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Neonatal Seizure in a Tertiary Center Work

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Abstract

Background: Neonatal seizures are the most common neurological emergency in newborns, often associated with significant mortality and long-term neurodevelopmental disabilities. The aim is to determine the incidence, etiological causes, and risk factors associated with neonatal seizures.

Patients and Methods: This prospective case-control study was conducted over eight months, from January 1 to August 31, 2022, the study was conducted at the neonatal care unit of Children Welfare Teaching Hospital. Neonates who developed clinically recognizable seizures before 28 days of life in term infants, or up to 44 weeks corrected gestational age in preterm infants, were included. Data collection involved demographic information, prenatal, perinatal, and postnatal history, family history, seizure characteristics, physical examination including growth parameters, and relevant laboratory and radiological investigations. Follow-up was conducted two months later via phone interviews.

Results: Among 180 neonates included, 50% had seizures while the remaining served as controls. The incidence of seizures among admitted neonates was 7.7%. The three main etiologies were birth asphyxia, infection, and metabolic disorders, each accounting for 25.6% of cases. The mean age at seizure onset was 9.3 ± 9.1 days, with a median of 5 days. Males were more affected (male-to-female ratio 1.57:1), and seizures were more common in term infants. Significant associations were found with family history of neurological disease and neonatal death. Vaginal delivery was more linked to birth asphyxia, while cesarean delivery was associated with neurological malformations. Mortality was three times higher in neonates with seizures.

Conclusion: Birth asphyxia, infection, and metabolic disorders are leading causes of neonatal seizures. A positive family history and prematurity increase susceptibility. Early onset may indicate etiology, but seizure type does not predict cause. Seizure presence significantly increases neonatal mortality.

More Information

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Introduction

The historical trajectory of epilepsy reveals remarkable continuity in its clinical manifestations despite evolving terminology over millennia. The term *seizure*, rooted in the Greek for "to take hold," has been interchanged with epilepsy, convulsion, and attack throughout historical texts. The earliest known description dates back to Sumerian records around 2500 BC, depicting symptoms akin to modern focal unaware tonic seizures. This condition, then termed *antašubbû* or "the falling disease," was believed to be linked to divine forces, notably the Moon God Sin. Babylonian texts such as *Sakikku* (c.1050 BC) used terms like *miqtu* and *ṣibtu* to denote seizure-like conditions, indicating a belief in

spiritual possession as a cause [1]. In Ancient Greece, thinkers like Alcmaeon of Croton proposed the brain as the center of cognition and spiritual function. Epilepsy was viewed with awe and reverence, often labeled the "sacred disease," especially given that figures like Hercules and Caesar were believed to have suffered from it, thereby elevating its status as a mark of genius [2]. Hippocrates (460–377 BC) challenged these notions, arguing epilepsy was not divine but had a natural origin and a worse prognosis in children than adults [3]. The Spartans, as described by Plutarch, even used undiluted wine to test for epilepsy in infants, believing seizures indicated weakness [2]. During the Roman era, Galen distinguished between brain-based "idiopathic" seizures and "sympathetic" ones resulting from systemic illness, a notion later reframed as "symptomatic" epilepsy [4]. In medieval times, seizures were attributed to demonic possession, and exorcism was considered a treatment. Epileptics were stigmatized and isolated, as seen in institutions like the Cloister of St. Valentine [5]. Avicenna contributed to a more nuanced understanding by categorizing epilepsies based on cerebral versus extracerebral causes [6]. The Renaissance sparked a revival in medical literature on epilepsy, as scholars began proposing scientific explanations and seizure classifications [5]. In the Enlightenment, William Cullen and Auguste Tissot further refined seizure descriptions, and William West provided the first clinical account of infantile spasms in his son [1]. A turning point came in 1909 with the formation of the International League Against Epilepsy (ILAE) in Budapest [7]. The introduction of electroencephalography (EEG) in the early 20th century revolutionized understanding, distinguishing between focal and generalized seizures based on electrical patterns [1]. By the mid-20th century, neonatal seizures were being studied using direct observation and later video-EEG, leading to classifications by Volpe and refinements by Mizrahi and Kellaway [8]. The aim is to determine the incidence, etiological causes, and risk factors associated with neonatal seizures.

Method

This prospective, hospital-based case-control study was conducted over eight months, from January 1 to August 31, 2022, at the Neonatal Care Unit (NCU) of Children Welfare Teaching Hospital (CWTH), a tertiary referral center in the Medical City of Baghdad. A total of 180 neonates were enrolled, with 90 clinically diagnosed neonatal seizure cases and 90 age-, gender-, and weight-matched neonates admitted for other reasons serving as controls. These were selected from a larger pool of 1,169 admitted neonates. Ethical approval was obtained from the relevant Medical Sciences Ethics Committee, and all ethical considerations were adhered to. Inclusion criteria encompassed neonates with clinical seizures occurring within the first 28 days of life in term infants or up to 44 weeks of postmenstrual age in preterm infants. Exclusion criteria included seizure-like activities such as jitteriness and seizures occurring beyond the neonatal period. Data collection was based on a structured questionnaire completed via direct interviews with mothers or close relatives, and supplemented by medical records. The questionnaire covered socio-demographic data, detailed pre-, peri-, and postnatal history, seizure characteristics, clinical examination, laboratory and radiological investigations, and management. Follow-up was conducted via phone calls approximately two months after discharge. Investigations included septic screening (CBC, CRP, blood and urine cultures, CSF analysis), metabolic screening (glucose, calcium, electrolytes, ammonia, lactate), liver and renal function tests, ABG, and neuroimaging (cranial ultrasound, CT, MRI). EEG and PCR for HSV were conducted when indicated. Radiological tests were used to rule out structural, infectious, and hypoxic causes. Statistical analysis was performed using SPSS® version 23.0. Continuous variables were presented as mean ± SD, while categorical data were expressed as frequencies and percentages. The Student's t-test and chi-square test were used for comparison, with a p-value ≤ 0.05 considered statistically significant.

Results

This study included a total of (180) neonatal patients, ninety of them were cases of neonatal seizures and the remaining ninety where controls. Mean age of participants (9.3 ± 9.1) days and a median of 5 days. Age groups distribution of study patients. The majority of participants were males, forming (59.4%) of total study sample (see Figure 2). No significant difference was observed between two groups regarding gender, P-value = 0.649. According to the etiology of neonatal seizure, the major causes were perinatal asphyxia, infections, and metabolic disorders with same percentage as in Table 1.

Table 1: Gender, Age Group, and Etiology of Neonatal Seizures

Category	Subcategory	Frequency (Case)	Frequency (Control)	Percentage (%)
Gender	Male			59.4%
Gender	Female			40.6%
Age Group (days)	0–7	43	58	
Age Group (days)	8–14	21	14	
Age Group (days)	15–21	9	9	

Age Group (days)	>21	17	7	
Etiology	Perinatal asphyxia	23		25.6%
Etiology	Infection - Septicemia	9		10.0%
Etiology	Infection - Meningitis with CNS malformation	7		7.8%
Etiology	Infection - Meningitis	6		6.7%
Etiology	Infection - Meningitis with ICH	1		1.1%
Etiology	Metabolic disorders - Hypocalcemia	17		18.9%
Etiology	Metabolic disorders - Hypoglycemia	3		3.3%
Etiology	Metabolic disorders - Inborn error	2		2.2%
Etiology	Metabolic disorders - Hypernatremia + Hypocalcemia	1		1.1%
Etiology	CNS malformation	10		11.1%
Etiology	Intracranial hemorrhage	2		2.2%
Etiology	Others*	3		3.3%
Etiology	Idiopathic	6		6.7%
Etiology	Total	90		100%

Regarding maternal risk factors, there were significant differences between cases and controls regarding both family histories of neurological diseases and family history of neonatal death, P-value = 0.002 and 0.036, respectively (see Table 2). No other significant differences were observed.

Table 2. Comparison Between Studied Groups Regarding Maternal Risk Factors (n=180)

Variable	Group	Case (n=90)	Control (n=90)	Total (n=180)	P-value
Maternal chronic disease	Yes	26 (47.3%)	29 (52.7%)	55 (100%)	0.627
	No	64 (51.2%)	61 (48.8%)	125 (100%)	
ANC (Antenatal Care)	Yes	21 (60.0%)	14 (40.0%)	35 (100%)	0.187
	No	69 (47.6%)	76 (52.4%)	145 (100%)	
Maternal infection	Yes	47 (51.6%)	44 (48.4%)	91 (100%)	0.655
	No	43 (48.3%)	46 (51.7%)	89 (100%)	
ROM (Rupture of Membrane)	Yes	30 (50.8%)	29 (49.2%)	59 (100%)	0.874
	No	60 (49.6%)	61 (50.4%)	121 (100%)	
Parity	Primi	24 (47.1%)	27 (52.9%)	51 (100%)	0.620
	Multi	66 (51.2%)	63 (48.8%)	129 (100%)	
Consanguinity	Yes	34 (42.5%)	46 (57.5%)	80 (100%)	0.072
	No	56 (56.0%)	44 (44.0%)	100 (100%)	
Family history of neurological disease	Yes	28 (71.8%)	11 (28.2%)	39 (100%)	0.002*
	No	62 (44.0%)	79 (56.0%)	141 (100%)	
Family history of neonatal death	Yes	12 (75.0%)	4 (25.0%)	16 (100%)	0.036*
	No	78 (47.6%)	86 (52.4%)	164 (100%)	
Mode of Delivery	NVD	36 (56.3%)	28 (43.8%)	64 (100%)	0.213
	C/S	54 (46.6%)	62 (53.4%)	116 (100%)	

Comparison between types of seizure and categories of diagnosis revealed no significant relationship with any of birth asphyxia, infection, metabolic disorders, CNS malformations, or others/idiopathic, P-value = 0.170, 0.243, 0.782, 0.842, and 0.774, respectively (see Table 3).

Table 3: Comparison between Diagnosis and Types of Seizure

Diagnosis		Types of seizure				Total	P-value
		Subtle	Clonic	Tonic	Myo-clonic		
Birth asphyxia	Yes	5 (21.7%)	9 (39.1%)	7 (30.4%)	2 (8.7%)	23 (100%)	0.170
	No	27 (40.3%)	28 (41.8%)	9 (13.4%)	3 (4.5%)	67 (100%)	
Infection	Yes	9 (39.1%)	12 (52.2%)	2 (8.7%)	-	23 (100%)	0.243
	No	23 (34.3%)	25 (37.3%)	14 (20.9%)	5 (7.5%)	67 (100%)	
Metabolic disorders	Yes	9 (39.1%)	9 (39.1%)	3 (13.0%)	2 (8.7%)	23 (100%)	0.782
	No	23 (34.3%)	28 (41.8%)	13 (19.4%)	3 (4.5%)	67 (100%)	
CNS mal-formation	Yes	4 (40.0%)	3 (30.0%)	2 (20.0%)	1 (10.0%)	10 (100%)	0.842
	No	28 (35.0%)	34 (42.5%)	14 (17.5%)	4 (5.0%)	80 (100%)	
Others	Yes	5 (45.5%)	4 (36.4%)	2 (18.2%)	-	11 (100%)	0.774
	No	27 (34.2%)	33 (41.8%)	14 (17.7%)	5 (6.3%)	79 (100%)	

Comparison between diagnosis and age at onset of seizure was performed using ANOVA test. There was significant difference among different diagnoses regarding age at onset of seizure, with ANOVA F-value = 7.894, P-value < 0.001 (see Table 4).

Table 4: Comparison of Age at Onset of Seizure among Different Diagnoses*

Group	N	Age (days) Mean ± SD	F	P-value
Birth asphyxia	23	3.2 ± 5.0	7.894	**< 0.001****
Infection	23	14.8 ± 8.6		
Metabolic disorders	23	6.7 ± 5.7		
CNS malformation	10	10.2 ± 9.8		
Others/Idiopathic	11	8.7 ± 8.7		
Total	90	8.5 ± 8.3		

Note: *ANOVA test used in this comparison; ** Significant at P ≤ 0.05

Post-hoc analysis further revealed that significant differences regarding age at onset of seizure were observed between each of birth asphyxia vs. infection (P<0.001); birth asphyxia vs. CNS malformation (P=0.012), birth asphyxia vs. others (P=0.040), infection vs. metabolic disorders (P<0.001), and infection vs. others (P=0.025), as detailed in Table 5.

Table 5: Post-Hoc Analysis of Age at Onset of Seizure among Different Diagnoses

Comparison	Mean Difference	P-value
Birth asphyxia vs. **Infection**	11.6	**< 0.001***
Birth asphyxia vs. **Metabolic disorders**	3.6	0.099
Birth asphyxia vs. **CNS malformation**	7.0	**0.012***
Birth asphyxia vs. **Others**	5.6	**0.040***
Infection vs. **Metabolic disorders**	8.0	**< 0.001***
Infection vs. **CNS malformation**	4.6	0.099
Infection vs. **Others**	6.1	**0.025***
Metabolic disorders vs. **CNS malformation**	3.5	0.211

Metabolic disorders vs. **Others**	2.0	0.457
CNS malformation vs. **Others**	1.5	0.643

Note: * Significant at P ≤ 0.05

Comparison of diagnosis and demographic characteristics was performed using chi-square test (see Table 6). There was significant relationship between diagnosis and mode of delivery, P-value = 0.039. According each variable of demographic characteristics, there was significant relation between infection and gestational age <37wk (p value = 0.003), birth asphyxia and body weight ≥2.5kg (p value = 0.036), CNS malformation and female gender (p value = 0.012), birth asphyxia and normal vaginal delivery (p value = 0.034), CNS malformation and cesarean section (p value = 0.043) (see Table 6).

Table 6: Comparison between Diagnosis and Demographic Characteristics

Variable*		**Total**	**Diagnosis**					**P-value between causes**
			Birth asphyxia	**Infection**	**Meta-bolic disorder**	**CNS mal-formation**	**Others**	
GA	<37wk	33	7 (21.2%)	10 (30%)	9 (27.2%)	5 (15.1%)	2 (6%)	0.51 ns
	≥37wk	57	16 (28%)	13 (22.8%)	14 (24.5%)	5 (8.7%)	9 (51.3%)	
P. value within cause			0.637 ns	**0.003 s**	0.97 ns	0.56 ns	0.31 ns	
Body-weight	<2.5kg	25	2 (8%)	8 (32%)	8 (32%)	5 (20%)	2 (8%)	0.082 ns
	≥2.5kg	65	21 (32.3%)	15 (23%)	15 (23%)	5 (20%)	9 (13.8)	
P. value within cause			**0.036 s**	0.55 ns	0.55 ns	0.20 ns	0.69 ns	
Gender	M	55	15 (27.2%)	16 (29%)	14 (25.4%)	2 (3.6%)	8 (14.5%)	0.072 ns
	F	35	8 (22.8%)	7 (20%)	9 (25.7%)	8 (22.8%)	3 (8.5%)	
P. value within cause			0.83 ns	0.47 ns	0.82 ns	**0.012 s**	0.61 ns	
Mode of delivery	NVD	36	14 (38.8%)	6 (16.6%)	10 (27.7%)	1 (2.7%)	5 (13.8%)	**0.039 s**
	C/S	54	9 (16.6%)	17 (31.4%)	13 (24%)	9 (16.6%)	6 (11.1%)	
P. value within cause			**0.034 s**	0.18 ns	0.88 ns	**0.043 s**	0.95 ns	

Note: * Chi square test used in comparison of Birth asphyxia, Infection and Met. Disorders.; Fisher's exact test used in comparison of CNS malformation and others; s: significant, ns: not significant

Comparison between cases and controls regarding outcome had revealed significant difference between the two groups (P-value = 0.014). Odds ratio was 3.26 (95% C.I.: 1.22 – 8.71). This indicates that cases with neonatal seizure were 3 times more likely to die than other neonates not complaining of neonatal seizure (see Table 7).

Table 7: Comparison of Outcome between Cases and Controls

Outcome	**Group**		**P-value**
	Case	**Control**	
Dead	17 (73.9%)	6 (26.1%)	**0.014***
Alive	73 (46.5%)	84 (53.5%)	
Total	90 (50.0%)	90 (50.0%)	
Odds ratio: **3.26** (95% C.I. 1.22 – 8.71)			

Note: * Significant at P < 0.05

Regarding duration of hospitalization, no significant difference was observed between cases (15.4 ± 15.8 days) and controls (12.1 ± 10.4 days), Student's t-test = 1.63, P-value = 0.106. as in Table 8.

Table 8: Comparison of Neonatal Death between Cases and Controls at Short and Long Outcome

Group	Short outcome*	Causes	Long outcome**	Causes	Total
Cases	17 (18.8%)	Birth asphyxia(6), Infections(4), Metabolic causes(4), CNS malformations(2), Kernicterus(1)	6 (8.2%)	Infection(2), CNS malformation(2), Metabolic(1), Idiopathic(1)	23 (25,5%)
Controls	6 (6.6%)	Post op TEF(2), prematurity(2), RDS+CHD(1), Sepsis(1)	4 (5.4%)	CHD(2), CNS malformation(1), Meningitis(1)	10 (11.1%)

Note: *death at hospital; **death after discharge

Discussion

Neonatal seizures remain a major global neurological concern, often reflecting serious underlying pathology and posing diagnostic challenges due to the immature neonatal central nervous system and seizure-mimicking paroxysmal movements, particularly in critically ill infants [9,10]. The accurate diagnosis is crucial yet frequently hindered by clinical ambiguity, leading to potential over- or under-diagnosis [10].

In this hospital-based case-control study, 180 neonates were assessed, revealing a neonatal seizure incidence of 7.7%, consistent with the study by Misanović et al. (7.19%) [11], slightly lower than Singh et al. (12.26%) [12], and higher than Khuntdar et al. (4.57%) [13]. This variability may reflect our tertiary hospital's referral status, receiving more complex and high-risk cases. The study population was matched for gender, gestational age, and birth weight, with males predominating (1.57:1), in line with studies by Hashish [14], and Das [15], potentially due to sex-specific vulnerability or demographic factors [16]. Seizures were more common in term neonates (≥37 weeks) and those with ≥2.5 kg birth weight, as supported by Nemati [17], Sharma [18], and Dickmark [19], likely due to increased prevalence of birth asphyxia and metabolic complications in these infants. However, other studies found higher seizure rates among preterm neonates [20,21]. In this study, birth asphyxia, infection, and metabolic disorders each accounted for 25.6% of seizure cases. Birth asphyxia, a consistent finding in numerous studies [14,22], was often linked to home delivery and inadequate antenatal care, echoing findings by Aslam [23]. Infections, particularly meningitis (15.6%), were notably higher in neonates delivered by cesarean section, likely due to emergency procedures in high-risk cases, similar to Khalessi [24]. The rate of seizure due to infection has decreased compared to earlier local data (38.6%) [25], suggesting improved infection control. Metabolic disorders contributed equally (25.6%), mostly hypocalcemia (18.9%), similar to Agarwalla [26] and Thomas [27]. Hypoglycemia was less frequent (3.3%), possibly due to better monitoring and feeding practices [25,28]. CNS malformations were significant (18.9%), attributed to the hospital's neuro-specialty referral role, with intracranial hemorrhage, kernicterus, and hypothyroidism contributing less frequently [9,13]. Idiopathic seizures accounted for 6.7%, consistent with other regional study [29]. A significant association was observed between neonatal seizures and a family history of seizures or neonatal death [17,30], reinforcing the likely genetic predisposition [31], though genetic testing was unavailable locally. Seizures were more common in neonates born to mothers with poor antenatal care, prolonged PROM, and NVD, although these did not reach statistical significance. A notable finding was the association between C/S and CNS malformations [32], and between birth asphyxia and vaginal delivery [33]. Clonic seizures were most frequent (41%), followed by subtle (35.5%) and tonic (18%), consistent with Sharma [18] and Mishra [34], though seizure type showed no correlation with etiology [20]. The timing of seizure onset correlated with etiology: birth asphyxia appeared earlier (mean 3.2 days), infections later (14.8 days), and metabolic disorders in between (6.7 days), reflecting typical pathophysiological timelines [35]. Mortality was significantly higher in the seizure group (18.8%) vs. controls (6.6%), with an odds ratio of 3.2, indicating a threefold increased risk of death, aligning with findings by Kumari [20] and Heljic [36]. Follow-up revealed a substantial proportion of seizure survivors (22%) experienced further complications, highlighting the importance of early identification and comprehensive management to improve long-term outcomes [13].

Conclusion

Neonatal seizures are usually caused by birth hypoxia, infection, or metabolic abnormalities. Epilepsy onset linked to pathology. Clonic seizure is the most prevalent, and no newborn seizure type can indicate the aetiology. Neonates with seizures died three times more than those without. Positive family history of seizure or neurological illness increases newborn seizure risk. Delivery type is strongly linked to seizure aetiology.

References

[1] Patel P, Moshé SL. The evolution of the concepts of seizures and epilepsy: What's in a name? Epilepsia Open. 2020;5(1):22–35. doi:10.1002/epi4.12375.

[2] Panteliadis C. Historical sources on epilepsy surgery: From antiquity through the end 20th century. Open Neurol J. 2021;15(1):52–58. doi:10.2174/1874205X02115010052.

[3] Magiorkinis E, Sidiropoulou K, Diamantis A. Hallmarks in the history of epilepsy: from antiquity till the twentieth century. In: Novel Aspects on Epilepsy. 2011 Oct 12:131–156.

[4] Wolf P. History of epilepsy: nosological concepts and classification. Epileptic Disord. 2014;16(3):261–269. doi:10.1684/epd.2014.0676.

[5] Diamantis A, Sidiropoulou K, Magiorkinis E. Epilepsy during the Middle Ages, the Renaissance and the Enlightenment. J Neurol. 2010;257(5):691–698. doi:10.1007/s00415-009-5433-7.

[6] Zali F, Bahrami M, Akhtari E. Uterine epilepsy: a historical report from Avicenna's point of view. Neurol Sci. 2020;41(1):229–232. doi:10.1007/s10072-019-04019-7.

[7] Feindel W, Leblanc R, de Almeida AN. Epilepsy surgery: historical highlights 1909–2009. Epilepsia. 2009;50 Suppl 3:131–151. doi:10.1111/j.1528-1167.2009.02043.x.

[8] Pressler RM, Cilio MR, Mizrahi EM, Moshé SL, Nunes ML, Plouin P, et al. The ILAE classification of seizures and the epilepsies: Modification for seizures in the neonate. Epilepsia. 2021;62(3):615–628. doi:10.1111/epi.16815.

[9] Kancherla P, Harsha, Gowtham, Basha DA, Chandran, Golluri R. Clinical profile and short-term outcome of Neonatal Seizures in a tertiary care hospital, Kuppam – A retrospective study. J Evid Based Med Healthc. 2021;8(27):2381–2386.

[10] Hart AR, Pilling EL, Alix JJ. Neonatal seizures—part 1: Not everything that jerks, stiffens and shakes is a fit. Arch Dis Child Educ Pract Ed. 2015;100(4):170–175. doi:10.1136/archdischild-2014-306385.

[11] Misanović V, Hodžić E, Terzić S, Vukas-Salihbegović E, Ključić A. Neonatal seizures: Etiology, treatment and prognosis. Sanamed. 2022;17(2):91–97.

[12] Singh SD, Shrestha RP, Shrestha A. Etiological Profile of Neonatal Seizures and Prognostic Factors for Adverse Outcome: A Single Center Prospective Study. J Lumbini Med Coll. 2018;6(2):1–5.

[13] Khuntdar BK, Mondal S, Naik S, Mohanta MP. Evaluation of Adverse Outcome Predictors in Neonatal Seizure: A Longitudinal Study from a Tertiary Centre of Eastern India. Indian J Neonatal Med Res. 2023;11(1):1–5.

[14] Hashish ME, Darwish A, Bassiouny MR. Predictors of Adverse Outcomes in Neonates with Seizures. Am J Pediatr. 2022;8(3):158–167.

[15] Das D, Debbarma SK. A study on clinico-biochemical profile of neonatal seizure. J Neurol Res. 2016;6(5-6):95–101.

[16] Akman O, Moshé SL, Galanopoulou AS. Sex-specific consequences of early life seizures. Neurobiol Dis. 2014;72:153–166. doi:10.1016/j.nbd.2014.08.013.

[17] Nemati H, Karimzadeh P, Fallahi M. Causes and Factors Associated with Neonatal Seizure and its Short-term Outcome: A Retrospective Prognostic Cohort Study. Iran J Child Neurol. 2018;12(3):59–68.

[18] Sharma J, Nair B, Chaudhary S. Clinicoetiological profile of neonatal seizure in a newborn care unit of a tertiary care teaching hospital in Northern India. J Clin Neonatol. 2020;9(1):27–31.

[19] Dickmark M, Ågren J, Hellström-Westas L, Jonsson M. Risk factors for seizures in the vigorous term neonate: A population-based register study of singleton births in Sweden. PLoS One. 2022;17(2):e0264117. doi:10.1371/journal.pone.0264117.

[20] Kumari K, Raman GV. Etiological Factors Of Neonatal Seizures And Their Outcome At A Tertiary Care Hospital. Int J Sci Res. 2020;9(3):69–71.

[21] Golwara S, Ahmed MN, Kumar D, Agrawal R, Singh BK. Clinical And Biochemical Characteristics of Newborn Seizures Admitted in NICU of A Tertiary Care Hospital in Bihar. Int J Pharm Clin Res. 2021;13(4):226–230.

[22] Patil R, Patel B. Correlation of various causes of neonatal seizures with different patterns of EEG waveforms. Glob J Res Anal. 2016;5(1):45–47.

[23] Aslam HM, Saleem S, Afzal R, Iqbal U, Saleem SM, Shaikh MWA, et al. Risk factors of birth asphyxia. Ital J Pediatr. 2014;40(1):94. doi:10.1186/s13052-014-0094-2.

[24] Khalessi N, Afsharkhas L. Neonatal meningitis: risk factors, causes, and neurologic complications. Iran J Child Neurol. 2014;8(4):46–50.

[25] AL-Naddawi MN, Hameed NN, Kadum MJ. Clinical types and possible etiologies of neonatal seizures: A hospital based study. J Fac Med Baghdad. 2011;53(1):1–5.

[26] Agarwalla S, Sahu P. Neonatal Seizure - Etiological & Clinical differentiation. IP Int J Med Paediatr Oncol. 2020;6(3):121–124.

[27] Thomas TC, Smith JM, White PC, Adhikari S. Transient neonatal hypocalcemia: presentation and outcomes. Pediatrics. 2012;129(6):e1461–e1467. doi:10.1542/peds.2011-2792.
[28] De Angelis LC, Brigati G, Polleri G, Malova M, Parodi A, Minghetti D, et al. Neonatal hypoglycemia and brain vulnerability. Front Endocrinol (Lausanne). 2021;12:634305. doi:10.3389/fendo.2021.634305.
[29] Almonaem E, Dabour AS, Elawady MA, Haie O. Clinco-etiological Profile and Predictors of Outcome of Neonatal Seizures: A Prospective Observational Study from Egypt. Iran J Neonatol. 2022;13(2):22–31.
[30] Panjan M, Paro-Panjan D, Salamon AS. Genetic-cellular epilepsy: Clues to diagnosing newborns with neonatal seizures. Seizure. 2021;92:68–75. doi:10.1016/j.seizure.2021.08.005.
[31] Kaur S, Pappas K. Genetic etiologies of neonatal seizures. Neoreviews. 2020;21(10):e663–e672. doi:10.1542/neo.21-10-e663.
[32] Friedler JM, Mazor M, Shoham-Vardi I, Bashiri A. Characteristics of pregnancy and delivery of fetuses affected by either central nervous system malformations or chromosomal abnormalities. Harefuah. 2011;150(11):829–832.
[33] Kiyani AN, Khushdil A, Ehsan A. Perinatal factors leading to birth asphyxia among term newborns in a tertiary care hospital. Iran J Pediatr. 2014;24(5):637–642.
[34] Steer P, Scherjon S. Congenital cerebral palsy: an update. BJOG. 2016;123(5):e135–e137. doi:10.1111/1471-0528.13719.
[35] Nair MKC, Nair G. Baby-Friendly Hospital Initiative: Relevance to Neonatal Health and Neonatal Seizures. Indian Pediatr. 2017;54(7):559–563.
[36] Volpe JJ. Neonatal seizures: current concepts and revised classification. Pediatrics. 2019;144(5):e20192186. doi:10.1542/peds.2019-2186.