) seizure type followed by focal clonic (25.7%), multifocal clonic (11.7%), tonic (7.5%) and myoclonic (2.8%). Birth asphyxia was most common cause seen in 58.4% cases followed by septicemia/meningitis in 20.6% cases. 42% had biochemical abnormalities but only 9.3% cases had pure metabolic seizure. Hyponatremia was the most common (60.8%) biochemical abnormalities; however hypoglycemia was most common cause for pure metabolic seizure constituting 70% of all pure metabolic seizures. Mortality rate in our study was 15.4% (33 cases, 21 male and 12 female). Most common aetiology leading to death was HIE (58%), followed by infection (24%). **Conclusion:** Out of total 214 cases, most neonates were normal birth weight, AGA and born through spontaneous vaginal delivery. Majority of neonates had onset of seizure <3 days (<72 hours). Subtle seizure was the most common seizure type and birth asphyxia the most common cause. Health care workers and parents need to be made aware of subtle seizures and the importance of timely and appropriate treatment to decrease any further complications.

**Keywords:** Birth asphyxia, Neonate, Seizures, Incidence, Aetiology

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**Introduction**

Neonatal seizures (NS) by definition occur within the first 4 weeks of life in a full-term infant and up to 44 weeks from conception for premature infants.[1] These neonatal seizures are most frequent during the first 10 days of life.[2] Neonatal seizures are relatively common neurological problem occurring with a frequency of 1.8 to 3.5 per 1000 live births.[3] It occurs with even greater frequency in premature or low birth weight babies, with incidence being as high as 57.5 per 1000 in very low birth weight infants.[4] A seizure is defined as paroxysmal electrical discharge from brain which may manifest as motor, sensory, behavioural or autonomic dysfunctions. [5] As the

immature brain in neonates are less capable of propagating generalized or organized electrical discharges, neonatal seizures differ considerably from seizures observed in older children.[6] Neonatal seizures are clinically important as they may be the initial and sometimes the only, manifestation of neurological disorder in the newborn child. Hypoxic-ischemic encephalopathy (HIE) is the commonest cause of NS, occurring in approximately 1 to 2 per 1000 live births.[7] Other causes of NS include cerebral infarction, vascular stroke, intracranial bleed, subarachnoid haemorrhage, intracranial infections, congenital malformation, metabolic and biochemical disturbances.[1] Although in about 10% of cases, no cause for NS may be identified.[8] Various evidences suggest that neonatal seizures results in adverse neurodevelopment outcome and may lead to cognitive, behavioural or epileptic complication later in life.[9] Neonatal seizures usually indicate potentially treatable aetiology and thus should be immediately evaluated to establish the

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specific cause and to institute treatment accordingly. Hence, the clinical diagnosis, classification of neonatal seizures and appropriate management are critical for the neonate's care. As neonatal seizure not only have short term but also long term effects on morbidity and mortality in these neonates, hence this study was conducted to find out incidence, etiological factor, clinical types and various biochemical abnormalities and outcome in cases of neonatal seizures which could help in early recognition and treatment of neonatal seizures and hence better prognosis in these neonates.

#### Methods

This Descriptive type of prospective cohort observational study was conducted in Neonatal intensive care unit (NICU), Department of Pediatrics, Nalanda Medical College & Hospital, Patna, Bihar, India. This study aimed to determine incidence, clinical profile, aetiology, clinical seizure types, biochemical abnormalities and short term outcome in neonatal seizures.

#### Study population

Both inborn and outborn neonates, from birth to 4 weeks of life in a full term infant and up to 44 weeks from conception for premature infants, admitted to NICU of the hospital with complaints or history of seizures, clinically apparent seizures and those neonates developing seizure during course of their NICU stay, during the study period of 1 year, were enrolled in the study. It was a hospital based descriptive type of prospective cohort observational study.

**Study duration:** The study was conducted over 1 year from 1<sup>st</sup> April 2019 till 31<sup>st</sup> March 2020.

**Sample:** 214 neonates fulfilling the inclusion criteria were included in the study after consent from parents.

#### Inclusion criteria

- 1) Newborns from birth to 4 weeks of life in a full term infants and up to 44 weeks from conception for premature infants.
- 2) Complaints or history of seizures, clinically apparent seizures and those neonates developing seizure during course of their NICU stay,
- 3) Informed consent was given by the parents.

#### Exclusion criteria

- 1) Neonates with jitteriness, tetanic spasms, isolated subtle phenomenon, apnoea or paroxysmal autonomic changes i.e. only subtle motor movements or apnoea without tachycardia
- 2) Gross congenital malformations e.g., anencephaly, large occipital meningomyelocele, microcephaly, multiple malformations
- 3) Dysmorphic features with "syndromic appearance"
- 4) Where consent was not given

Written informed consent was taken from the parent or caregiver prior to enrolment of neonate for the study. At the time of enrolment of the patients, all relevant clinical and demographic information like gender, gestational age at birth, birth weight was noted on predesigned proforma. History regarding antenatal, natal and postnatal risk factors, maternal history of any infection, drug intake, hypertension, diabetes, haemorrhage, prolonged rupture of membrane, mode of delivery, perinatal asphyxia, resuscitation (if any), traumatic delivery, preterm, small for date, low birth weight, septicaemia, intracranial bleed and hyperbilirubinemia was noted. History of consanguinity and family history of any inborn error of metabolism (IEM) also taken.

Accurate clinical description of seizure episodes reported by the informant and/or subsequently observed by resident doctors was noted for each enrolled patient. A detailed history was recorded with special emphasis on occurrence of 1<sup>st</sup> seizure, duration of seizure, number, type and consciousness during and between seizure episodes. A complete description of the seizure was obtained with associated eye movement, restraint of episode by passive flexion limbs, any associated colour change or autonomic

phenomenon and state of consciousness at the time of the seizure, and the type of seizure was taken. Seizures were classified according to Volpe's classification into subtle, multifocal clonic, focal clonic, tonic and myoclonic.[10]

A report of seizures associated with poor feeding, prolonged lethargy, recurrent unexplained vomiting, dysmorphic face, unusual body odor, hypersomnolence, with a family history of consanguinity and/or neonatal seizure with early fetal and neonatal death were taken and base-line investigation like serum ammonia, serum lactate, arterial blood gas analysis and urinary ketone and reducing substance were also done to rule out inborn error of metabolism.

A complete examination of all vital along with detailed neurological examination was done. Any congenital malformation, cutaneous markers were noted. Stage of consciousness of the patient, condition of anterior fontanel, tone, posture, deep tendon reflexes, neonatal reflexes and papillary reactions were noted. Sarnat and Sarnat score was considered for HIE grading.[11] A complete systemic examination, including the cardiovascular system, signs of respiratory distress, bleeding from the gastrointestinal tract or other sites was particularly noted as signs of multi-system involvement due to asphyxia or infection. Anthropometry was recorded and gestational age assessed according to New Ballard Scoring.

All the necessary investigation for evaluation of neonatal seizures, which included Complete Blood Counts, C-reactive protein, random blood sugar, serum calcium, magnesium and sodium, Blood urea, serum creatinine, serum bilirubin, blood culture & sensitivity, CSF examination, ultrasound head, CT scan, MRI (wherever feasible and indicated) and EEG were done as per the neonatal requirement.[12]

Criteria for diagnosing various biochemical abnormalities were[13]: 1) hypoglycemia: blood sugar < 40 mg/dL, 2) hypocalcemia: total serum calcium < 7 mg/dL, 3) hypomagnesaemia: serum magnesium < 1.5 mg/dL, 4) hypernatremia: serum sodium > 150 mEq/dL, 5) hyponatremia: serum sodium < 130 mEq/dL, 6) hypokalemia: serum potassium < 3.5 mEq/dL, and 7) hyperkalemia: serum potassium > 5.5 mEq/dL.

A total of 214 newborn satisfying our inclusion criteria were enrolled in the study after receiving the written consent from parents/caregiver. A predesigned patient performs was charted out and filled up for each newborn enrolled in the study.

#### Statistical analysis

Statistical analysis was done, using the statistical package for social science (SPSS 20) for Windows Software. Continuous data were presented as mean  $\pm$  standard deviation (SD), if normally distributed and median [interquartile range (IQR)], if data were non-normal. Categorical variables were presented as frequency and percentages (n; %). Comparability of groups was analyzed by Chi-square test, Student's t test or Mann-Whitney test as appropriate. P value of < 0.05 was considered significant.

#### Results

The total number of neonates admitted to NICU during study period of 1 year was 1488, out of which 214 neonates had one or more episodes of neonatal seizures, comprising 14.38% of all NICU admission.

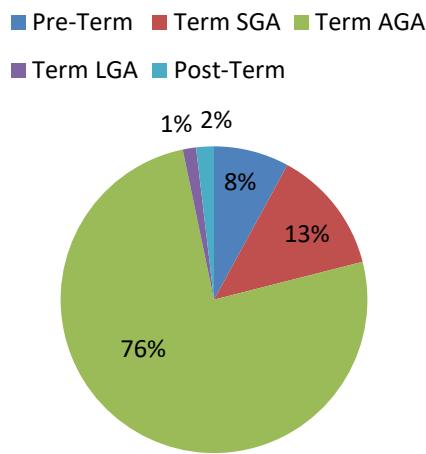
Among the 214 babies, 140 (65.4%) were male and 74 (34.6%) were female in our study. 5 (2.3%) babies were born at home and 209 (97.7%) babies at hospital. In our study, 189 (88.3%) babies were born by spontaneous vaginal delivery, 18 (8.4%) babies by caesarean section and 7 (3.3%) were by assisted delivery (forceps/ventouse). 147 (68.7%) babies were > 2.5 kg, 50 (23.4%) neonates between 2 and 2.5 kg, and 17 (7.9%) neonates were < 2 kg.(Table 1)

**Table 1: Demographic profile of study population**

	Number	Percentage
<b>Sex wise distribution</b>		
Male	140	65.4%
Female	74	34.6%
<b>Mode of delivery</b>		
Vaginal	189	88.3%
Caesarean Section	18	8.4%
Assisted	7	3.3%
<b>Place of delivery</b>		
Home	5	2.3%
Institutional	209	97.7%
<b>Birth weight</b>		
< 2kg	17	7.9%
2-2.5 KG	50	23.4%
>2.5 kg	147	68.7%

Out of

214 neonates, preterm babies were 17 (7.9%), term babies were 193 (90.2%) and post-term babies were 4 (1.9%). Among term babies (n=193), 162 (83.9%) were AGA, 28 (14.5%) were SGA and 3 (1.6%) were LGA. Of total 214 neonates, 80.8% (n=173) babies were AGA and 17.3% (n=37) neonates were SGA and 1.9% (n=4) babies were LGA in our study. (Fig 1)

**Fig 1:Weight and gestational distribution of enrolled neonates**

In our study, onset of seizure within 24 hours of delivery was found in 117 (54.7%) neonates, while convulsions from 24-48 hours of delivery were seen in 12 (5.6%) babies. Convulsions between 48-72 hours occurred in 25 (11.7%), while from the fourth day of life to seventh day of life 20 (9.3%) neonates presented with convulsions. 40 (18.7%) neonates developed seizures during 8 - 28 days. (Table 2)

**Table 2: Distribution of study population based on time of onset of 1<sup>st</sup> episode of seizure**

Time of onset of 1 <sup>st</sup> seizure	<24 hours	24-48 hours	48-72 hours	4 <sup>th</sup> – 7 <sup>th</sup> day	8 <sup>th</sup> – 28 <sup>th</sup> day	Total
Number of cases	117	12	25	20	40	214
Percentage	54.7%	5.6%	11.7%	9.3%	18.7%	100%

Most common type of seizure was subtle type which presented in 97 (45.3%) newborns. Focal clonic was 2<sup>nd</sup> most common type, presenting in 55 (25.7%) neonates. Multifocal clonic, tonic and myoclonic seizures were present in 40 (18.7%), 16 (7.5%), 6 (2.8%) neonates respectively. (Table 3)

**Table 3: Distribution of study population based on Type of seizure**

	Subtle	Focal clonic	Multifocal Clonic	Tonic	Myoclonic	Total
Number	97	55	40	16	6	172
Percentage	45.3%	25.7%	18.7%	7.5%	2.8%	100%

Birth asphyxia was most common aetiology of neonatal seizure in our study, found in 58.4% (n=125) of cases. Neonates with meningitis or septicaemia constituted 20.6% cases (n= 44, second most common). Metabolic derangement as cause of neonatal convulsion was found in 20 cases (9.3%) in our study. Intracranial bleed was aetiology for seizures in 6 cases (2.8%). Other causes of neonatal seizure were Kernicterus in 3 cases (1.4%), structural disorders in 4 cases (1.9%), and seizure disorder in 4 cases (1.9%). Cause of seizure was idiopathic/undetermined in 8 cases (3.7%).(Table 4)

**Table 4: Distribution of study population based on Aetiology of seizure**

Aetiology	Number (%)	Subtle	Focal Clonic	Multifocal Clonic	Tonic	Myoclonic
<b>Birth asphyxia</b>	125 (58.4%)	55	34	24	8	4
<b>Septicaemia/ Meningitis</b>	44 (20.6%)	21	10	8	4	1
<b>Metabolic</b>	20 (9.3%)	9	6	2	3	-
<b>Intracranial Bleed</b>	6 (2.8%)	4	1	1	-	-
<b>Structural Disorders</b>	4 (1.9%)	1	1	1	-	1
<b>Seizure disorder</b>	4 (1.9%)	2	1	1	-	-
<b>Kernicterus</b>	3 (1.4%)	1	1	1	-	-
<b>Idiopathic</b>	8 (3.7%)	4	1	2	1	-
<b>Total</b>	214	97	55	40	16	6

Out of 214 neonates, biochemical abnormalities were seen in 97 (45.3%) cases, among which non-metabolic seizures constituted 77 (79.4%) cases and pure metabolic seizures were seen in 22 (22.6%) cases. The most common biochemical abnormality in our study was hyponatremia (59, 60.8%), seen only in non-metabolic seizures, out of which 46 cases (78.9%) were due to HIE and 9 (15.2%) cases due to neonatal meningitis. The most common metabolic seizure seen was hypoglycemia (n=14), being 70% of all pure metabolic seizures. 6 (7.8%) cases of non-metabolic seizures also had hypoglycemia.(Table 5)

**Table 5: Distribution of study population based on Biochemical Abnormalities**

Biochemical Abnormalities	Hyponatremia	Hypoglycemia	Hypocalcemia	Hypomagnesemia	Hypermagnesemia	Total
Non- Metabolic (77)	59 (100%)	6 (30%)	9 (69.2%)	2 (66.7%)	1 (50%)	77(79.4%)
Metabolic(20)	0	14 (70%)	4 (30.8%)	1 (33.3%)	1 (50%)	20(22.6%)
Total (%)	59 (60.8%)	20 (20.6%)	13 (13.4%)	3 (3.1%)	2 (2.1%)	97

Mortality rate in our study was 15.4% (33 cases, 21 male and 12 female). The most common aetiology leading to death was HIE (58%), followed by infection (24%).

## Discussion

The total number of neonates admitted to NICU during study period of 1 year was 1488, out of which 214 neonates had one or more episodes of neonatal seizures, comprising 14.38% of all NICU admission, intramural incidence being 2.12% and extramural incidence being 15.06%. Bhatt et al [13] found 1% intramural and 18% extramural incidence, Kumar et al. [14] found 1.17% intramural incidence and Sahana et al. [15] found 14% extramural incidence of neonatal seizures. All of these findings were comparable with our study.

65.4% of NS cases in our study were male and 34.6% were female. This male preponderance in our results were in accordance with the studies carried out by Moayedi AR et al [16], Eghbalian F et al [17], Digras SK et al [18], and Pravin R et al [19], in which males constituted 58.2%, 73.55%, 70.58% and 76.5% of total neonatal seizure cases respectively. Sahana G et al, [20] and Sabzehei MK et al [21] also found slightly more male predominance in their respective studies. However according to study by Lanska et al [22], male sex was not associated with increased risk of neonatal seizures. Male preponderance in our study may be attributed to gender bias towards male child in our society and to the fact that male infants are better cared for and are more likely to seek prompt medical attention.

In our study, 189 (88.3%) babies were born by spontaneous vaginal delivery, 18 (8.4%) babies by caesarean section and 7(3.3%) were by assisted delivery (forceps/ventouse). This finding was similar with the study done by Das and Debbarma.[23] This higher percentage of vaginal deliveries is may be due to higher contribution of birth asphyxia to total number of neonatal seizure. Babies delivered through vaginal route had more likelihood of developing birth asphyxia. In our study, out of 214 cases neonatal seizure, 90.2% were term, 7.9% were preterm and 1.9% were posr-term babies. This result is consistent with other studies like Marzoki J et al, in which 95.4% cases of NS were among full term neonates.[24] Parvin R et al also had similar finding where 80.4% cases of NS were term and 19.6% were pre-term.[19] In our study only 68.7% cases were of birth weight > 2.5 kg. This result was consistent with other studies like Eghbalian F et al, Digras SK et al and Marzoki J et al, in which the majority of neonates had the birth weights more than 2.5 kg.[17, 18, 24] However in studies by Lanska et al [22], Ronnen et al [25] neonatal seizures occurred more commonly in premature infants than in term infants. This difference from our study may be due to fact that exact birth weight was not known in majority of our subjects and weight was taken at time of admission. In our study, onset of seizure within 24 hours of delivery was found in 117 (54.7%) neonates, while convulsions from 24-48 hours of delivery were seen in 12 (5.6%) babies. Convulsions between 48-72 hours occurred in 25 (11.7%), while from the fourth day of life to seventh day of life 20 (9.3%) neonates presented with convulsions. 40 (18.7%) neonates developed seizures during 8-28 days. These findings are in agreement with those of in studies by the Sahana G et al [20], Ronen

et al [26], Ajay Kumar et. Al [27], Joseph J. Volpe [28] and Lanska study [22] where majority of neonatal seizure cases had early onset of seizure. Birth asphyxia was found to be most common (58.4%) cause of neonatal seizure in our study, followed by meningitis or septicaemia (20.6%). Metabolic derangement, intracranial bleed, kernicterus, structural disorders, seizure disorders were cause in 9.3%, 2.8%, 1.4%, 1.9% and 1.9% cases respectively. No cause could be ascertained in 3.7% cases. These findings were consistent with studies of Digra SK et al and Parvin R et al.[18, 19] Parvin R et al found perinatal asphyxia as a cause of NS in 56.86% cases. Other causes were septicaemia (15.67%), meningitis (11.76%), kernicterus (3.92%), neurometabolic disorder (3.92%), and idiopathic (1.96%). Similarly in the study by Kumar A et al, perinatal asphyxia was the most common etiology for neonatal seizure. In a study by Digra SK et al, birth asphyxia was seen in 67.65% cases, followed by infections (septicaemia and meningitis).[18] Birth asphyxia was also the commonest cause of neonatal seizures reported by Soni et al. [29] In present study birth asphyxia is most common (58.4%) cause of neonatal seizure and comparable to Ronen and Penny (40%), Volpe JJ (50-60%) [25, 28]. Incidence of birth asphyxia in the study Ajay Kumar et al, (44.44%), Menkes et al (46%), Bridgers et al (60%) is also comparable with present study.[27, 30, 31] Pyoamnitis in 20.6% of cases in present study is also comparable to Bergman et al, (30%), Tekgul H et al (40%), Thomas and Schubert (32%), Levene and Trounce (53%).[32-35] In these studies also birth asphyxia is most common cause of neonatal seizures. In present study hypoglycaemia is cause of neonatal seizure in 6.5% cases and comparable to Menkes JH' (5%), Tekgul H et al, (2%).[30, 32] Intracranial haemorrhage is found in 2.8% cases and is comparable to Ajay Kumar et al, (4.44%), Ronen and Penny et. al. (7%).[25, 27]

In our study subtle seizure was most common (45.3%) seizure type, followed by focal clonic (25.7%), multifocal clonic (18.7%), tonic (7.5%) and myoclonic seizures (2.8%). These findings were in agreement with those seen in study by Brunquell J et al [36], in which subtle seizures were the commonest occurring in 51%, followed by focal clonic (42%), multifocal clonic (30%) and GTS (23%). Lakra et al also reported that subtle seizures were the commonest type.[37] According to John H. Menkes and Harvey B. Sarnat subtle seizure were the most common type, accounting for 71% of seizures seen in term infant, and 68% of seizures seen in preterm infants.[30] According to Joseph J. Volpe in one study of infants more than 36 weeks of gestation subtle seizure comprises of 85% and in another study subtle seizure account for 70% to 75% of all clinical seizure.[28] Out of 214 neonates, biochemical abnormalities were seen in 97 (45.3%) cases, among which non-metabolic seizures constituted 77 (79.4%) cases and pure metabolic seizures were seen in 20 (20.6%) cases. The most common biochemical abnormality in our study was hyponatremia (n=59, 60.8%), seen only in non-metabolic seizures, out of which 46 cases (78.9%) were due to HIE and 9 (15.2%) cases due to neonatal meningitis. The most common metabolic seizure seen was hypoglycemia (n=14), being 70% of all pure metabolic seizures. 6 (7.8%) cases of non-metabolic seizures also had hypoglycemia. Hypoglycemia was seen in total of 20 (9.3%) cases, contributing to 20.6% of all metabolic abnormalities. Kumar et al [27], found biochemical abnormalities in 62.8% of total cases, with hypoglycemia seen in 50%, hypocalcemia in 31.8% and hyponatremia in 45.5% of these cases of biochemical abnormalities. Among cases with primary metabolic abnormalities, hypocalcaemia was observed in 70% cases while hypoglycemia as a metabolic abnormality was detected 40% cases. Findings in our study were consistent with that of Kumar et al. Mortality rate in our study was 15.4% (33 cases, 21 male and 12 female). This is in accordance with mortality rate of 7-16% seen in studies by various authors.[25, 32, 38]

## Conclusion

Neonatal seizure occurred more commonly among male (65.4%) than female admissions, male female ratio being 1:89. 88.3% of NS occurred in babies delivered through spontaneous vaginal delivery and 97.7% neonates with seizure were hospital delivered. Most (68.7%) of these cases had birth weight > 2.5 kg and 90.2% of these case were term babies. 54.7% babies developed seizure within 24 hours of life and total 72% developed seizure within 72 hours of life. Subtle seizure was most common (45.3%) seizure type followed by focal clonic (25.7%), multifocal clonic (11.7%), tonic (7.5%) and myoclonic (2.8%). Birth asphyxia was most common (58.4%) cause followed by septicaemia/meningitis (20.6%). 42% had biochemical abnormalities but only 9.3% cases had pure metabolic seizure. Hyponatremia was the most common (60.8%) biochemical abnormalities; however hypoglycemia was most common cause for pure metabolic seizure constituting 70% of all pure metabolic seizures. The most common cause for death was HIE (58%), followed by infection (24%). To conclude it is of utmost important to improve antenatal and perinatal care of pregnant women to reduce the incidence of birth asphyxia, which was the most common cause of neonatal seizure in our study. Early recognition and aggressive management of neonatal infection is keystone to reducing meningitis. It is vital to educate parents and health care providers for them to be able to identify a subtle seizure, one of the most common types of seizure in neonates. Timely recognition and early correction of biochemical abnormalities have better outcome than other causes.

## Limitations

It is a single centre study with relatively small sample size. It included both inborn as well as outborn neonates and details about the quality of antenatal and perinatal care as exact birth weight of most outborn children was not known. A more robust, multicentric study is need of the hour.

## References

1. Singh M. Neurology disorders. In: Singh M, eds. Textbook of Care of Newborn. 2nd ed. Delhi: Sagar Publication; 1999: 340-344.
2. Vigavano F. Benign familial infantile seizures. *Brain Dev*. 2005;27:172.
3. Mizrahi Eli M. Neonatal Seizures and Neonatal Epileptic Syndromes. *Neurologic Clinics In Epilepsy*. 2001; 19 (2): 427-456.
4. Sankar MJ, Agarwal R, Aggarwal R, Deorari AK, Paul VK. Seizures in the newborn. *Indian J Pediatr*. 2008;75(2):149-55.
5. Mikati MA, Kliegman RM, Behrman RE, Stanton BF. Seizures in childhood; Nelson textbook of paediatrics,19th edition. Philadelphia: WB Saunders[586]. 2011; 2013-2037.
6. Gregory HL, Roustem K, Yehezkel B. New concepts in neonatal seizures. *Neuroreport*. 2002;31:A3-8.
7. Volpe JJ. Hypoxic-Ischemic Encephalopathy: Biochemical and Physiological Aspects. In: *Neurology of the Newborn*. 4th ed. Philadelphia: WB Saunders; 2000:217-276.
8. Neurological disorders. In: Singh M. Textbook Of Care Of New Born 5th ed., New Delhi: Sagar publication,1999: 340-344.
9. Levene M. Clinical Conundrum Of Neonatal Seizures. *Arch Dis Child Fetal Neonatal* 2002; 86 (2): 75-77.
10. Volpe J. Neonatal seizures. *N Engl J Med*. 1973;289(8):413-6.
11. Sarnat HB, Sarnat MS. Neonatal encephalopathy following fetal distress. A clinical and electroencephalographic study. *Arch Neurol* 1976;33:696-705.
12. Hallberg B, Blennow M. Investigations for neonatal seizures. *Semin Fetal Neonatal Med* 2013;18:196-201.
13. Bhatt S, Raju N, Phanse S, Patel SV, Madan G, Mehta S, et al. Clinico-etiological and EEG profile of neonatal seizures. *Ind J Clin Pract*. 2013;24(1):69-75.

14. Kumar A. Clinicoetiological and EEG profile of neonatal seizure. Indian J Paediatr. 2007;74(1):33-7.
15. Sahana G, Anjaiah B. Clinical profile of neonatal seizures. Int J Med Applied Sci. 2014;3(1):21-7.
16. Moayedi AR, Zakeri S, Moayedi F. Neonatal seizure: etiology and type. Iran J Child Neurol. 2008;23-26.
17. Eghbalian F, Monsef A, Taghdiri MM. Neonatal seizures: etiology and frequency. Iran J Child Neurol. 2008;39-42.
18. Digras SK, Gupta A. Prevalence of seizures in hospitalized neonates. JK Sci. 2007;9:27-29.
19. Parvin R, Salim AF, Rahman M, Chowdhury K, Sultana A, Ahmed S, et al. Neonatal seizures: correlation between clinico-etiological profile and EEG findings. Bangladesh J Child Health. 2014;38(1):19-23.
20. Sahana G, Anjaiah B. Clinical profile of neonatal seizures. Intern J Med Applied Sci. 2014;3(1):9
21. Sabzehei MK, Basiri B, Bazmamoun H. The etiology, clinical type and short outcome of seizure in Newborns hospitalized in Basat hospital, Hamadan, Iran. Iran J Child Neurol. 2014; 8(2):24-8.
22. Lanska MJ, Lanska DJ, Baumann RJ, Kryscio RJ. A population-based study of neonatal seizures in Fayette County, Kentucky. Neurol. 1995;45(4):724-32.
23. Das D, Debbarma SK. A Study on Clinico-Biochemical Profile of Neonatal Seizure. J Neurol Res. 2016;6(5-6):95-101.
24. Marzoki JMA. Clinico-Biochemical profile of neonatal seizures (Research Thesis). Hilla-Iraq- Babylon: Babylon University; 2009.
25. Ronen GM, Penney S, Buckley D, Streiner DL. Long-term prognosis in children with neonatal seizures: a population-based study. Neurol. 2007;69:1816-22.
26. Ronen GM, Rosales TO, Connolly M, Anderson VE, Leppert M. Seizure characteristics in chromosome 20 benign familial neonatal convulsions. Neurol. 1993;43(7):1355.
27. Kumar A, Gupta V, Kachhwaha JS, Singla PN. Biochemical abnormalities in neonatal seizures. Indian Pediatr. 1995; 32(4):424-428.
28. Volpe JJ. Neonatal Seizures, Neurology of the newborn, 5th ed. Philadelphia: Saunders Elsevier; 2008:203-244.
29. Soni A, Sabarwal RK, Amita K. Clinical profile of seizures in neonatal intensive care unit. Manual abstracts of XXIII Annual Convention of National Neonatology Forum. 2003(43):109-110.
30. Menkes JH, Sarnat HB. Child Neurology, 7th ed. Philadelphia: Lippincott William & Wilkins; 2005:991-995.
31. Bridgers SL, Ebersole JS, Ment LR, Ehrenkranz RA, Silva CG. Cassette electroencephalography in the evaluation of neonatal seizures. Archives Neurol. 1986;43(1):49-51.
32. Tekgul H, Gauvreau K, Soul J, Murphy L, Robertson R, Stewart J, et al. The current etiologic profile and neuro developmental outcome of seizures in term newborn infants. Pediatrics. 2006;117(4):1270-80.
33. Bergman I, et al. Outcome in neonates with convulsions treated in an intensive care unit. Annals of Neurology. 1983;14:642-7
34. McInerny TK, Schubert WK. Prognosis of Neonatal Seizures. Am J Dis Child. 1969;117(3):261-264.
35. Levene MI, Trounce JQ. Cause of neonatal convulsions. Towards more precise diagnosis. Arch Dis Child. 1986 Jan;61(1):78-9.
36. Brunquell PJ, Glennon CM, DiMario FJ Jr, Lerer T, Eisenfeld L. Prediction of outcome based on clinical seizures type in newborn infants. J Pediatr. 2002;140:707-12.
37. Mahaveer L, Vilhekar KY, Chaturvedi P. Clinico-biochemical profile of neonatal seizures in a rural medical college. In: Fernandez AIH, Dadhich JPP, Saluja YS, Editors, Abstracts, XXIII Annual Convention of National Neonatology Forum, Dec. 18-21, 2003; Hyderabad. 2003:121-122
38. Jensen FE. Neonatal Seizures: An Update on Mechanisms and Management. Clin Perinatol. 2009;36(4):881-900.

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