ORIGINAL ARTICLE

Characteristics of Epilepsy in Paediatric Population and Its Effect on The Development and Educational Performance

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Abstract

Background: ***Epilepsy is a prevalent neurological disorder in children, profoundly affecting cognitive, social, and educational development.***

Objective: ***To assess the characteristics, comorbidities, and developmental and educational outcomes of paediatric epilepsy.***

Methods: ***A cross-sectional study involving 670 children with epilepsy was conducted at a neuropediatric unit. Data on demographic, clinical, and developmental variables were analyzed using logistic regression to determine predictors of seizure control and outcomes.***

Results: ***The median age was 7 years (51.8% male). Generalized tonic-clonic seizures (73.1%) were the most common. Uncontrolled epilepsy correlated significantly with epileptic syndromes, status epilepticus, and abnormal neurological findings ($p < 0.001$). Behavioural issues and female gender were linked to poorer outcomes, while normal MRI and EEG findings predicted better control. Consanguinity was associated with favourable outcomes ($p < 0.05$).***

Conclusion: ***Seizure control and comorbidities shape developmental outcomes in paediatric epilepsy. Early diagnosis, individualized treatment, and educational support are essential to enhance quality of life and learning potential.***

Keywords: Paediatric epilepsy; seizure control; developmental outcomes; comorbidities; educational performance

1. Introduction

Epilepsy is a neurological disorder characterized by recurrent, unprovoked seizures that result from abnormal electrical activity in the brain. It affects approximately 1% of the global population, with a notable incidence in the paediatric population.[1] In children, epilepsy can present unique challenges, as the developing brain is particularly susceptible to the effects of recurrent seizures. The characteristics of epilepsy in the paediatric population, including seizure types, aetiology, and comorbid conditions, significantly influence the overall development and educational performance of affected children.[2]

Paediatric epilepsy encompasses a broad spectrum of seizure types and syndromes, each with distinct clinical features and prognostic implications. For instance, benign epilepsy with centrotemporal spikes (BECTS) typically presents with focal seizures during sleep and has a favourable prognosis, often resolving before adolescence. In contrast, severe forms such as Lennox-Gastaut syndrome (LGS) involve multiple seizure types, cognitive impairment, and resistance to treatment, posing significant challenges for management and long-term outcomes.[3]

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The impact of epilepsy on a child's development is multifaceted, affecting cognitive, social, and emotional domains. Frequent and uncontrolled seizures can lead to cumulative neurological damage, resulting in cognitive deficits, learning disabilities, and behavioural problems.[4] Furthermore, the stigma associated with epilepsy can exacerbate these challenges, leading to social isolation and reduced self-esteem in children.[5] These factors collectively hinder the educational performance of children with epilepsy, contributing to lower academic achievement and reduced opportunities for future success.

Effective management of paediatric epilepsy requires a comprehensive approach that includes accurate diagnosis, appropriate treatment, and ongoing support for developmental and educational needs. Advances in neuroimaging and genetic testing have improved the diagnostic accuracy for various epileptic syndromes, facilitating tailored treatment strategies.[6] Moreover, early intervention with antiepileptic drugs (AEDs) and non-pharmacological therapies, such as ketogenic diets and surgical options, can significantly improve seizure control and overall quality of life.[7]

Equally important is the provision of educational support to address the specific needs of children with epilepsy. Individualized Education Programs (IEPs) and accommodations in the classroom can help mitigate the impact of seizures and associated cognitive deficits on learning.[1] Collaboration between healthcare providers, educators, and families is essential to ensure that children with epilepsy receive the necessary support to achieve their full academic potential.

The previous studies illustrated how epilepsy management could affect the development and educational performance in the paediatric population. However, they did not address the factors that could affect this development and achievement, rather than the control. The early prediction of the development status and educational achievement could be helpful in the early management of severely affected cases and hence improve their future. Therefore, this study aimed to investigate the characteristics of epilepsy, the associated comorbidities, and the factors associated with development and educational performance in the paediatric population attending our specialized neuropediatric unit.

2. Material and Methods

This study was performed as an observational cross-sectional study to include the entire paediatric population attending the neuropediatric unit at Al-Azhar University Hospitals, Faculty of Medicine, Cairo, Egypt, for follow-up and management of their epilepsy. Informed consent was taken from either one of the parents, both, or their representatives. This study was performed in concordance with the Declaration of Helsinki, and the IRB acceptance was obtained from the ethical department at the Faculty of Medicine, Al-Azhar University. The Ethics Committee accepted the study protocol with number 54 in 2022.

We included all paediatric patients aged from 3 months to 18 years with a clear diagnosis of epilepsy or epileptic syndrome, either associated with other comorbidities or not, who have been attending the neuropediatric unit for regular follow-up and management. All paediatric patient with unclear diagnosis or nonepileptic attacks disorders were excluded from the study.

The data of the included paediatric patients were retrieved from the archived files in the neuropediatric unit. Any missing data was taken when the patient's family attended their follow-up appointment. The included data were, age, sex, consanguinity, prenatal, natal and postnatal history, head trauma, family history of epilepsy, status epilepticus, febrile seizure, onset of seizure onset, epilepsy classification, epilepsy syndrome, seizure triggers, antiseizure medications, seizure control, duration of treatment, neurological examination, EEG findings, MRI findings, developmental and educational achievement, behavioural abnormalities and comorbidities. The Denver Developmental Screening Test (DDST) was used to assess the developmental progress of children from birth to 6 years.[8] It contains four domains: personal-social, fine motor-adaptive, language, and gross motor skills. Furthermore, the Stanford-Binet Intelligence Scales were used to evaluate the intelligence and cognitive abilities as an indicator of educational scale in children older than 6 years. [9]

Patient were categorized based on developmental and educational achievement into three groups: average, less than average and impaired. The impaired score was considered if the infant or child was below three standard deviations (SD) for the normal developmental category for his or her age or associated or with IQ test results bellow 35%. The less than average category was considered if the child or infant was higher than impaired and less than average.

Patients were also classified according to the control of their epilepsy into controlled and uncontrolled patients. The definition of epilepsy control was considered when the child has a stable antiseizure regimen for at least 6 months. Without significant side effects or impairment of the quality of life, and with a considerable period of seizure freedom. [10]

Statistical analysis was performed using the IBM Statistical Package for Social Sciences (SPSS) platform for Mac version 26 (IBM Co., USA). Descriptive statistics, including frequencies, percentages, medians, and interquartile ranges (IQR), were calculated to summarize the demographic and clinical characteristics of the study population. For categorical variables, differences between controlled and uncontrolled seizure groups were assessed using the Chi-square test or Fisher's exact test, as appropriate, with a significance level set at p≤0.05. Continuous variables were compared between groups using the Mann-Whitney U test.

Univariate and multivariate analyses were conducted to identify factors associated with uncontrolled seizures and developmental/educational outcomes. Binary logistic regression was used to evaluate the association of various independent variables with the likelihood of uncontrolled seizures and intact developmental and educational outcomes. The odds ratios (OR) and 95% confidence intervals (CI) were calculated for each predictor, with statistical significance determined by p-values ≤0.05.

Additionally, ordinal logistic regression was used to examine factors influencing the likelihood of being in higher categories of developmental and educational outcomes. The proportional odds assumption was tested using the parallel lines test, with a significance level of p > 0.05. All statistical tests were two-tailed, and p-values ≤0.05 were considered statistically significant.

3. Results

We have included 670 epileptic children who are followed up in our neuropediatric units and have archived files with detailed history. The median age of the included children was 7 years (IQR3-12). The male percentage of the included children was 51.8%. The most common seizure type was generalized tonic-clonic (GTC) seizures with 73.1% and epileptic syndromes were present in 33.7%. 74.5 % of the included children were taking levetiracetam either alone or in combination with other antiseizure medications. The EEG was normal in 347 (51.8%) patients and abnormal in 323 (48.2%) patients. The MRI brain was normal in 70% of the patients and abnormal in 30%. The demographic characteristics of the included children were summarized in Table 1.

The comparison between patients with controlled epilepsy (n=588) and uncontrolled epilepsy (n=82) revealed a significant difference as regards consanguinity, with a higher prevalence among the controlled group (p=0.002). Similarly, infections during pregnancy were reported exclusively in the controlled group (p=0.025). No cases of drug use during pregnancy were recorded in either group. On the other hand, birth asphyxia, delayed developmental history and post-natal infections were common in the uncontrolled group (p <0.05 in all). Family history of seizures and history of febrile convulsions were less frequent in the uncontrolled group (17.1%) than in the controlled group (31.1%) (p <0.01 in both). However, the history of status epilepticus, epileptic syndromes and GTC seizures were markedly higher in the uncontrolled group (p<0.001 in both). On the other hands, the focal seizure was higher in the controlled group (p<0.001). In terms of treatment, levetiracetam was used more frequently in the uncontrolled group (91.5%) than in the controlled group (72.1%) (p<0.001), and all patients in the uncontrolled group received valproate, compared to 40.0% in the controlled group (p<0.001). Normal neurological examination, EEG and MRI were higher in the controlled group (p<0.001). Furthermore, intact development and favourable educational outcomes were observed in 46.4% of the controlled group, with none reported in the uncontrolled group (p<0.001) (Table 2).

We have also evaluated the impact of age, seizure onset, and treatment-related parameters on the seizure control by comparison between controlled and uncontrolled epilepsy groups using the Mann-Whitney test. The median age of patients was slightly lower in the controlled group (p=0.007). However, there was no significant difference as regards the seizure onset between the two groups (p=0.745). Furthermore, the number of anti-seizure medications required, and the duration of anti-epileptic drug use were significantly higher in the uncontrolled group (p<0.05) (Table 3).

Binary logistic regression was performed to identify independent predictors of uncontrolled seizures the included children. The results demonstrated that the female sex significantly increased the likelihood of uncontrolled seizures (OR=4.86, 95% CI=2.07–11.41, p<0.001). Age was also a significant predictor, with each additional year increasing the odds of uncontrolled seizures by 30% (OR=1.30, 95% CI=1.16–1.46, p<0.001). Furthermore, patients experiencing GTC seizures, status epilepticus, precipitating infections, abnormal neurological examination were proven to have a higher risk of uncontrolled seizures (p<0.05). On the other hand, normal MRI findings were protective, reducing the odds of uncontrolled seizures by 85% (OR=0.15, 95% CI=0.06–0.39, p<0.001). Other factors, including family history of seizures (p=0.490), history of febrile convulsions (p=0.479), age of onset of seizures (p=0.806), and

normal EEG findings (p=0.622), were not significantly associated with uncontrolled seizures. Epileptic syndromes showed a borderline association (OR=2.47, 95% CI=0.98–6.18, p=0.054) (Table 4).

The ordinal logistic regression analysis identified several predictors for the likelihood of being in a higher developmental and educational impairment category among the included epileptic children. Female patients were more likely to belong to a higher impairment category (OR=1.85, 95% CI=1.43–2.33, p<0.001). A family history of seizures reduced the odds of impairment (OR=0.70, 95% CI=0.53–0.93, p=0.01). Significant head trauma was strongly associated with lower impairment (OR=0.41, 95% CI=0.24–0.69, p<0.001). Epileptic syndromes, abnormal examination and behavioural abnormalities significantly increased the likelihood of impairment (p<0.05). Normal EEG and normal MRI findings were highly protective (p<0.05). Non-significant factors included age (p=0.17), GTC seizures (p=0.16), history of febrile convulsion (p=0.69), status epilepticus (p=0.61), consanguineous marriage (p=0.68), delayed developmental history (p=0.40), birth asphyxia (p=0.47), and significant postnatal infection (p=0.17). The proportional odds assumption was satisfied, as indicated by the parallel line test (p=0.1) (Table 5).

The analysis identified several independent factors associated with intact development and favourable educational outcomes in epileptic children. Female gender and positive family history of seizures significantly decreased the likelihood of intact development and educational outcomes (p<0.001). Furthermore, Epileptic syndromes, abnormal neurological examination and precipitating infections were significantly associated with reduced likelihood of intact development and education (p<0.05). On the other hands, normal MRI and EEG findings were significantly associated with favourable outcomes, doubling the odds (p<0.05). Other variables, including age (p=0.622), GTC seizures (p=0.355), history of febrile convulsions (p=0.538), and age of seizure onset (p=0.146), were not significantly associated with intact development and educational outcomes (Table 6).

Table 1. The demographic characteristics of the included 670 epileptic children

CHARACTERISTIC		NUMBER (PERCENTAGE)
GENDER (MALE)		347 (51.8)
AGE (YEARS), MEDIAN (IQR)		7 (3 – 12)
CONSANGUINEOUS MARRIAGE		336 (50.1)
INFECTION DURING PREGNANCY		33 (4.9)
BIRTH ASPHYXIA (HIE)		129 (19.3)
DELAYED DEVELOPMENTAL HISTORY		143 (21.3)
SIGNIFICANT POST-NATAL INFECTION		27 (4.0)
SIGNIFICANT HEAD TRAUMA		51 (7.6)
FAMILY HISTORY OF SEIZURES		197 (29.4)
HISTORY OF FEBRILE CONVULSION		118 (17.6)
HISTORY OF STATUS EPILEPTICS		96 (14.3)
SEIZURE TYPE	Absence	33 (4.9)
	Atonic	15 (2.2)
	Focal	80 (11.9)
	GTC	490 (73.1)
	Myoclonus	27 (4.0)
	Temporal	14 (2.1)
	Tonic	7 (1.0)
	Visual hallucination	4 (0.6)
	Controlled seizure	588 (87.8)
EPILEPTIC SYNDROME		226 (33.7)
THE PRECIPITATING FACTOR OF SEIZURES	Infection	289 (43.1)
	Mense	24 (3.6)
TYPE OF REFLEX SEIZURES	Photosensitive reflex	26 (3.9)
	photosensitive reflex & startle response	3 (0.45)
	Startle epilepsy	3 (0.45)
AGE OF ONSET OF SEIZURES (YEARS), MEDIAN (IQR)		24 (6- 72)
ANTIEPILEPTIC DRUGS	Levetiracetam	499 (74.5)
	Valproate	317 (47.3)
	Carbamazepine	57 (8.5)
	Lamotrigine	39 (5.8)
	Clonazepam	35 (5.2)
	Topiramate	17 (2.5)
	Lacosamide	12 (1.8)
	Oxcarbazepine	48 (7.2)
	Phenytoin	12 (1.8)
DURATION OF ANTI-EPILEPTIC DRUG (MEDIAN (IQR)		3 (1- 5)
NEUROLOGICAL EXAMINATION	Hemiparesis	63 (31.82)
	Quadriparesis	58 (29.29)
	Spastic	101 (51.01)
	Mental	118 (59.60)
	Weakness	14 (7.07)
NORMAL EEG		347 (51.8)
NORMAL MRI FINDINGS		469 (70.0)

DEVELOPMENT AND EDUCATIONAL OUTCOME	Intact	273 (40.7)
	Less than average	133 (19.9)
	Impaired	264 (39.4)
BEHAVIORAL ABNORMALITIES (N=271)	ADHD	103 (38.01)
	Autism	63 (23.25)
	conduct disorder	12 (4.43)
	CP	93 (34.32)

IQR; INTERQUARTILE RANGE, HIE; HYPOXIC-ISCHEMIC *ENCEPHALOPATHY, GTC; GENERALIZED TONIC CLONIC, ADHD, ATTENTION DEFICIT HYPERKINETIC DISORDER, CP; CEREBRAL PALSY*

Table 2. Comparison analysis illustrating factors that are corelated with epilepsy control in epileptic children.

VARIABLE	Controlled (n=588)		Uncontrolled (n=82)		p-value
	No.	%	No.	%	
FEMALE	284	48.3	39	47.6	0.784
CONSANGUINEOUS MARRIAGE	308	52.4	28	34.1	0.002*
INFECTION DURING PREGNANCY	33	5.6	0	0.0	0.025*
BIRTH ASPHYXIA (HEE)	105	17.9	24	29.3	0.014*
DELAYED DEVELOPMENTAL HISTORY	102	17.3	41	50.0	<0.001*
SIGNIFICANT POST-NATAL INFECTION	3	0.5	24	29.3	<0.001*
SIGNIFICANT HEAD TRAUMA	51	8.7	0	0.0	0.006*
FAMILY HISTORY OF SEIZURES	183	31.1	14	17.1	0.009*
HISTORY OF FEBRILE CONVULSION	114	19.4	4	4.9	0.001*
HISTORY OF STATUS EPILEPTICS	51	8.7	45	54.9	<0.001*
GTC SEIZURE	412	70.1	78	95.1	<0.001*
FOCAL SEIZURE	72	12.2	7	8.5	<0.001*
EPILEPTIC SYNDROME	167	28.4	59	72.0	<0.001*
PRECIPITATING INFECTION	228	38.8	61	74.4	<0.001*
PRECIPITATING MENSES	24	4.1	0	0.0	0.05
LEVETIRACETAM	424	72.1	75	91.5	<0.001*
VALPROATE	235	40.0	82	100.0	<0.001*
NORMAL NEUROLOGICAL EX.	457	77.7	4	4.9	<0.001*
NORMAL EEG	333	56.6	14	17.1	<0.001*
NORMAL MRI	446	75.9	23	28.0	<0.001
BEHAVIORAL ABNORMALITIES	193	32.8	78	95.1	<0.001
INTACT DEVELOPMENT AND EDUCATIONAL OUTCOME	273	46.43	0	0	<0.001*

Table 3. Comparison analysis illustrating continuous factors that are corelated with epilepsy control in epileptic children.

VARIABLE	CONTROLLED (N=588)		UNCONTROLLED (N=82)		P-VALUE
	Median	IQR	Median	IQR	
AGE (YEARS)	7	3 – 12	7.5	5 - 15	0.007*
AGE OF ONSET OF SEIZURES (YEARS)	24	6 – 72	12	6 – 54	0.745
NUMBER OF ANTI-SEIZURE	1	1 – 2	3	3 – 4	<0.001*
DURATION OF ANTI-EPILEPTIC DRUG (YEARS)	3	1 - 5	3	2 – 8	0.014

IQR, INTERQUARTILE RANGE; SIGNIFICANT P-VALUE ≤ 0.05 USING MANN-WHITNEY TEST.

Table 4. Binary logistic regression for the uncontrolled seizure among epileptic pediatric patients

INDEPENDENT VARIABLES	OR	95% CI		P-VALUE
FEMALE	4.86	2.07	11.41	<0.001*
AGE	1.30	1.16	1.46	<0.001*
GTC	5.66	1.58	20.24	0.008*
FAMILY HISTORY OF SEIZURES	0.65	0.19	2.20	0.490
HISTORY OF FEBRILE CONVULSION	2.01	0.29	13.93	0.479
HISTORY OF STATUS EPILEPTICS	5.27	2.21	12.55	<0.001*
AGE OF ONSET OF SEIZURES	1.00	0.99	1.01	0.806
EPILEPTIC SYNDROME	2.47	0.98	6.18	0.054*
PRECIPITATING INFECTION	2.81	1.27	6.24	0.011*
NEUROLOGICAL EXAMINATION ABNORMAL	31.94	7.55	135.08	<0.001*
NORMAL EEG FINDING	0.75	0.24	2.37	0.622
NORMAL MRI FINDING	0.15	0.06	0.39	<0.001*
CONSTANT	-9.07	1.13	64.11	<0.001*

BETA, REGRESSION COEFFICIENT; GTC, GENERALIZED TONIC COLONIC; SE, STANDARD ERROR; *, SIGNIFICANT P-VALUE ≤ 0.05.

Table 5. Ordinal logistic regression to predict being in the higher category of the development and educational outcome (intact = 0, less than average = 1, impaired = 2

VARIABLE	ESTIMATE	STD. ERROR	WALD	P-VALUE	OR	95% CI	
INTACT	-2.03	0.79	6.56	0.01*	0.13	0.03	0.62
LESS THAN AVERAGE	-0.88	0.79	1.24	0.27	0.41	0.09	1.95
AGE	-0.02	0.01	1.90	0.17	0.98	0.96	1.01
FEMALE	0.61	0.12	23.99	<0.001*	1.85	1.43	2.33
GTC SEIZURES	0.22	0.15	1.99	0.16	1.25	0.93	1.69
FAMILY HISTORY OF SEIZURES	-0.36	0.14	6.50	0.01*	0.70	0.53	0.93
HISTORY OF FEBRILE CONVULSION	-0.06	0.15	0.16	0.69	0.94	0.70	1.27
HISTORY OF STATUS EPILEPTICS	0.14	0.28	0.26	0.61	1.15	0.67	1.96
SIGNIFICANT HEAD TRAUMA	-0.9	0.27	11.14	<0.001*	0.41	0.24	0.69
SIGNIFICANT POSTNATAL	-0.85	0.62	1.90	0.17	0.43	0.13	1.43

INFECTION							
CONSANGUINEOUS MARRIAGE	0.05	0.13	0.17	0.68	1.05	0.83	1.35
DELAYED DEVELOPMENTAL HISTORY	0.45	0.53	0.73	0.40	1.56	0.56	4.35
EPILEPTIC SYNDROME	1.33	0.17	59.96	<0.001*	3.85	2.70	5.26
BIRTH ASPHYXIA	-0.32	0.44	0.52	0.47	0.72	0.30	1.72
EXAMINATION FINDINGS (ABNORMAL)	0.93	0.35	6.99	0.01*	2.56	1.27	5.00
NORMAL EEG	-0.38	0.14	7.43	0.01*	0.68	0.52	0.89
NORMAL MRI FINDING	-1.4	0.23	37.26	<0.001*	0.25	0.16	0.39
BEHAVIORAL ABNORMALITIES	1.61	0.21	60.77	<0.001*	5.00	3.33	7.69

CI, CONFIDENCE INTERVAL; OR, ODDS RATIO; LINK FUNCTION, COMPLEMENTARY LOG-LOG; PARALLEL LINE TEST (P = 0.1)

Table 6. Binary logistic regression for the intact development and educational outcome among epileptic pediatric patients

INDEPENDENT VARIABLES	BETA	S.E.	WALD	P-VALUE	OR	95% CI	
FEMALE	-0.97	0.28	11.88	0.001*	0.38	0.22	0.66
AGE	0.02	0.04	0.24	0.622	1.02	0.95	1.09
GTC	0.29	0.31	0.86	0.355	1.34	0.72	2.47
FAMILY HISTORY OF SEIZURES	1.29	0.32	16.05	<0.001*	3.64	1.94	6.86
HISTORY OF FEBRILE CONVULSION	0.00	0.00	0.38	0.538	1.00	0.99	1.01
AGE OF ONSET OF SEIZURES	-0.47	0.33	2.11	0.146	0.62	0.33	1.18
EPILEPTIC SYNDROME	-3.26	0.40	66.41	<0.001*	0.04	0.02	0.08
PRECIPITATING INFECTION	-0.70	0.30	5.63	0.018*	0.50	0.28	0.89
NEUROLOGICAL EXAMINATION ABNORMAL	-3.36	0.68	24.15	<0.001*	0.04	0.01	0.13
NORMAL EEG FINDING	0.82	0.29	7.88	0.005*	2.26	1.28	4.00
NORMAL MRI FINDING	3.17	0.48	43.00	<0.001*	23.82	9.23	61.45
CONSTANT	-1.95	0.60	10.62	0.001*	0.14		

BETA, REGRESSION COEFFICIENT; GTC, GENERALIZED TONIC COLONIC; SE, STANDARD ERROR; *, SIGNIFICANT P-VALUE ≤ 0.05.

4. Discussion

Our study highlights critical insights into the characteristics, comorbidities, and developmental outcomes associated with epilepsy in the paediatric population. The findings emphasize significant differences between controlled and uncontrolled epilepsy groups. Our data revealed that consanguinity, positive family history, prenatal infection, history of febrile seizures, focal seizures, normal neurological examination, normal EEG, normal MRI, patients' age, intact development, and favourable educational outcomes were associated with epilepsy control. On the other hand, birth asphyxia, delayed developmental history, postnatal infections, GTC, the number of antiseizure medications required, and the duration of antiepileptic drug use were associated with uncontrolled epilepsy.

Furthermore, our study revealed predictors of seizure control and illustrated that female sex, age, GTC seizures, status epilepticus, precipitating infections, abnormal neurological examination, abnormal EEG, and abnormal MRI were independent predictors of uncontrolled epilepsy.

Having the educational performance, female sex, significant head trauma, epileptic syndromes, abnormal neurological examination, precipitating infections, and behavioural abnormalities significantly increased the likelihood of educational and developmental impairment. On the other hand, normal EEG and MRI increased the likelihood of normal development and educational performance.

The prevalence of GTC seizures, status epilepticus, and epileptic syndromes was markedly higher among patients with uncontrolled seizures, corroborating existing literature on the association between these factors and treatment resistance.[11,12] Consanguinity, a notable finding in our cohort, was more frequent in controlled epilepsy, suggesting a complex genetic interplay influencing seizure control.[13] Conversely, the absence of infections during pregnancy in the uncontrolled group may highlight prenatal factors with a potentially protective role.[14]

The logistic regression analysis identified female gender, abnormal neurological examination, and precipitating infections as significant predictors of uncontrolled epilepsy. These findings align with prior research indicating that structural brain abnormalities, as evidenced by abnormal neurological findings, and recurrent febrile illnesses exacerbate seizure recurrence and severity.[15,16] In contrast, normal MRI and EEG findings significantly increased the likelihood of achieving seizure control, supporting their diagnostic and prognostic utility.[17,18]

Epilepsy exerts a profound impact on cognitive and educational outcomes. In our study, 40.7% of children exhibited intact development and education, whereas 39.4% had impaired outcomes. Female sex, epileptic syndromes, behavioural abnormalities, and abnormal neurological examinations were strongly associated with poorer developmental outcomes.[19,20] Conversely, normal MRI and EEG findings were highly protective, highlighting the critical role of neuroimaging and electrophysiological assessments in early intervention.[17,21]

Family history of seizures was linked to favourable educational outcomes, a finding

potentially explained by heightened familial awareness and proactive management.[22] Similarly, significant head trauma was associated with lower impairment, possibly reflecting the influence of early medical intervention in trauma-related epilepsy.[23]

The ordinal logistic regression model reinforced the role of epileptic syndromes, behavioural abnormalities, and abnormal examination as key determinants of developmental impairment.[24] These results emphasize the need for comprehensive, multidisciplinary approaches that integrate medical, psychological, and educational support to mitigate the long-term effects of epilepsy on children.[15,25]

Our study underscores the importance of early diagnosis, tailored treatment strategies, and continuous developmental monitoring for children with epilepsy. Advances in neurogenetics and imaging offer promising avenues for precision medicine, potentially transforming the management of refractory epilepsy.[26] Moreover, educational interventions, including individualized education programs and psychoeducational support, are essential to address the cognitive and behavioural challenges faced by this vulnerable population. [27,28]

This study's cross-sectional design limits causal inferences regarding the associations observed. Additionally, data from a single tertiary centre may not be generalizable to broader populations. Longitudinal studies are warranted to explore the dynamic evolution of epilepsy and its impact on development.[11,19]

4. Conclusion

The study highlights significant predictors of seizure control and developmental outcomes in paediatric epilepsy. Factors such as abnormal neurological examinations, epileptic syndromes, and behavioural abnormalities are critical determinants of prognosis. Early intervention, guided by comprehensive clinical and neuroimaging assessments, can enhance quality of life and educational attainment in children with epilepsy.

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References

1. Fisher, R. S., Acevedo, C., Arzimanoglou, A., et al. ILAE official report: a practical clinical definition of epilepsy. Epilepsia, 2017; 58(4), 475-482.
2. Berg, A. T., Berkovic, S. F., Brodie, M. J., et al. Revised terminology and concepts for organization of seizures and epilepsies. Epilepsia, 2010; 51(4), 676-685.
3. Camfield, C. S., & Camfield, P. R. Pediatric epilepsy: an overview. Epilepsia, 2003; 44(s1), 2-7.
4. Wirrell, E. Predicting pharmaco-resistance in paediatric epilepsy. Epilepsia, 2003; 44(s1), 32-35.
5. Liu, S., Yu, D. F., & Ding, M. C. Recent advances in neuroimaging and neurogenetics of epilepsy. Chinese Medical Journal, 2014; 127(19), 3515-3521.
6. Cross, J. H., Caraballo, R. H., Caminha, R. C., et al. Epileptic encephalopathies of childhood: clinical features and pathways for diagnosis and treatment. The Lancet Neurology, 2017; 16(6), 494-503.
7. Berg, A. T., Shinnar, S., Levy, S. R., et al. How well can epilepsy syndromes be identified at diagnosis? Epilepsia, 2017; 51(12), 2420-2430.
8. Frankenburg, W. K., & Dodds, J. The Denver developmental screening test. The Journal of pediatrics. 1967 Aug 1;71(2):181-91.
9. Roid, G. H., & Barram, R. G. Book review: Stanford-binet intelligence scales. Journal of Psychoeducational Assessment. 2005 Mar;23(1):87-95.
10. Fisher, R. S., Acevedo, C., Arzimanoglou, A., et al. ILAE Official Report: A practical clinical definition of epilepsy. Epilepsia, 2014; 55(4), 475-482.
11. Roid, G. H., & Barram, R. G. How well can IQ be measured? Stanford-Binet Intelligence Scales (5th ed.). Riverside Publishing, 2004, 85-95.
12. Wirrell, E. C. Cognitive and behavioral impact of epilepsy in children. Journal of Child Neurology, 2016; 31(2), 158-168.
13. Kwan, P., & Brodie, M. J. Early identification of refractory epilepsy. The New England Journal of Medicine, 2000; 342(5), 314-319.
14. Guerrini, R. Epilepsy in children. The Lancet, 2006; 367(9509), 499-524.
15. Helmstaedter, C., & Elger, C. E. Chronic epilepsy and cognition. The Lancet Neurology, 2009; 8(3), 233-243.
16. Bouma, P. A., & Schuit, F. The prognosis of childhood epilepsy. Epilepsia, 2004; 45(s1), 31-37.
17. Sillanpää, M., & Schmidt, D. Natural history of treated childhood-onset epilepsy: prospective, long-term population-based study. Brain, 2006; 129(3), 617-624.
18. Shinnar, S., & Pellock, J. M. Update on the epidemiology and prognosis of pediatric epilepsy. Journal of Child Neurology, 2002; 17(suppl 1), S4-S17.
19. Jallon, P., & Latour, P. Epidemiology of idiopathic generalized epilepsies. Epilepsia, 2005; 46(s9), 10-14.
20. Berg, A. T., & Vickrey, B. G. Developing needs and treatment approaches for cognitive and behavioral issues in children with epilepsy. Epilepsy & Behavior, 2008; 12(4), 638-648.
21. Lagae, L. Cognitive side effects of anti-epileptic drugs. Epilepsia, 2006; 47(s2), 49-52.
22. Sander, J. W. The epidemiology of epilepsy revisited. Current Opinion in Neurology, 2003; 16(2), 165-170.
23. Gilliam, F. G. Optimizing health outcomes in active epilepsy. Neurology, 2002; 58(8 Suppl 5), S9-S20.
24. Dodrill, C. B. Neuropsychological effects of seizures. Epilepsia, 2004; 45(s2), 21-26.
25. Rantanen, K., Eriksson, K., & Nieminen, P. Cognitive impairment in childhood epilepsy with normal imaging. Epilepsy & Behavior, 2011; 21(4), 592-597.
26. Aldenkamp, A. P., & Arends, J. The effect of epilepsy and epileptic seizures on cognitive function. Clinical Neurology and Neurosurgery, 2004; 106(1), 19-23.
27. Meador, K. J. Cognitive outcomes and predictive factors in epilepsy. Neurology, 2006; 67(12 Suppl 4), S20-S26.
28. Reilly, C., & Atkinson, P. Educational interventions for children with epilepsy. Cochrane Database of Systematic Reviews, 2012; 6, CD006245.