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Original Research Article

Events of children responsible for the development of epilepsy in patients with cerebral palsy

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ABSTRACT

Background: Cerebral palsy (CP) is characterized by abnormal muscle tone, 'posture, and movement', thereby limiting the activity of the affected person. Epilepsy is said to occur in 15-90% of children with CP. The aim of this study was to evaluate the events of children responsible for the development of epilepsy considering natal, postnatal characteristics and associated impairment in patients with CP.

Methods: This was a case-control study and was conducted in the Department of Pediatric Neurology at the National Institute of Neurosciences and Hospital, Dhaka, Bangladesh during the period from January 2020 to December 2020. In total 150 children with cerebral palsy were enrolled in this study who were divided into two groups. In the 'CP with epilepsy' group, there were 50 patients, and in the CP without epilepsy group taken as a case group, there were 100 age-sex matched CP patients without epilepsy patients were taken as a control group. Informed written consent was taken from all the parents. Along with baseline characteristics, data regarding natal, postnatal events, general clinical findings, psychological assessment, computed tomography (CT) scan findings of the head, and electroencephalogram (EEG) findings in epilepsy cases were recorded in a predesigned questionnaire and analyzed.

Results: In distributing the CP patients' comparison regarding postnatal complications neonatal seizures were a strong predictor for epilepsy ($p \leq 0.001$). 1st seizure during 1st year of life and family history of epilepsy was also a significant event of epilepsy ($p \leq 0.001$). In the case of microcephaly, no significant difference was observed between groups ($p=0.278$). A significant difference was observed when the distribution of severity of intellectual disability of CP patients was compared between groups ($p<0.001$). Abnormal CT scan findings of the head were significantly higher in CP with epilepsy 42 (84.0%) than in CP without epilepsy 46 (46.0%) ($p \leq 0.001$). The total mean age at the onset of epilepsy was 13.58±14.47 months. Epilepsy was most common in spastic quadriplegic CP (54%). 38.0% had focal epileptiform activity on EEG. Clinically focal epilepsy was found in 36.0% of cases.

Conclusions: This study demonstrates the presence of a history of neonatal seizure, 1st seizure during 1st year of life, a family history of epilepsy, CT scan abnormalities, severe intellectual disability, and spastic quadriplegic CP were the events responsible for the development of epilepsy in children with CP.

Keywords: Cerebral palsy, Epilepsy, Children, Seizure, Epileptiform activity

INTRODUCTION

Cerebral palsy (CP) is a common physical disability in childhood.[1] It describes a group of permanent disorders of the development of movement and posture, causing activity limitations that are attributed to non-progressive disturbances that occur in the developing fetal or infant brain. Different kinds of maternal antenatal, prenatal, natal, and postnatal causes are responsible for the

development of cerebral palsy. In Nigeria, a hospital-based study was done where they reported that birth asphyxia, bilirubin encephalopathy, and post-infectious brain damage were the main causes of CP.[2] The fact that the pattern of association of these maternal and fetal factors with epilepsy, mental deficiency, and behavior disorders of childhood was similar to that found concerning CP indicated that those conditions should be included in the postulated continuum.[3] The clinical circumstances that led to each maternal admission and ultimately to each preterm delivery were operationally defined by using both data from the maternal interview and data abstracted from the medical record.[4] Problems in brain function that occur during fetal brain development or within the first two years of life are the causes of these movement disorders.[5] It can result in spasticity, dystonia, muscle contractures, weakness, and difficulty in coordination which ultimately affects the ability to control movements.[6] Impairment of vision, hearing, sensation, perception, cognition, communication, and behaviour, epilepsy often accompanies CP.[7] The occurrence of cerebral palsy is approximately 2 per 1000 live births. Globally, an estimated 17 million people are living with CP.[8] The prevalence of CP is 3.4 per 1000 children in rural Bangladesh. There are an estimated 2, 33,514 children with CP in Bangladesh.[9,10] For these reasons, in recent years, many studies investigated the prevalence, characteristics, and prognosis of epilepsy in patients with CP but few studies investigated the risk factors considering prenatal, perinatal, and natal history for the development of epilepsy in these patients.[11] The most commonly reported risk factor for later epilepsy was found to be a history of neonatal seizures.[12] However additional data regarding birth history parameters that could increase the risk for the development of epilepsy in these children were less consistent.[13]

Aim of the study

The aim of this study was to evaluate the events of children responsible for the development of epilepsy considering natal, postnatal characteristics and associated impairment in patients with CP.

METHODS

This prospective observational case-control study was conducted in the Department of Pediatric Neurology, National Institute of Neurosciences and Hospital, Dhaka, Bangladesh during the period from January 2020 to December 2020. In total 150 children with a diagnosis of CP were enrolled in this study as study subjects. The study was approved by the ethical committee of the mentioned hospital. Proper written consent was taken from their parents before data collection. As per the inclusion criteria of this study, patients with cerebral palsy with epilepsy between the ages of 18 months to 12 years were included. On the other hand, according to the exclusion criteria of this study, patients with nonspecific motor delay, provoked seizure, suspected cases of neurometabolic disease and neurodegenerative disorder were excluded. All the demographic and clinical data of the participants were recorded. A predesigned questionnaire was used in data collection. Every morning first case of CP with epilepsy was enrolled as a case group. Age (±2 months) and sex-matched next two cases of CP without epilepsy were enrolled as the control group sequentially. Diagnosis of epilepsy was based on history from a reliable eyewitness or video documentation if available and EEG was done in selected cases. All the diagnoses were made by the pediatric neurologist(s) based on the study definition. The children's intelligence was evaluated based on age-appropriate psychometric tests by psychologist. The standardized, Reynell-Zinkin developmental scale (RZS) was applied to evaluate cognitive development in non-verbal with visually impaired children aged 5 years and below.[14] Wechsler preschool and primary scale of intelligence III (WPPSI-III) junior intelligence was designed for children ages 2 years 6 months to 2 years 11 months and senior for children ages 4 years to 7 years 7 months.[15] Wechsler intelligence scale for children-IV (WISC-IV) was used to assess the intelligence quotient (IQ) for children above 6 years of age.[16] Children with a total intelligence score (IQ) level <70 were diagnosed as intellectual disability. Children with an intelligence score of <35 were diagnosed as having a severe intellectual disability, 49–35 were diagnosed as moderate intellectual disability, and 69–50 were diagnosed as mild intellectual disability.[17] CT scan of the head was done at the neuroradiology department of the National Institute of Neurosciences and Hospital (NINS&H) by Hitachi Eclos (Japan) with 16-mm axial slices, skilled professionals and expert opinions were taken from neuroradiologists and carefully reviewed by the pediatric neurologist. All data were processed, analyzed and disseminated by using Microsoft excel and statistical package for the social sciences (SPSS) version 23 program as per necessity.

RESULTS

In this study, the mean±SD age of the patients of 'CP with epilepsy' and 'CP without epilepsy' were 48.7±30.3 and 49.0±30.5 months respectively. In those groups male babies were 62% and 59% respectively (Table 1). In the CP with epilepsy group, among three-fourths of the cases (72%) gestational age was found at 38-41 weeks which age range was found in another group among 56% of cases. In both groups, normal vaginal delivery was performed in the majority of the cases (58% and 53% respectively). In the majority of the cases in CP with epilepsy group, the delivery place was a home, but in another group, it was a hospital. In both the groups, among the majority of the cases, the birth weight range was found 2500-4000 g. As the complication during labour, in CP with epilepsy group, prolonged labour was found in 30% of cases and obstructed labour was found in 34% of cases. On the other hand, those complications were found in 27% and 23% of cases respectively in the CP without epilepsy group. As the postnatal complication perinatal asphyxia was found in the majority of the cases in both groups but neonatal seizure

was significantly higher in CP with epilepsy 24 (48.0%) than CP without epilepsy 12 (12.0%) (p≤0.001) when compared between two groups (Table 2). First seizure during 1st year of life was observed significantly higher in CP with epilepsy 35 (70%) than CP without epilepsy 23 (23%) (p≤0.001). Family history of epilepsy in CP with epilepsy 10 (20.0%) and CP without epilepsy 2 (2.0%), their differences were statistically significant (p≤0.001). Microcephaly was found in CP with epilepsy 44 (88.0%) and CP without epilepsy 81 (81.0%) but no significant difference was observed between groups (p=0.278). A significant difference was observed when the distribution of severity of intellectual disability of CP patients was compared between groups (p<0.001). Severe disability was higher in CP with epilepsy 30 (60.0%) and moderate disability was higher in CP without epilepsy 51 (51.0%). The distribution of types of CP patients was significantly different when compared between groups (p≤0.001). Spastic quadriplegia was higher in CP with epilepsy 27 (54.0%) and spastic diplegia was higher in CP without epilepsy 36 (36.0%) (Table 3). In distributing the CP patients according to CT scan findings of the head, we found, abnormal findings, non-specific cerebral atrophy, and encephalomalacia as some more frequent findings in both the groups and also found a significant correlation between the groups (p<0.001) (Table 4). In this study distribution of CP patients with epilepsy by EEG findings, it was found that 28 patients (56.0%) had focal origin (focal epileptiform activity 38.0%, multifocal epileptiform activity 14.0%, focal epileptiform activity with secondary generalization 4.0%), 9 patients (18.0%) had epileptic encephalopathy and 2 patients (4.0%) had generalized epileptiform activity (Table 5). In this study, 28 patients (56.0%) had age at onset of epileptic seizure less than 12 months (Figure 1). Clinically focal epilepsy was found in 18 patients (36.0%), generalized epilepsy in 16 patients (32.0%), unknown epilepsy in 2 patients (4.0%), and syndromic epilepsy in 14 patients (28.0%) (Figure 2). Focal epilepsy is more common in spastic hemiplegic CP. Generalized and syndromic epilepsy is a more common spastic quadriplegic CP (Table 6). The mean age at onset of epilepsy was 8.30±3.34 months. The onset of epileptic seizure was earlier in spastic quadriplegic patients and later in spastic hemiplegic patients (Figure 3).

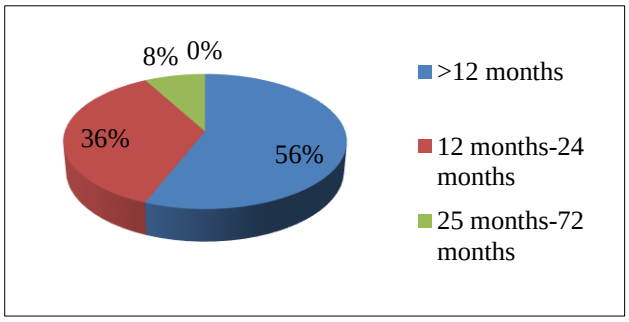

Figure 1: Distribution of patients according to age at onset of epileptic seizure (n=50).

Table 1: Distribution of the CP patients according to basic demographic characteristics (n=150).

Characteristics	CP with epilepsy (n=50)	CP without epilepsy (n=100)	P value
Age (months)			
<24	8 (16.0)	12 (12.0)	0.802
24–48	18 (36.0)	42 (42.0)	
48–72	9 (18.0)	18 (18.0)	
72–144	15 (30.0)	28 (28.0)	
Mean±SD	48.7±30.3	49.0±30.5	0.958
Sex			
Male	31 (62.0)	59 (59.0)	0.724
Female	19 (38.0)	41 (41.0)	

Table 2: Distribution of the CP patients according to natal events and postnatal complication (n=150).

Parameters	CP with epilepsy (n=50) (%)	CP without epilepsy (n=100) (%)	P value
Type of natal events			
Gestational age (weeks)			
<37 (preterm)	11 (22.0)	40 (40.0)	0.088
38-41 (term)	36 (72.0)	56 (56.0)	
>42 (post term)	3 (6.0)	4 (4.0)	
Mode of delivery			
NVD	29 (58.0)	53 (53.0)	0.204
LUCS	21 (42.0)	47 (47.0)	
Place of delivery			
Home	26 (52.0)	40 (40.0)	0.064
Hospital	24 (48.0)	60 (60.0)	
Birth weight (g)			
VLBW (<1500)	2 (4.0)	6 (6.0)	0.17
LBW (1500-2499)	9 (18.0)	34 (34.0)	
NBW (2500-4000)	36 (72.0)	54 (54.0)	
Macrosomia (>4000)	3 (6.0)	6 (6.0)	

Continued.

Parameters	CP with epilepsy (n=50) (%)	CP without epilepsy (n=100) (%)	P value
Complication during labour			
Prolonged labour	15 (30.0)	27 (27.0)	0.216
Obstructed labour	17 (34.0)	23 (23.0)	
History of postnatal complication			
Perinatal asphyxia	44 (88.0)	86 (86.0)	0.734
Neonatal seizure	24 (48.0)	12 (12.0)	<0.001
Neonatal jaundice	12 (24.0)	32 (32.0)	0.31

Table 3: Clinical features of CP patients with epilepsy and CP patients without epilepsy (n=150).

Clinical features	CP with epilepsy (n=50) (%)	CP without epilepsy (n=100) (%)	P value
1st seizure during 1st year of life			
Yes	35 (70.0)	23 (23.0)	<0.001
No	15 (30.0)	77 (77.0)	
Family history of epilepsy			
Yes	10 (20.0)	2 (2.0)	<0.001
No	40 (80.0)	98 (98.0)	
OFC			
Normal	6 (12.0)	19 (19.0)	0.278
Microcephaly	44 (88.0)	81 (81.0)	
Intelligence			
Normal	1 (2.0)	3 (3.0)	<0.001
Mild disability	7 (14.0)	29 (29.0)	
Moderate disability	12 (24.0)	51 (51.0)	
Severe disability	30 (60.0)	17 (17.0)	
Type of CP			
Spastic hemiplegia	8 (16.0)	21 (21.0)	<0.001
Spastic diplegia	6 (12.0)	36 (36.0)	
Spastic quadriplegia	27 (54.0)	15 (15.0)	
Dyskinetic	3 (6.0)	11 (11.0)	
Mixed	6 (12.0)	17 (17.0)	

Table 4: Distribution of the CP patients according to CT scan findings of head (n=150).

CT scan of the head	CP with epilepsy (n=50) (%)	CP without epilepsy (n=100) (%)	P value
Abnormal findings	42 (84.0)	46 (46.0)	<0.001
Non-specific cerebral atrophy	14 (28.0)	13 (13.0)	
Encephalomalacia	10 (20.0)	09 (9.0)	
Hydrocephalus	4 (8.0)	7 (7.0)	
Calcification	0 (0.0)	1 (1.0)	
Infarction	5 (10.0)	8 (8.0)	
Cystic lesion	3 (6.0)	4 (4.0)	
Cerebral malformation	2 (4.0)	2 (2.0)	
Periventricular hypodensity	4 (8.0)	2 (2.0)	

Table 5: EEG findings of CP patients with epilepsy (n=50).

EEG findings	Frequency (N)	Percentage (%)
Normal findings	4	8
Generalized epileptiform activity	2	4
Focal origin		
Focal epileptiform activity	19	38

Continued.

EEG findings	Frequency (N)	Percentage (%)
Multifocal epileptiform activity	7	14
Focal epileptiform activity with secondary generation	2	4
Epileptic encephalopathy	9	18
Hypoarrythmia	4	8
Burst suppression	2	4
Electrical status epilepticus	1	2

Table 6: Epilepsy type (clinical) in different types of cerebral palsy (n=50).

Clinical epilepsy type	Spastic (%) Hemiplegia	Spastic (%) Diplegia	Spastic (%) Quadriplegia	Dyskinetic (%)	Mixed (%)
Focal	7 (87.5)	1 (16.7)	6 (22.2)	2 (66.7)	2 (33.3)
Generalized	0 (0.0)	5 (83.3)	7 (25.9)	1 (33.3)	3 (50.0)
Unknown	0 (0.0)	0 (0.0)	2 (7.4)	0 (0.0)	0 (0.0)
Combined generalized and focal	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)
West syndrome	0 (0.0)	0 (0.0)	8 (29.6)	0 (0.0)	1 (6.7)
Lennox Gastaut syndrome	1 (12.5)	0 (0.0)	4 (14.8)	0 (0.0)	1 (16.7)

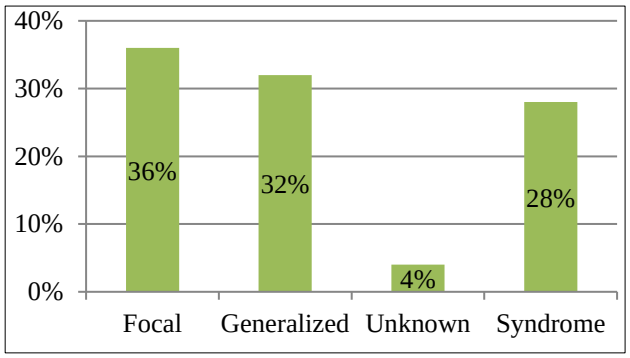

Figure 2: Clinical epilepsy type of CP patients (n=50).

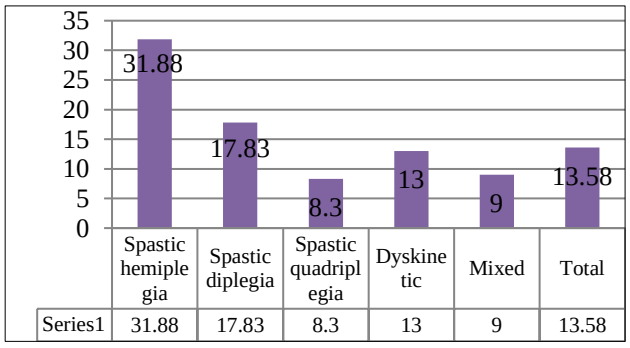

Figure 3: Mean age (in months) at onset of epileptic seizure in children with different types of cerebral palsy (n=50).

DISCUSSION

Children aged between 18 months to 12 years were included in our study and the age range between two to less than four years was commonest in both CP with epilepsy group 18 (36.0%) and CP without epilepsy group 42 (42.0%). In this study 90 patients (60%) were male. A similar observation was found by Singhi et al.[18] In their study, they found 65 patients (62%) out of 105 with cerebral palsy who were male. Male cases were more, 31 (62.0%) in CP with epilepsy and 59 (59.0%) in CP without epilepsy. There was no significant difference in terms of age and sex between CP patients with epilepsy and CP patients without epilepsy. Karatoprak et al also did not find any relation between age and risk for epilepsy development.[19] The results from various studies determining the relation between gestational age, mode of delivery, place of delivery, birth weight, complication during labour, and epilepsy development in CP patients were conflicting. Mert et al found that both prematurity and low birth weight were not related to epilepsy development.[20] Kulak et al did not find any relation between gestational age and the risk of epilepsy development.[21] However, they determined an increased risk of epilepsy in patients with low birth weight. On the other hand, Zelnik et al demonstrated that epilepsy was more frequent in term infants than in premature infants whereas they found no relation between birth weight, mode of delivery, and risk of epilepsy development.[22] Gruraj et al also reported that term delivery had an increased association with epilepsy development.[12] Sellier et al determined the association between epilepsy development and term and ≥2500 g infants in 17 European registers.[23] Karatoprak et al did not find any relation between these labour problems and the risk for epilepsy development.[19] In our study regarding the gestational age, mode of delivery, place of delivery, birth weight, and complication during labour, when compared we also did not find any significant difference between groups (p=0.088, 0.204, 0.064, 0.170, 0.216). As per the analysis of postnatal events, previous studies reported that a history of neonatal seizure in patients with cerebral palsy is a risk factor for epilepsy development.[21] Bruck et al reported that 30 out of 62 (48.4%) children with a history of neonatal seizure subsequently developed epilepsy.[24] Similar to the literature, in our study during the analysis of postnatal events neonatal seizure was significantly higher in CP with epilepsy 24 out of 50 (48.0%) than CP without epilepsy (12.0%) (p≤0.001). In contrast to our finding Kulak et al

and Kwong et al noted neonatal seizure in 17% and 19% of children with CP and epilepsy respectively.[21,25] In our study, 1st seizures during 1st year of life were significantly higher in CP with epilepsy 35 (70%) than in CP without epilepsy 23 (23%) (p≤0.001) which following Zafeiriou et al they found that first seizures occurred during the first year of life in 69.7%.[26] In our study, a family history of epilepsy was significantly higher in CP with epilepsy 10 (20.0%) than CP without epilepsy 2 (2.0%) (p≤0.001). Kulak et al and Bruck et al found 10.9%, and 29% of epileptic children with cerebral palsy had a family history of epilepsy.[21,24] In the current study, we did not find an association between microcephaly and risk for epilepsy development between groups (p=0.278). Zelnik et al also found that the ratio of microcephaly did not differ among the CP with epilepsy and CP without epilepsy patients.[22] We demonstrated a higher proportion of CT abnormalities in children with CP with epilepsy in 42 patients (84.0%) compared with the CP without epilepsy in 49 patients (49.0%) and their differences were statistically significant (p≤0.001). Kulak et al also found CT abnormalities in CP with epilepsy was 68 patients (82.9%) compared with the CP without epilepsy in 56 patients (48.2%).[10] Regarding the abnormal findings, cerebral atrophy was most often associated with epileptics, a finding consistent with data found in the literature.[12,21,22] In the present study, we found that severe intellectual disability was significantly higher in CP with epilepsy in 30 patients (60.0%) than in CP without epilepsy in 17 patients (17.0%) (p≤0.001). Karatoprak et al also found severe intellectual disability in CP with epilepsy (46%) and CP without epilepsy (12.0%) respectively.[19] In contrast to our study El-Tallawy et al observed no relationship between total IQ and epilepsy development in children with epilepsy than in those without epilepsy (84.6% versus 66.7%).[11] In this current study, the distribution of types of CP patients was significantly different when compared between groups (p≤0.001). Spastic quadriplegia was higher in CP with epilepsy 27 (54.0%) and Spastic diplegia was higher in CP without epilepsy 36 (36.0%). Karatoprak et al also found in CP with epilepsy spastic quadriplegia was 66.1% and in CP without epilepsy spastic diplegia was 40.6%.[19] In contrast, Singhi et al reported that the rate of epilepsy was the highest in spastic hemiplegic patients (65.9%).[27] In our study, it was found that 28 patients (56.0%) had age at onset of epileptic seizure less than 12 months. Zaferiou et al also found that 69% of patients with CP had their first epileptic attack before they were a year old.[26] This indicates the severity of the underlying brain injury. In our study, we found the mean age of onset of epilepsy was 31.88±28.27 months in spastic hemiplegia patients, 17.83±11.03 months in spastic diplegia patients, and 8.30±3.34 months in spastic quadriplegia patients respectively. Similar to our study Carlsson et al reported that the mean age of epilepsy onset was 2.5 years in hemiplegic cerebral palsy, 12 months in diplegic cerebral palsy, and 6 months in quadriplegic cerebral palsy.[28] In this current study, we found that the total mean age at onset of epilepsy was 13.58±14.47 months. These findings were consistent with the previous study by Bruck et al they found the average age at onset of epilepsy was 12.59 months.[24] In contrast to our study, Delgado et al found mean age at the onset of epilepsy is 2.5 years (range, 1 month to 11 years).[29] EEG is essential in the work-up of children with CP and suspected seizures. It can lend support to the diagnosis of epilepsy and assist in seizure/epilepsy classification to better guide the choice of antiseizure drugs. In our study regarding EEG findings, we found epilepsy of focal origin was more common in 28 patients (54.0%) in CP patients with epilepsy. Similar to our study focal epileptiform activity was the common EEG findings observed by Şenbil et al (48.39%).[30] In contrast to our study, generalized epileptiform abnormality was detected in a large proportion of patients with cerebral palsy and epilepsy 50 patients (41.4%) by Hanci et al.[31] In our study clinical focal epilepsy (18 patients 36.0%) was more common in CP. Similarly, focal epilepsy was more commonly observed by Gururaj et al (39.3%), Kwong et al (37.5%), Aksu et al (93.1%), Delgado et al (71%), in CP with epilepsy.[12,14,25,32] In contrast to our study, Hadjipanayis et al reported generalized epilepsy (36.8%) followed by focal epilepsy (33%) in cerebral palsy with epilepsy.[33] Classification of the type of epilepsy is difficult in children with CP for the following reasons. First, partial seizure onset that rapidly becomes generalized may not be witnessed or reported reliably. Second, impairment of consciousness during an episode may be impossible to clarify in patients with multiple handicaps. Third, the differentiation between myoclonic, tonic, and atonic seizures occasionally could be extremely difficult without ictal EEG or video EEG.

In this current study we found focal epilepsy was more common in the hemiplegic variety of CP and epilepsy was less common in dyskinetic CP. Other studies have found a similar high incidence of focal epilepsy in this variety of CP ranging from 69 to 73%.[34] In the dyskinetic type of CP, epilepsy is believed to be uncommon.[35]

Limitations

This was a single-centered study with small-sized samples. Moreover, the study was conducted in a tertiary care hospital or referral center. Although they came from different tiers of society and districts of Bangladesh, they were not representative of all children with CP in this country.

CONCLUSION

This study demonstrates the presence of a history of neonatal seizure, 1st seizure during 1st year of life, family history of epilepsy, CT scan abnormalities, severe intellectual disability, and spastic quadriplegic CP were the events responsible for the development of epilepsy in children with cerebral palsy. To get more specific results, we would like to recommend conducting similar studies in several places with larger-sized samples.

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Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Donald KA, Kakooza AM, Wammanda RD, Mallewa M, Samia P, Babakir H, et al. Pediatric cerebral palsy in Africa: where are we? J Child Neurol. 2015;30(8):963-71.
2. Bakare MO, Bello-Mojeed MA, Munir KM, Ogun OC, Eaton J. Neurodevelopmental delay among children under the age of three years at immunization clinics in Lagos State, Nigeria– Preliminary report. Sci Rep. 2016;6:25175.
3. Lilienfeld AM, Pasamanick B. The association of maternal and fetal factors with the development of cerebral palsy and epilepsy. Am J Obstet Gynecol. 1955;70(1):93-101.
4. McElrath TF, Hecht JL, Dammann O, Boggess K, Onderdonk A, Markenson G, et al; ELGAN Study Investigators. Pregnancy disorders that lead to delivery before the 28th week of gestation: an epidemiologic approach to classification. Am J Epidemiol. 2008;168(9):980-9.
5. Bax M, Goldstein M, Rosenbaum P, Leviton A, Paneth N, Dan B, et al. Proposed definition and classification of cerebral palsy, 2005. Dev Med Child Neurol. 2005;47(8):571-6.
6. Gormley ME Jr. Treatment of neuromuscular and musculoskeletal problems in cerebral palsy. Pediatr Rehabil. 2001;4(1):5-16.
7. Rosenbaum P, Paneth N, Leviton A, Goldstein M, Martin B. A report: the definition and classification of cerebral palsy. Dev Med Child Neurol. 2007;49:8-14.
8. Graham HK, Rosenbaum P, Paneth N. Cerebral palsy. Nat Rev Dis Primers. 2016;2:15082.
9. Khandaker G, Muhit M, Karim T, Smithers-Sheedy H, Novak I, Jones C, et al. Epidemiology of cerebral palsy in Bangladesh: a population-based surveillance study. Dev Med Child Neurol. 2019;61(5):601-9.
10. Khandaker G, Smithers-Sheedy H, Islam J, Alam M, Jung J, Novak I, et al. Bangladesh Cerebral Palsy Register (BCPR): a pilot study to develop a national cerebral palsy (CP) register with surveillance of children for CP. BMC Neurol. 2015;15(1):173.
11. El-Tallawy HN, Farghaly WM, Shehata GA, Badry R, Rageh TA. Epileptic and cognitive changes in children with cerebral palsy: an Egyptian study. Neuropsych Dis Treat. 2014;10:971.
12. Gururaj AK, Sztriha L, Bener A, Dawodu A, Eapen V. Epilepsy in children with cerebral palsy. Seizure. 2003;12:110-4.
13. Sun Y, Vestergaard M, Pedersen CB, Christensen J, Olsen J. Apgar scores and long-term risk of epilepsy. Epidemiology. 2006;17:296-301.
14. Reynell J. The Reynell-Zinkin scales: Developmental scales for young visually handicapped children. Windsor. 1979.
15. Wechsler D. Wechsler Preschool and Primary Scale of Intelligence–third Edition (WPPSI-III). San Antonio, TX: The Psychological Corporation, 2002.
16. Wechsler D. Wechsler intelligence scale for children–Fourth Edition (WISC-IV). San Antonio, TX: The Psychological Corporation. 2003.
17. Cooper J. Diagnostic and statistical manual of mental disorders (4th edn, text revision) (DSM–IV–TR) Washington, DC: American Psychiatric Association. Am J Psychiatry. 2001;179(1):85.
18. Singhi P, Jagirdar S, Khandelwal N, Malhi P. Epilepsy in children with cerebral palsy. J Child Neurol. 2003;18(3):174-9.
19. Karatoprak E, Sözen G, Saltık S. Risk factors associated with epilepsy development in children with cerebral palsy. Child's Nervous System. 2019;1-7.
20. Mert GG, Incecik F, Altunbasak S, Herguner O, Mert MK, Kiris N, et al. Factors affecting epilepsy development and epilepsy prognosis in cerebral palsy. Pediatr Neurol. 2011;45(2):89-94.
21. KulakW, Sobaniec W. Risk factors and prognosis of epilepsy in children with cerebral palsy in North-Eastern Poland. Brain Dev. 2003;25(7):499-506.
22. Zelnik N, Konopnicki M, Bennett-Back O, Castel-Deutsch T, Tirosh E. Risk factors for epilepsy in children with cerebral palsy. Eur J Paediatr Neurol. 2010;14(1):67-72.
23. Sellier E, Uldall P, Calado E, Sigurdardottir S, Torrioli MG, Platt MJ, et al. Epilepsy and cerebral palsy: characteristics and trends in children born in 1976–1998. Eur J Paediatr Neurol. 2012;16(1):48-55.
24. Bruck I, Antoniuk SA, Spessatto A, Bem RS, Hausberger R, Pacheco CG. Epilepsy in children with cerebral palsy. Arq Neuropsiquiatr. 2001;59(1):35-9.
25. Kwong KL, Wong SN, So KT. Epilepsy in children with cerebral palsy. Pediatr Neurol. 1998;19(1):31-6.
26. Zafeiriou DI, Kontopoulos EE, Tsikoulas I. Characteristics and prognosis of epilepsy in children with cerebral palsy. J Child Neurol. 1999;14:289-94.
27. Singhi P. The child with cerebral palsy-clinical consideration and management. Indian J Pediatr. 2001;68:531-7.
28. Carlsson M, Hagberg G, Olsson I. Clinical and aetiological aspects of epilepsy in children with cerebral palsy. Dev Med Child Neurol. 2003;45:371-6.
29. Delgado MR, Riela AR, Mills J, Pitt A, Browne R. Discontinuation of antiepileptic drug treatment after two seizure-free years in children with cerebral palsy. Pediatrics. 1996;97(2):192-7.
30. Şenbil N, Sonel B, Aydin ÖF, Gürer YK. Epileptic and non-epileptic cerebral palsy: EEG and cranial imaging findings. Brain Dev. 2002;24(3):166-9.

31. Hanci F, Türay S, Dilek M, Kabakuş N. Epilepsy and drug-resistant epilepsy in children with cerebral palsy: A retrospective observational study. Epilepsy Behav. 2020;112:107357.
32. Aksu F. Nature and prognosis of seizures in patients with cerebral palsy. Dev Med Child Neurol. 1990;32(8):661-8.
33. Hadjipanayis A, Hadjichristodoulou C, Youroukos S. Epilepsy in patients with cerebral palsy. Dev Med Child Neurol. 1997;39(10):659-63.
34. Fisher RS, Acevedo C, ArzimanoglouA, Bogacz A, Cross JH, Elger CE, et al. ILAE official report: a practical clinical definition of epilepsy. Epilepsia. 2014;55(4):475-82.
35. Wallace SJ. Epilepsy in cerebral palsy. Dev Med Child Neurol. 2001;43:713-7.

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