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RESEARCH ARTICLE

Neurodevelopmental outcome of new-borns with neonatal seizures at 6 and 12 months of age – a prospective cohort study at Children Hospital Bemina, GMC Srinagar, Jammu and Kashmir, India.

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Abstract:

Introduction: Neonatal seizures represent the most prevalent neurological disorder in newborns and are critical for neurodevelopmental outcomes [1]. These seizures frequently occur during the neonatal period, with an incidence ranging from 1.5 to 5.5 per 1000 live births, and are more prevalent in preterm infants due to brain immaturity and increased risk of injury [2,3]. They signify neurological dysfunction, which may be reversible or persistent depending on the underlying cause. Prompt diagnosis and intervention are imperative to reduce mortality and mitigate long-term neurological consequences.

Methods: This prospective cohort study was conducted on neonates admitted with seizures between January 2022 and March 2023 in the Neonatology Division, Department of Pediatrics, GMC Srinagar. Following history taking, physical examination, and etiological screening, data were systematically recorded. Neonates were monitored in a high-risk neonatal outpatient department, conducted biweekly. The Amiel Tison neurological examination was performed at discharge and during follow-up. Neurodevelopmental assessment was conducted at 6 and 12 months using the Denver Developmental Screening Test II (DDST – II) in collaboration with a clinical psychologist. Univariate analysis was employed to examine the relationship between risk factors and neurodevelopmental outcomes. Categorical data were compared using the Chi-square or Fisher's exact test, with statistical significance set at $p \leq 0.05$. Relative Risk (RR) with a 95% confidence interval (CI) was calculated.

Results: During the study period, 367 neonates were admitted to the NICU with seizures. After applying the selection criteria, 120 neonates were included in the study. It was observed that 61.66% (n=74) exhibited normal development,

11.66% (n=14) experienced developmental delay in two domains with scores below 70%, 8.33% (n=10) had Global Developmental Delay, 5% (n=6) were diagnosed with cerebral palsy, 1.66% (n=2) developed postnatal epilepsy, 6.66% (n=8) succumbed to complications, and 5% (n=6) were lost to follow-up.

Conclusion: Hypoxic-ischemic encephalopathy was significantly associated with developmental delay in 33% of patients, with a relative risk (RR) of 2.26. Developmental delay was also observed in cases of meningitis and intraventricular hemorrhage; conversely, metabolic causes such as hypoglycemia and hypocalcemia demonstrated favorable outcomes when treated promptly. Prolonged and recurrent hypoglycemia was linked to impaired neurodevelopmental outcomes. Adverse prognostic factors included gestational age at birth, APGAR Score at 5 minutes, the necessity for resuscitation beyond 5 minutes, neonatal status epilepticus, onset of seizures within 24 hours, abnormal neurological examination at discharge, and abnormal EEG or neuroimaging findings.

Introduction

Neonatal seizures significantly impact neurodevelopmental outcomes [1]. They are common in neonates, with an incidence of 1.5 to 5.5 per 1000 live births, higher in preterm infants due to brain immaturity and injury risks. Outcomes depend on etiology, gestational age, EEG activity, and antiepileptic response [2]. Prompt diagnosis and treatment are crucial to reduce mortality and long-term neurological issues. Most seizures are acute, symptomatic, and caused by severe brain insults like HIE or ICHs, but some involve neonatal-onset epilepsy linked to structural, metabolic, or genetic disorders [3,4,5]. Few studies focus on neurodevelopmental outcomes, especially in developing countries. This study assessed outcomes of neonates with seizures, emphasizing etiology-based outcomes over a year.

Material and Methods

After INSTITUTIONAL ETHICAL COMMITTEE clearance (Notification No: F(BOPGS-Medicine) Acad/KU/23 Dated-31-08-2023), the study was conducted on neonates admitted with seizures in the Division of Neonatology, Department of Paediatrics at Govt.Children HOSPITAL, a 500 Bedded Tertiary Care Hospital of Govt. Medical College, Srinagar. Neonates admitted from January 2022 to March 2023 were included. Neonates aged 34-41 weeks and weighing 2-4 KG were included. Exclusions were congenital anomalies, inborn errors of metabolism, and syndromic features.

History, examination, and etiologic screening with investigations were done. Neurological Assessment used the Amiel Tison method, a clinical tool to evaluate newborns' neurological status, focusing on muscle tone, reflexes, posture, and head control.

Developmental Assessment

Developmental assessment was supervised by a clinical psychologist using the Denver Developmental Screening Test II (DDST – II), assessing child development in:

- Gross motor.
- Fine motor-adaptive.
- Language.
- Personal social.

It includes 125 items divided into categories, arranged chronologically by age. The test took 10 – 20 minutes.

Interpretation of DDST-II:

- Normal – if a child passes, fails, or refuses an item where the age line falls between the 25th and 75th percentiles.

- Delayed – When a child fails or refuses a task within the age line cut through >75th percentile.

Statistical analysis:

Baseline data was recorded in a case record form, and a master sheet was prepared in an MS Excel worksheet. This was analyzed using SPSS Version 22. Standard statistical methods were used, with continuous variables shown as Mean (± Standard Deviation) or Median (IQR) based on data distribution. A descriptive analysis of the study population was done. A univariate analysis studied the relationship of risk factors with neurodevelopmental outcomes. Chi-square or Fischer's exact test compared categorical data. A P-Value ≤ 0.05 was considered statistically significant. Relative Risk (RR) with a 95% confidence interval (C.I) was calculated.

Results:

A total of 7234 out born neonates were admitted during the study period, of which 367 (5.07%) had neonatal seizures. After applying inclusion and exclusion criteria, 120 neonates were enrolled for study outcomes. Of these, 8 (6.6%) died due to complications, 6 (5%) were lost to follow up. Finally, 106 (88.3%) neonates were followed up and neurodevelopmental assessment was done at 6 and 12 months of age. Of 120 neonates, 56.6%[n=68] were males and 43.3%[n=52] females, 17.5% (n=21) were low birth weight (LBW) and 82.5% [n=99] had normal birth weight, 30% (n=36) were moderate to late preterm and 70%(n=84) were term babies (table 3). 36.6% (n=44) babies were delivered by normal vaginal delivery (NVD) while 63.3% (n=76) by LSCS. 63.3%(n=76) had an APGAR Score at 5 minutes ≥7 (normal), 21.6%(n=26) had a score of 4 to 6 (moderately depressed) and 15% (n=18) were severely depressed with a score of 0 to 3.

Onset of seizures within 24 hours was seen in 45% (n=54) neonates, 30% (n=36) presented between 24 to 72 hours of life while 25% (n=30) had seizures after 72 hours. Most common type of seizures were subtle seizures seen in 50% (n=60), followed by clonic 26.6%(n=32), myoclonic 12.5%(n=15) and Tonic seizures in 10.8% (n=13). The most common cause of neonatal seizures was Hypoxic Ischemic Encephalopathy (HIE) seen in 45.83% (n=55), followed by metabolic causes (33.3%) like hypoglycemia 13.33%(n=16), hypocalcemia 20% (n=24). Meningitis was seen in 11.6% (n=14), Post-natal epilepsy in 5% (n=6) cases, intraventricular haemorrhage (IVH) in 2.5%(n=3), bilirubin encephalopathy with seizures in 1.66% (n=2).

Ultrasonography cranium was normal in 71.6%(n=76) and abnormal findings like dilated ventricles, raised periventricular echogenicity, IVH were seen in 28.3% (n=30) cases. MRI Brain was done in 90 cases, abnormal in 33.9% (n=36). Electroencephalography (EEG) was done in 85 cases, normal in 51.8%(n=55) and abnormal in 28.3% (n=30) cases. Abnormal EEG findings included focal or multifocal discharges, abnormal background EEG, burst suppression pattern. BERA was done in 19 out total 22 cases of meningitis and bilirubin encephalopathy with seizures and it was abnormal in 13.6% of cases. Abnormal BERA was reported as increased threshold or latency seen in I, III, or V waves.

At the time of discharge from hospital, neurological assessment revealed 83% (n=88) to be mentally alert, 17% (n=18) were neurologically depressed, 69.8%(n=74) had normal tone, 27.3%(n=29) had hypertonia with exaggerated reflexes, 2.8%(n=3) had hypotonia. Head circumference for age was normal in 92.4% (n=98), microcephaly was seen 1.8%(n=2) , macrocephaly was seen in

5.6% (n=6) cases as per Fenton growth charts. Incomplete Moros reflex was seen 9.4%(n=10) cases while it was completely absent in 7.5%

(n=8) cases. Suckling reflex was Sustained in 84.9% (n=90), Unsustained in 9.4% (n=10%) and absent in 5.6% (n=6).

Poor prognostic factors associated with impaired neurodevelopmental outcome included Gestational age, APGAR Score at 5 minutes, need for resuscitation after 5 min., neonatal status epilepticus, onset of seizures less than 24 hours, abnormal neurological exam. at discharge, abnormal EEG or Neuroimaging. Unfavourable outcome (developmental delay, cerebral palsy) was seen in 17.9% (n=17) patients born as preterm compared to 14% (n= 15) term babies. This was found to be statistically significant with a p value < 0.05 and confidence interval 1.8-5.2. APGAR score less than seven after five minutes of birth was associated with unfavourable outcome in 18.8%(n=20) patients. This was statistically significant with p value 0.002 and C. I = [1.3-3.9]. Patients who needed extra resuscitation after 5 minutes of birth like intubation, chest compressions were associated with unfavourable outcome in 19.8%(n=21) and this was statistically significant with p value 0.0002 and C.I [1.5-3.9].

Similarly, onset of seizures less than 24 hours was associated with unfavourable outcome in 23.5% (n=25) with a statistically significant p value 0.0015 and C. I [1.34-4]. Neonatal status epilepticus was associated with unfavourable outcome in 17.9%(n=19) which is statistically significant with p value 0.00007 and C. I [1.7-5]. Abnormal neurological exam. at discharge was associated with unfavourable outcome in 16.9%(n=18) with a statistically significant p value 0.0004and C.I [1.5-4.4].Abnormal EEG was associated with unfavourable outcome in 16%(n =17) which was statistically significant with a p value 0.001 and C.I [1.44.6].Abnormal cranial ultrasonography or MRI Brain was associated with unfavourable outcome in 16%(n=17) which was statistically significant with p value 0.012 and C. I [1.153.8].

As far as aetiology based outcome is concerned, Hypoxic Ischemic encephalopathy was found to be strongly associated with developmental delay in 33%(n=35) with a relative risk (R.R) of 2.26, C.I [1.48-3.44] and p value 0.000044. Metabolic causes like hypoglycemia and hypocalcaemia have favourable outcome with developmental delay seen in only 6%(n=7) and R. R 0.36 and significant p value 0.00028. This is because most of the metabolic causes being reversible and early diagnosis and treatment prevent long Neurological sequeale. Meningitis was associated with developmental delay in 5.6%(n=6) with statistically significant p value 0.047, relative risk of 0.54 and C.I [0.271.10]. Intraventricular haemorrhage(IVH) was associated with developmental delay in 1.8%(n=2) with a R.R of 1.34 and C.I [0.59- 3.06]. This was statistically insignificant with p value 0.55, possibly due to small sample size of preterm babies. Postnatal epilepsy was associated with developmental delay in 2.8%(n=3) with R. R 2.06, Confidence interval (C.I) [1.68-2.5] and p value 0.07. We found 61.66% (n=74) babies with neonatal seizures had normal development, 11.66% (n=14) had developmental delay in or two domains less than 70%, 8.33%(n=10) had Global Developmental Delay, 5%(n=6) developed cerebral palsy, 1.66%(n=2) had postnatal epilepsy, 6.66%(n=8) died due to complications and 5% (n=6) were lost to follow up. (table 1).

Table 1. Neurodevelopmental outcome of neonatal seizures at 6 and 12 months

Outcome	At 6 months		At 12 months	
	Number	Percentage	Number	Percentage
Normal	68	56.6%	74	61.66%
Developmental delay in one or two domains < 70%	24	20%	14	11.66%
Global developmental delay	6	5%	10	8.33%
Cerebral palsy	6	5%	6	5%
Postnatal epilepsy	2	1.6%	2	1.6%
Death	8	6.6%	8	6.66%
Loss to follow up	6	5%	6	5%

Table 2. Association of Etiology of Neonatal seizures with Developmental delay at 1 year of age.

Outcome ► / Etiology ▼	Developmental Delay			Total	Relative Risk (R.R)	Confidence internal (C.I)	Chisquare	P-Value
Hypoxic Ischemic Encephalopathy		Yes	No		2.26	1.48-3.44	16.7	0.000044
	Yes	35	14	49				
	No	18	39	57				
Metabolic causes	Yes	7	24	31	0.36	0.18-0.72	13.17	0.00028
	No	46	29	75				
Meningitis	Yes	6	14	20	0.54	0.27-1.10	3.9	0.047
	No	47	39	86				
Intraventricular Hemorrhage (IVH)	Yes	2	1	3	1.346	0.59-3.06	0.34	0.55
	NO	51	52	103				
Postnatal Epilepsy	YES	3	0	3	2.06	1.68-2.5	3.08	0.07
	No	**50**	**53**	**103**				

Table 3: Association of different prognostic variables of neonatal seizures with neurodevelopmental outcome.

Variables		Unfavourable outcomes (n%)	Favourable outcomes (n%)	Total	Confidence interval	P-value
Mode of delivery	NVD	10 (9.4%)	29 (27.3%)	39	0.5-2.2	>0.05
	LSCS	15 (14.1%)	52 (49%)	67		
Gestational age	Preterm	19 (17.9%)	12 (11.3%)	31	1.8-5.2	<0.05
	Term	15 (14%)	60 (56.6%)	75		
APGAR at 5 min	< 7	20 (18.8%)	19 (17.9%)	39	1.3-3.9	0.002
	≥ 7	15 (14%)	52 (49%)	67		
Need for resuscitation after 5 min of birth	Extra	21 (19.8%)	13 (12%)	34	1.5-3.9	0.0002
	Routine	18 (16.9%)	54 (50.9%)	72		
Onset of seizures	<24 hours	25 (23.5%)	23 (21.6%)	48	1.34-4.0	0.0015
	>24 hours	13 (12.2%)	45(42.4%)	58		
Neonatal Status epilepticus	Present	19 (17.9%)	13 (12.2%)	32	1.7-5.0	0.00007
	Absent	15 (14%)	59 (55.6%)	74		
Neurological examination	Abnormal	18 (16.9%)	14 (13.2%)	32	1.5-4.4	0.0004
	Normal	16 (15%)	58 (54.7%)	74		
EEG	Abnormal	17 (16%)	13 (12.2%)	30	1.4-4.6	0.001
	Normal	12 (11.3%)	43 (40.5%)	55		
USG cranium /MRI brain	Abnormal	17 (16%)	19 (17.9%)	36	1.15-3.8	0.012
	Normal	12 (11.3%)	42 (39.6%)	54		

Discussion:

In our study, 7234 outborn neonates were admitted in Neonatology Division of which 367 had neonatal seizures, showing an incidence of 5.07%, similar to other studies like Perrine Plouin et al. (2013) [35] who reported an estimated incidence between 1.5 to 5.5 per thousand live births. After applying inclusion and exclusion criteria, 120 neonates were selected for the study. Baseline clinical data was obtained with proper history and exam. and appropriate lab. investigations were done for etiologic screening. Data was entered in case record form. All enrolled cases were investigated and managed as per standard hospital protocol. Before discharge, neurological assessment using Amiel tison method was done in every baby and recorded. These were kept on regular follow up in high risk neonatal OPD conducted twice a week and again followed up at 6 and 12 months to access their neurodevelopmental outcome. DDST-II scale was used for neurodevelopmental assessment. After assessing neurodevelopmental outcome of neonatal seizures at 6 and 12 months, we found that at 6 months: 56.6% of infants demonstrated normal development. At 12 months, this increased to 61.6%. This finding aligns with Glass et al. (2009) [37] who reported that approximately 50-60% of infants with neonatal seizures exhibited normal neurodevelopmental outcomes by 12 months[7]. However, variations in the definition of "normal development", underlying causes, population studied or interventions used and assessment tools can affect these percentages.

At 6 months, 20% of infants had developmental delays in one or two domains with individual DQ less than 70%, but at 12 months this proportion decreased to 11%. This could be explained by continuous CNS maturation and early stimulation/ intervention leading to improved developmental outcome at 12 months. This necessitates early prognostication and intervention. In our study, Global Developmental Delay (GDD) was found in 5% of infants at 6 months, increasing to 8.3% at 12 months. Overall developmental delay was seen in 26.65% cases. These results align with previous studies like Pellegrin S et al (2019) [11] and Spagnoli C et al (2024) [12] which reported overall developmental delay of 30-40% including GDD. The proportion of cerebral palsy cases at 6 and 12 months was 5%, comparable to previous studies. Glass et al. (2009) observed a cerebral palsy rate of approximately 6-8% in their cohort by 12 months [37]. This minor variation may be due to differences in underlying causes of seizures or population studied. Tekgul et al. (2006) reported cerebral palsy in 5-10% of infants who experienced neonatal seizures, particularly in those with severe hypoxic-ischemic encephalopathy (HIE) [13]. Our findings fall within this range. Postnatal Epilepsy was seen in 1.6% of infants at 6 and 12 months. Pisani F et al. (2012) reported during their seven-year follow-up that approximately 17.6% of infants with neonatal seizures developed postnatal epilepsy [14]. This higher rate could be due to differences in population studied, longer follow-up, genetic predispositions, limited genetic studies in our setup, or severity of initial brain injury. Among enrolled neonates, mortality was 6.6% after one-year follow-up. Similar results were obtained in other studies like Tekgul et al. (2006), who reported a 7% mortality rate in infants with neonatal seizures, particularly those associated with severe HIE [13]. The mortality rate has significantly decreased with time, possibly due to improved antenatal, intranatal and postnatal care. However, mortality after neonatal seizures ranges from 7-30% as per literature review.

Heljic S et al (2016) reported 23% mortality after one year [15]. The difference in mortality rates across studies could be due to different sample sizes or population characteristics. Overall, comparison of our study's findings with other research indicates both consistency and variation in neurodevelopmental outcomes following neonatal seizures. Our study shows similar trends in prevalence of normal development, developmental delays, cerebral palsy, and mortality. However, there are differences in rates of postnatal epilepsy and developmental delays in specific domains, which could be attributed to differences in study populations, interventions, and follow-up protocols.

Our study showed that neonates with severe HIE had a significantly higher risk of developmental delay (R.R = 2.26, C.I. 1.48-3.44, p = 0.000044), consistent with Glass et al. (2017) [9] and Sanjeev Sudia et al. [6] who reported poor outcomes with HIE-III. Neonatal seizures from metabolic causes had a favorable outcome, with a lower risk of developmental delay (R.R = 0.36, C.I. = 0.18-0.72, p = 0.00028). Treated hypocalcemia seizures were not linked to developmental delay, aligning with Yi-Chieh Huang et al [17]. Hypoglycemic seizures showed varied outcomes, with R Shah et al [20] noting no universal safe blood glucose threshold. We aggressively treated hypoglycemia in high-risk neonates, finding 2 of 16 hypoglycemic seizure cases with developmental delay, one developing West Syndrome and another with visual defects and occipital lobe atrophic changes, both with severe hypoglycemia. Early identification of metabolic causes can prevent seizures and improve long-term outcomes. Christopher J D McKinlay et al. found that maintaining blood glucose at least 47 mg/dL did not increase neurosensory impairment risk [19], supported by Ramesh Bhat Y. et al. [16] and Rasmussen et al [18].

To conclude, Mild and transient hypoglycemia when treated early has less chances of neurodevelopmental impairment as brain utilizes ketones, amino acids till hypoglycemia gets corrected. However, severe and recurrent hypoglycemia is definitely associated with neurodegenerative changes in brain. It may lead to developmental delay, cerebral palsy or even death, Emily W.Y. Tam et al [21].

In our study, neonatal meningitis was associated with developmental delay with a statistically significant association (R.R=0.54, C. I 0.27-1.10, p =0.047). This is similar to the study by Darrah N Haffner et al. [22] where 30% survivors had development delay. Another study by G Klinger et al.2000 [23] showed 16.8%(17) infants having moderate to severe disability at one year. Infants with IVH showed a relative risk of 1.34 for developmental delay with C. I. 0.59-3.06 and p value=0.55. The statistically insignificant p value could be due to small sample size of extreme and very low preterm babies excluded from our study. Literature suggests grade III-IV IVH is associated with poor neurodevelopmental outcome. Srinivas Bolisetty et al. [24] found infants with grade III-IV intraventricular haemorrhage (IVH; n = 93) had higher rates of developmental delay (17.5%), cerebral palsy (30%), deafness (8.6%), and blindness (2.2%). Infants who developed postnatal epilepsy had the highest relative risk of developmental delay (R.R = 2.06, C.I 1.68-2.5, p = 0.07). A study by Pisani F et al (2007) [25] shows post neonatal epilepsy presents with distinct EEG patterns and seizure types, is highly refractory, and carries an adverse prognosis. Battaglia D et al. [26] concluded that Developmental and Epileptic Encephalopathies are usually genetic and carry poor neurodevelopmental outcome. In conclusion, HIE III, post-natal epilepsy and meningitis are associated with the highest risk of developmental delay, while metabolic causes seem to carry a favourable prognosis.

Association between perinatal and neonatal

factors affecting neurodevelopmental outcome of neonatal seizures:

1. Mode of Delivery (NVD vs. LSCS) Our study shows no statistically significant association between mode of delivery (Normal Vaginal Delivery [NVD] vs. Lower Segment Caesarean Section [LSCS]) and neurodevelopmental outcomes ($p > 0.05$). Both groups had comparable incidence of unfavourable outcomes. Previous studies show conflicting results. Some report elective caesarean sections may reduce HIE risk, a major cause of neonatal seizures. However, a cohort study by Zhu JJ et al. (2014) [27] suggested no significant difference in long-term outcomes.
2. Gestational Age (Preterm vs. Term) A significant association was found between gestational age and neurodevelopmental outcomes ($p < 0.05$). Preterm infants showed higher rate of unfavourable outcomes (17.9%) compared to term infants. This could be attributed to high risk injury to immature brain of preterm neonates. Preterm birth is a well-established risk factor for adverse neurodevelopmental outcomes in neonates with seizures. Studies by Song IG et al. (2023) [28]and Pisani et al. (2016) [29] have consistently shown that preterm infants have poorer outcomes compared to term infants due to increased vulnerability of the immature brain to injury.
3. APGAR Score at 5 Minutes Infants with an APGAR score < 7 at 5 minutes had more unfavourable outcomes (18.8%) than those with a score >= 7 (14%), with a p-value of 0.002. Low scores are linked to higher risks of neonatal encephalopathy and poor outcomes. Studies by Razaz N et al. (2019) [30] highlight risks of seizures and developmental delays.
4. Need for Resuscitation After 5 Minutes Neonates needing resuscitation beyond 5 minutes had worse outcomes (19.8% unfavourable) compared to those needing routine care (16.9% unfavourable), with a p-value of 0.0002. Prolonged resuscitation indicates underlying asphyxia or perinatal complications leading to hypoxic-ischemic injury, as shown by Perlman et al. (2016).[31]
5. Onset of Seizures (< 24 hours vs. > 24 hours) Early-onset seizures had a higher rate of unfavourable outcomes (23.5%) than late-onset seizures (12.2%), with a p-value of 0.0015. Early-onset seizures relate to acute brain injuries like HIE, posing a higher risk for long-term impairment. Studies by Anand V et al. (2014) [32] found early-onset seizures strongly predict poor outcomes.
6. Neonatal Status Epilepticus Neonates with status epilepticus had more unfavourable outcomes (17.9%) than those without (14%), with a p-value of 0.00007. Status epilepticus is linked to high mortality and morbidity. Studies by Shellhaas et al. (2018) [33] associate it with extensive brain injury and poor outcomes.
7. Neurological Examination (Abnormal vs. Normal) An abnormal neurological examination at discharge was linked to worse outcomes (16.9% unfavorable), with a p-value of 0.0004. Neurological abnormalities strongly predict adverse outcomes in neonates with seizures, as shown by K.Famra et al.[8].
8. EEG Findings Abnormal EEG findings were linked to unfavourable outcomes (16%), with a p-value of 0.001. Abnormal EEG patterns like burst suppression and ictal spread to the contralateral hemisphere predict poor outcomes[10]. Wusthoff et al. (2019) [34] reported similar findings.
9. Imaging Findings (USG Cranium/MRI Brain) Neonates with abnormal cranial ultrasound or MRI findings had worse outcomes (16%

unfavourable), with a p-value of 0.012. Abnormal neuroimaging findings are linked to adverse outcomes. Studies by Yvonne W Wu et al. (2023) [36] emphasized MRI's role in predicting outcomes in neonates with seizures.

To conclude, these factors impact the neurodevelopmental outcome of neonatal seizures. These findings align with existing literature, emphasizing the importance of early identification and intervention.

Conclusion:

Hypoxic-ischemic encephalopathy (HIE) has been identified as being strongly associated with developmental delay, accounting for the highest number of cases. Developmental delay was also observed in instances of meningitis, intraventricular haemorrhage, and postnatal epilepsy. In contrast, metabolic causes such as hypoglycemia and hypocalcaemia tend to have favourable outcomes when treated promptly. However, prolonged and recurrent hypoglycemia is linked to impaired neurodevelopmental outcomes. Poor prognostic factors associated with impaired neurodevelopmental outcomes include gestational age at birth, APGAR score at 5 minutes, the necessity for resuscitation after 5 minutes, neonatal status epilepticus, seizure onset within 24 hours, abnormal neurological examination at discharge, and abnormal EEG or neuroimaging findings. The most common cause of neonatal seizures was found to be HIE, followed by metabolic causes such as hypoglycemia, hypocalcaemia, and infections like meningitis. This underscores the need for improvements in antenatal, perinatal, and postnatal care of neonates. Additionally, comprehensive follow-up and individualized care plans are essential for enhancing long-term outcomes.

Limitations:

Due to non-availability of aEEG/cEEG in our setting, some neonates with only electrographic seizures could have been missed leading to decrease in sample size. This emphasizes the need of aEEG/cEEG to monitor subtle seizures so that these patients can be kept on follow up to assess their neurodevelopmental outcome.

Research Implications:

- Neurodevelopmental outcome of neonatal seizures based on Continuous EEG monitoring needs to be studied in detail to predict outcome of even subtle seizures.
- Long-term and multicenter studies are required in order to to develop accurate risk models of poor neurodevelopmental outcome.

Conflict of interest:

None

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