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Prevalence and Electro-Clinical Profile of Epilepsy in Children with Cerebral Palsy: A Cross-Sectional Study from a Tertiary Care Centre.

Dr. Harishma R Nath, MBBS[1]*, Dr. Shachi Jain Taran, MD[2], Dr. Monika Porwal Bagul, MD[3], Dr. Dimpal Dodiyar, MD[4].

[1]*Junior Resident, Department of Paediatrics, MGM Medical College & MY Hospital, Indore, Madhya Pradesh, India.*

[2]*Associate Professor, Department of Paediatrics, MGM Medical College & MY Hospital, Indore, Madhya Pradesh, India.*

[3]*Assistant Professor, Department of General Medicine, MGM Medical College & MY Hospital, Indore, Madhya Pradesh, India.*

[4]*Assistant Professor, Department of Paediatrics, MGM Medical College & MY Hospital, Indore, Madhya Pradesh, India.*

Correspondence:
Dr. Harishma R Nath.
Junior Resident, Department of Paediatrics, MGM Medical College & MY Hospital, Indore, Madhya Pradesh, India.

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Abstract

Background: Epilepsy is one of the most clinically significant comorbidities in children with cerebral palsy. The present study aimed to determine the prevalence and describe the electro-clinical profile of epilepsy in children with cerebral palsy attending a tertiary care centre in Central India. ***Methods:*** A hospital-based, cross-sectional, observational study was conducted at a tertiary care centre over an 18-month period. One hundred and fifty children aged 6 months to 15 years with a confirmed diagnosis of cerebral palsy were enrolled. All participants underwent detailed clinical assessment, electroencephalography, and magnetic resonance imaging of the brain. Epilepsy was classified as per the International League Against Epilepsy 2017 criteria. Statistical analysis was performed using SPSS version 25.0; the chi-square test and the Fisher-Freeman-Halton exact test were applied as appropriate, with $p<0.050$ considered statistically significant. ***Results:*** Epilepsy was present in 47.3% (n=71) of children with cerebral palsy. Spastic quadriplegia was the most common subtype (34.7%). Focal seizures were the most frequent seizure type (52.1%), followed by generalised (39.4%) and unknown onset (8.5%) seizures. Seizure onset was predominantly before 5 years of age (74.65%). Abnormal electroencephalographic background was observed in 66.0% and epileptiform discharges in 46.0% of all 150 children. Electroencephalographic abnormalities showed a highly significant association with epilepsy ($p<0.001$), whereas magnetic resonance imaging abnormalities alone did not ($p=0.235$). Periventricular leukomalacia was the most common neuroimaging finding (25.4%). Magnetic resonance imaging findings showed a significant association with cerebral palsy subtype ($p=0.027$). ***Conclusions:*** Epilepsy is a major comorbidity in nearly half of children with cerebral palsy. Electroencephalography is a significantly stronger predictor of epilepsy than magnetic resonance imaging in this population. An integrated electro-clinical-radiological approach is essential for early identification and optimal management of epilepsy in children with cerebral palsy.

Keywords: Cerebral palsy; Epilepsy; Electroencephalography; Seizures; Leukomalacia, Periventricular; Magnetic Resonance Imaging; Child.

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INTRODUCTION

Cerebral palsy (CP) represents one of the most common causes of chronic motor disability in childhood, arising from a non-progressive insult to the developing brain during the antenatal, perinatal, or early postnatal period. While the defining feature is a disturbance of movement and posture, the condition is rarely confined to motor impairment alone (1). Children with CP frequently experience a wide range of associated neurological comorbidities that substantially influence their quality of life, functional potential, and long-term outcomes (2).

Among these associated conditions, epilepsy occupies a particularly important place, both because of its high prevalence and because of the complex way in which it interacts with the underlying brain injury. Epilepsy in children with CP is not merely an additional diagnosis; it is often a reflection of the severity, timing, and nature of the original cerebral insult (3). The prevalence of epilepsy in children with CP is significantly higher than in the general paediatric population, with reported rates varying widely across studies — from approximately 20% in population-based registries to as high as 68.4% in hospital-based tertiary cohorts (4,5). This variability reflects differences in study design, population characteristics, diagnostic criteria, and healthcare settings.

The clinical expression of epilepsy in CP is remarkably heterogeneous. Seizure types range from focal seizures with or without impaired awareness to generalised seizures and epileptic spasms. Certain CP subtypes, particularly spastic

quadriplegia and mixed forms, consistently carry the highest epilepsy risk (6). Electroencephalography (EEG) plays a central role in characterising epilepsy in CP, and neuroimaging provides important structural context for understanding the underlying substrate (7). Despite the clinical significance of this comorbidity, population-specific data from tertiary care centres in Central India remain limited.

The present study aimed to determine the prevalence and describe the electro-clinical profile of epilepsy in children with CP attending the Neurodevelopmental Clinic of a tertiary care centre, with the objective of providing region-specific data to guide clinical management.

MATERIALS AND METHODS

Study Design and Setting

This hospital-based, cross-sectional, observational study was conducted in the Department of Paediatrics of a tertiary care referral centre in Central India over a period of 18 months. The study was conducted in accordance with the ethical standards laid down in the Declaration of Helsinki. Approval was obtained from the Institutional Ethics and Scientific Review Committee prior to commencement. Written informed consent was obtained from all parents or legal guardians. Confidentiality was maintained, participation was voluntary, and participants could withdraw at any stage without affecting clinical care.

Participants

A total of 150 children with CP, presenting to the Outpatient Department, Inpatient Department, or Neurodevelopmental Clinic during the study period, were enrolled after fulfilling the predefined inclusion and exclusion criteria.

Inclusion criteria

Children aged 6 months to 15 years with a confirmed diagnosis of CP — confirmed by a paediatric neurologist or neurodevelopmental paediatrician based on standard clinical criteria — presenting during the study period were included.

Exclusion criteria

Children were excluded if (i) parents or legal guardians declined to provide informed consent, or (ii) an EEG recording could not be obtained due to technical reasons, non-cooperation of the child, or loss to follow-up before investigations were completed.

Study Procedures

After enrolment, relevant clinical, developmental, and perinatal history was recorded using a predesigned, structured case record form. All enrolled children underwent: (i) detailed clinical history and neurological examination; (ii) classification of CP type (spastic, dyskinetic, ataxic, or mixed) and severity grading using the Gross Motor Function Classification System (GMFCS); (iii) classification of epilepsy and seizure type as per the International League Against Epilepsy (ILAE) 2017 criteria; (iv) EEG recording with assessment of background activity and epileptiform discharges; and (v) magnetic resonance imaging (MRI) of the brain with assessment of the predominant structural abnormality. EEG and MRI reports were reviewed and interpreted by a consultant neurologist and a consultant radiologist, respectively.

Sample Size

Sample size was calculated using Cochran's formula: $n = Z^2PQ/d^2$, with Z = 1.96 (95% confidence interval), P = 42.5% (estimated prevalence of epilepsy in CP, taken as the midpoint of the reported range of 35–50% based on Archana et al., 2022), Q = 57.5%, and d = 10% (absolute margin of error). The calculated sample size was 94, adjusted to 118 after accounting for a 20% non-response rate. A total of 150 patients were ultimately enrolled, as a higher number of eligible children were available during the study period.

Statistical Analysis

Data were entered into Microsoft Excel (Microsoft Corporation, Redmond, WA, USA) and analysed using SPSS version 25.0 (IBM Corp., Armonk, NY, USA). Categorical variables were expressed as frequencies and percentages. The chi-square test was applied for 2×2 contingency tables where assumptions were met. The Fisher-Freeman-Halton exact test (Monte Carlo simulation, 100,000 samples) was used for larger contingency tables where expected cell frequencies were insufficient for valid chi-square application. A p-value <0.050 was considered statistically significant. Exact p-values are reported to three decimal places.

RESULTS

The study included 150 children with CP. The age distribution showed a higher proportion of younger children, with 44.0% aged ≤5 years, 36.0% aged 5–10 years, and 20.0% aged 10–15 years. The cohort demonstrated a male predominance (56.0%, n=84) over females (44.0%, n=66). The majority of participants were from rural areas (68.7%, n=103). Regarding

perinatal history, 42.0% had low birth weight (<2.5 kg) and 42.0% were preterm (<37 weeks of gestation). Normal vaginal delivery was the most common mode (47.3%), followed by lower-segment caesarean section (LSCS) (41.3%) and instrumental delivery (11.3%). Among neonatal risk factors, perinatal asphyxia was documented in 51.3%, neonatal seizures in 41.3%, neonatal intensive care unit (NICU) stay in 36.7%, and pathological jaundice in 24.7% of cases (Table 1).

Table 1. Sociodemographic, perinatal, and neonatal profile of study participants (N=150).

Characteristic	Category	n	%
Age group	≤5 years	66	44.0
	5–10 years	54	36.0
	10–15 years	30	20.0
Sex	Male	84	56.0
	Female	66	44.0
Residence	Rural	103	68.7
	Urban	47	31.3
Birth weight	<2.5 kg	63	42.0
	2.5–3.0 kg	66	44.0
	>3.0 kg	21	14.0
Gestational age	Term (≥37 weeks)	87	58.0
	Preterm (<37 weeks)	63	42.0
Mode of delivery	Normal vaginal delivery	71	47.3
	LSCS	62	41.3
	Instrumental	17	11.3
NICU stay	Yes	55	36.7
Perinatal asphyxia	Yes	77	51.3
Neonatal seizures	Yes	62	41.3
Pathological jaundice	Yes	37	24.7

LSCS = lower-segment caesarean section; NICU = neonatal intensive care unit.

Spastic CP was the predominant type (79.4%), with spastic quadriplegia being the most common subtype (34.7%, n=52), followed by spastic diplegia (24.0%, n=36) and spastic hemiplegia (20.7%, n=31). Dyskinetic CP accounted for 15.3% (n=23), while ataxic (3.3%) and mixed (2.0%) types were rare. Regarding GMFCS severity, moderate disability (GMFCS III) was most common (40.0%), followed by severe (GMFCS IV, 29.3%), mild (GMFCS I–II, 15.3%), and very severe (GMFCS V, 15.3%). Epilepsy was present in 47.3% (n=71) of children with CP, while 52.7% (n=79) did not have epilepsy (Table 2).

Table 2. Clinical profile of cerebral palsy and prevalence of epilepsy (N=150).

Characteristic	Category	n	%
CP type	Spastic quadriplegia	52	34.7
	Spastic diplegia	36	24.0
	Spastic hemiplegia	31	20.7
	Dyskinetic	23	15.3
	Ataxic	5	3.3
	Mixed	3	2.0
GMFCS severity	Mild (I–II)	23	15.3
	Moderate (III)	60	40.0
	Severe (IV)	44	29.3
	Very severe (V)	23	15.3
Epilepsy	Present	71	47.3
	Absent	79	52.7

CP = cerebral palsy; GMFCS = Gross Motor Function Classification System.

Among children with epilepsy (n=71), focal seizures were the most common type (52.1%, n=37) — comprising focal seizures with preserved consciousness (29.6%) and impaired consciousness (22.5%). Generalised motor seizures accounted for 39.4% (n=28). Unknown onset seizures constituted 8.5% (n=6), comprising epileptic spasms (2.8%) and behavioural arrest (5.6%). No absence seizures were recorded. Seizure onset was predominantly before 5 years of age (74.65%); onset after 10 years was rare (2.81%). Monotherapy was used in 57.78% and polytherapy in 42.22% of children. Seizures were controlled in 42.25% (n=30), partially controlled in 26.76% (n=19), and refractory in 30.99% (n=22). A history of febrile convulsions was present in 25.35% and status epilepticus in 15.50% of patients (Table 3).

Table 3. Clinical profile of epilepsy in children with cerebral palsy (N=71).

Characteristic	Category	n	%
Seizure type (ILAE 2017)	Focal – preserved consciousness	21	29.6
	Focal – impaired consciousness	16	22.5
	Total focal	37	52.1
	Generalised motor seizures	28	39.4
	Unknown – epileptic spasms	2	2.8
	Unknown – behavioural arrest	4	5.6
Age at seizure onset	<5 years	53	74.65
	5–10 years	16	22.54
	>10 years	2	2.81
Anti-seizure medication	Monotherapy	41	57.78
	Polytherapy	30	42.22
Seizure control	Controlled	30	42.25
	Partially controlled	19	26.76
	Uncontrolled/refractory	22	30.99
History of febrile convulsions	Yes	18	25.35
History of status epilepticus	Yes	11	15.50

ILAE = International League Against Epilepsy.

Among all 150 children, abnormal EEG background was observed in 66.0% (n=99) and epileptiform discharges in 46.0% (n=69). Generalised synchronous slowing was the most common EEG background abnormality (30.0%, n=45), followed by generalised asynchronous slowing (24.0%, n=36) and focal slowing (12.0%, n=18); hypsarrhythmia was noted in 2 patients and was included under generalised slowing. Among epileptiform discharge patterns, focal epileptiform discharges were most common (21.3%, n=32), followed by generalised (14.0%, n=21) and multifocal discharges (10.7%, n=16). MRI brain was abnormal in 86.7% (n=130) of children. Periventricular leukomalacia (PVL) was the most frequent neuroimaging finding (25.4%, n=38), followed by cortical/subcortical lesions consistent with hypoxic-ischaemic encephalopathy (HIE) sequelae (21.3%, n=32) and deep grey matter (basal ganglia/thalamus) involvement (16.7%, n=25). Kernicterus changes were seen predominantly in dyskinetic CP (3.3%, n=5) (Table 4).

Table 4. Electroencephalography and magnetic resonance imaging findings in children with cerebral palsy (N=150).

Investigation	Finding	n	%
EEG background	Normal	51	34.0
	Focal slowing	18	12.0
	Generalised asynchronous slowing	36	24.0
	Generalised synchronous slowing*	45	30.0
EEG epileptiform discharges	Absent	81	54.0
	Focal epileptiform discharges	32	21.3
	Generalised epileptiform discharges	21	14.0
	Multifocal epileptiform discharges	16	10.7
MRI brain	Normal	20	13.3
	Periventricular leukomalacia	38	25.4
	Cortical/subcortical lesion (HIE sequelae)	32	21.3
	Deep grey matter (basal ganglia/thalamus)	25	16.7
	Intraventricular haemorrhage sequelae	15	10.0
	Maldevelopment	9	6.0
	Kernicterus	5	3.3
	Arterial infarction	6	4.0

*Includes 2 patients with hypsarrhythmia. EEG = electroencephalography; HIE = hypoxic-ischaemic encephalopathy;

MRI = magnetic resonance imaging.

EEG background abnormality was significantly more common in children with epilepsy compared to those without (88.7% vs. 45.6%; χ^2=31.045, df=1, p<0.001). Epileptiform discharges showed an even stronger association with epilepsy (85.9% vs. 10.1%; χ^2=86.468, df=1, p<0.001). MRI abnormalities, however, did not significantly differentiate children with epilepsy from those without (90.1% vs. 83.5%; χ^2=1.408, df=1, p=0.235). Among neonatal factors assessed — NICU stay, perinatal asphyxia, neonatal seizures, and pathological jaundice — none showed a statistically significant independent association with epilepsy (p=0.314, p=0.636, p=0.436, and p=0.846, respectively) (Table 5). MRI findings showed a statistically significant association with CP subtype (Fisher-Freeman-Halton exact test, p=0.027), with PVL predominating in spastic subtypes, deep grey matter involvement in dyskinetic CP, and arterial infarction in hemiplegia. The association between seizure type and CP subtype (p=0.070) and between EEG epileptiform discharge pattern and CP subtype (p=0.554) did not reach statistical significance.

Table 5. Association of electroencephalography, magnetic resonance imaging, and neonatal factors with epilepsy (N=150).

Variable	Category	Epilepsy Yes (n=71)	Epilepsy No (n=79)	χ^2	df	p-value
EEG background	Abnormal	63 (88.7%)	36 (45.6%)	31.045	1	<0.001*
EEG epileptiform discharges	Present	61 (85.9%)	8 (10.1%)	86.468	1	<0.001*
MRI brain	Abnormal	64 (90.1%)	66 (83.5%)	1.408	1	0.235
NICU stay	Yes	29 (40.8%)	26 (32.9%)	1.014	1	0.314
Perinatal asphyxia	Yes	35 (49.3%)	42 (53.2%)	0.224	1	0.636
Neonatal seizures	Yes	27 (38.0%)	35 (44.3%)	0.607	1	0.436
Pathological jaundice	Yes	17 (23.9%)	20 (25.3%)	0.038	1	0.846

*Statistically significant (p<0.050). Chi-square test applied. df = degrees of freedom; EEG = electroencephalography; MRI = magnetic resonance imaging; NICU = neonatal intensive care unit.'

DISCUSSION

The present study evaluated the prevalence and electro-clinical characteristics of epilepsy in children with CP attending a tertiary care centre in Central India. Epilepsy was found in 47.3% of children with CP, consistent with the higher prevalence figures reported from hospital-based and tertiary care-based cohorts globally. Gong et al. reported a pooled prevalence of 38.0% (95% CI: 34.8–41.2%) across 72 studies comprising 53,969 individuals (4), while Alyoubi et al. reported a notably high prevalence of 68.4% in a hospital-based cohort from Saudi Arabia (5). Kwong et al., in an earlier hospital-based study, also reported a prevalence of 41% in children with CP, broadly comparable to our findings (7). The relatively high prevalence in our study is consistent with the known referral bias of tertiary centres, which disproportionately attract children with more severe and complex neurological disease.

Spastic CP was the predominant type in our cohort (79.4%), with spastic quadriplegia being the most common subtype (34.7%), consistent with most Indian and South Asian hospital-based studies. Bhati et al., in a North Indian clinico-etiological profile of children with CP, similarly reported a predominance of spastic CP with quadriplegia as the leading subtype (8). Delacy and Reid, using the Australian Cerebral Palsy Register, further demonstrated that the spectrum of associated impairments — including epilepsy — varies significantly by CP subtype and GMFCS level (14). The high proportion of perinatal asphyxia (51.3%) as the predominant aetiological factor reflects the continued burden of preventable perinatal complications in resource-limited settings, corroborated by observations of Serdaroğlu et al. (9) and Alyoubi et al. (5). The predominance of rural participants (68.7%) underscores the importance of strengthening neurodevelopmental services in peripheral healthcare settings.

Focal seizures were the most common seizure type (52.1%), followed by generalised motor seizures (39.4%) and unknown onset seizures (8.5%). This pattern is consistent with Gururaj et al., who also reported focal epileptic discharges as the predominant electro-clinical pattern in CP (6). Archana et al. similarly noted a wide spectrum of seizure types across CP subtypes, reflecting the heterogeneity of the underlying cortical injury (10). The predominance of early seizure onset (74.65% before 5 years) is in agreement with Pavone et al., who reported a mean epilepsy onset age of 21 months in children with CP plus epilepsy, compared to 67 months in children with epilepsy alone, underscoring the early epileptogenic impact of perinatal brain injury (11). Wallace earlier emphasised that the timing and nature of the original cerebral insult often shape the clinical phenotype of epilepsy in CP — an observation that remains pertinent to our findings (3).

A notable finding was the high rate of refractory epilepsy (30.99%), reflecting the complexity of epileptogenesis in children with structural brain injury. Kułak and Sobaniec reported intractable epilepsy in 51.2% of their cohort enriched for spastic tetraplegia (12). Archana et al. reported a lower refractoriness rate of 10.1%, likely reflecting a broader, less severe CP population (10). The polytherapy requirement in 42.22% of our patients is consistent with prior observations that diffuse brain injury, multifocal epileptiform activity, and network dysfunction reduce responsiveness to single-drug therapy (12).

EEG was highly informative in our study. Abnormal background was present in 66.0% and epileptiform discharges in 46.0% of all 150 children. The strong and highly significant association of EEG abnormalities with epilepsy (both $p<0.001$) confirms EEG as the most reliable electrophysiological predictor of epilepsy in this population, consistent with prior literature (7). In contrast, MRI abnormalities — while prevalent (86.7%) — did not significantly differentiate children with epilepsy from those without ($p=0.235$). This suggests that structural brain injury, while near-universal in CP, does not by itself reliably predict clinical epilepsy, and that functional network-level disruption captured by EEG is a more sensitive marker.

MRI findings showed a statistically significant association with CP subtype ($p=0.027$). PVL predominated in spastic subtypes, deep grey matter involvement characterised dyskinetic CP, and arterial infarction was predominantly associated with hemiplegia — findings consistent with the well-established neuropathological substrates of each CP subtype, as systematically reviewed by Krägeloh-Mann and Horber (13). The high frequency of PVL (25.4%) reflects the large proportion of preterm births (42%) in our cohort. The association between seizure type and CP subtype did not reach statistical significance ($p=0.070$), possibly due to small subgroup sizes in rarer CP subtypes. Similarly, EEG epileptiform discharge patterns did not show a significant association with CP subtype ($p=0.554$), underscoring the network-level rather than purely focal nature of epileptogenesis in CP.

None of the individual neonatal factors assessed — including perinatal asphyxia, neonatal seizures, NICU stay, and pathological jaundice — showed a statistically significant independent association with epilepsy. This finding should be interpreted cautiously: these factors contribute collectively to the severity and pattern of brain injury rather than acting as isolated predictors of epilepsy. The cross-sectional design precludes causal inference, and multivariate analysis in larger prospective studies may better delineate their independent contributions. Odding et al. similarly noted that the epidemiology of CP is shaped by the cumulative interplay of antenatal, perinatal, and postnatal risk factors rather than by any single isolated exposure (2).

The study has several strengths, including a well-defined study population, systematic use of the ILAE 2017 seizure classification, and comprehensive electro-clinical-radiological evaluation of all participants. The standardised definition and classification framework proposed by Rosenbaum et al. was applied consistently throughout, enabling meaningful comparison with international literature (1). Several limitations should be acknowledged. First, the cross-sectional design precludes causal inference. Second, the single-centre, tertiary-care setting introduces referral bias and may overestimate prevalence compared with community-based cohorts. Third, subgroup sizes for rarer CP subtypes (ataxic, mixed) were limited, restricting statistical power for subtype-specific analyses. Fourth, detailed genetic and metabolic aetiological evaluation was not performed. Finally, some historical data relied on caregiver recall, introducing the possibility of recall bias.

CONCLUSION

This study highlights that epilepsy is a highly prevalent and clinically significant comorbidity in children with CP, affecting nearly half of the study population attending a tertiary care centre in Central India. The predominance of early-onset seizures, high rates of polytherapy requirement, and significant proportion of refractory epilepsy underscore the therapeutic complexity of this condition. EEG abnormalities are the most reliable predictor of epilepsy in children with CP, significantly outperforming MRI in this regard.

Distinct neuroimaging patterns correspond meaningfully to CP subtypes, reflecting differing aetiological substrates. The findings support the integration of systematic EEG evaluation into the routine assessment of all children with CP, regardless of the presence of overt clinical seizures.

An integrated electro-clinical-radiological approach is essential for early identification, accurate classification, and optimal management of epilepsy in this vulnerable population. Prospective, multicentre studies with long-term follow-up are recommended to further characterise seizure evolution, treatment outcomes, and quality of life in children with CP-associated epilepsy.

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AUTHOR CONTRIBUTIONS

All authors contributed substantially to the conception and design of the study, the acquisition, analysis, and interpretation of data, drafting and critical revision of the manuscript for important intellectual content, and final approval of the version to be published. All authors agree to be accountable for all aspects of the work.

REFERENCES

1. Rosenbaum P, Paneth N, Leviton A, Goldstein M, Bax M, Damiano D, et al. A report: the definition and classification of cerebral palsy April 2006. Dev Med Child Neurol Suppl 2007;109:8-14.
2. Odding E, Roebroeck ME, Stam HJ. The epidemiology of cerebral palsy: incidence, impairments and risk factors. Disabil Rehabil 2006;28(4):183-91.
3. Wallace SJ. Epilepsy in cerebral palsy. Dev Med Child Neurol 2001;43(10):713-7.
4. Gong C, Liu A, Lian B, Wu X, Zeng P, Hao C, et al. Prevalence and related factors of epilepsy in children and adolescents with cerebral palsy: a systematic review and meta-analysis. Front Pediatr 2023;11:1189648.
5. Alyoubi R, Mirza A, Busaleh F, Albuali W, Almahmoud R, Almarzooq A, et al. Risk factor predictors for developing epilepsy in cerebral palsy patients in a tertiary hospital in Saudi Arabia: a retrospective study. Cureus 2024;16(5):e59980.
6. Gururaj AK, Sztriha L, Bener A, Dawodu A, Eapen V. Epilepsy in children with cerebral palsy. Seizure 2003;12(2):110-4.
7. Kwong KL, Wong SN, So KT. Epilepsy in children with cerebral palsy. Pediatr Neurol 1998;19(1):31-6.
8. Bhati P, Sharma S, Jain R, Rath B, Beri S, Gupta VK, et al. Cerebral palsy in North Indian children: clinico-etiological profile and comorbidities. J Pediatr Neurosci 2019;14(1):30-5.
9. Serdaroğlu A, Cansu A, Ozkan S, Tezcan S. Prevalence of cerebral palsy in Turkish children between the ages of 2 and 16 years. Dev Med Child Neurol 2006;48(6):413-6.
10. Archana K, Saini L, Gunasekaran PK, Yadav J, Suthar R, Sahu JK, et al. The profile of epilepsy and its characteristics in children with cerebral palsy. Seizure 2022;101:190-6.
11. Pavone P, Gulizia C, Le Pira A, Greco F, Parisi P, Di Cara G, et al. Cerebral palsy and epilepsy in children: clinical perspectives on a common comorbidity. Children (Basel) 2020;8(1):16.
12. Kułak W, Sobaniec W. Risk factors and prognosis of epilepsy in children with cerebral palsy in north-eastern Poland. Brain Dev 2003;25(7):499-506.
13. Krägeloh-Mann I, Horber V. The role of magnetic resonance imaging in elucidating the pathogenesis of cerebral palsy: a systematic review. Dev Med Child Neurol 2007;49(2):144-51.
14. Delacy MJ, Reid SM; Australian Cerebral Palsy Register Group. Profile of associated impairments at age 5 years in Australia by cerebral palsy subtype and GMFCS level for birth years 1996 to 2005. Dev Med Child Neurol 2016;58 Suppl 2:50-6.