Original Article

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EEG and Radiological Findings Associated with Drug Resistant Epilepsy in Bangladeshi Children

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Abstract:

Background: Drug-resistant epilepsy (DRE) affects nearly one-third of children with epilepsy and is associated with significant neurocognitive and psychosocial morbidity. Electroencephalography (EEG) and neuroimaging provide essential insights into its underlying etiologies and predictors.

Methods: This case–control study was conducted at the Institute of Paediatric Neurodisorder and Autism (IPNA), BSMMU, Dhaka, from April 2022 to March 2023. Sixty children aged 1–18 years were enrolled, including 30 with DRE (case) and 30 with drug-responsive epilepsy (control).

Results: Abnormal EEG was present in all DRE cases versus 70% of controls (p=0.002). Diffuse background slowing (86.7% vs. 10%), multifocal discharges (30% vs. 3.3%), and paroxysmal fast activity (20% vs. 3.3%) were significantly more frequent in DRE. Neuroimaging revealed abnormalities in 73.3% of DRE children, including cortical atrophy, encephalomalacia, infarction, and malformations, compared to 16.7% of controls (p=0.003).

Conclusion: Pediatric DRE is strongly associated with severe EEG abnormalities and structural brain lesions. Early recognition of these features may facilitate timely referral, tailored interventions, and improved outcomes in resource-limited settings.

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Introduction:

Epilepsy is one of the most common chronic neurological disorders in children worldwide, imposing a substantial disease burden. Approximately 50 million people globally have epilepsy, with nearly 80% living in low- and middle-income countries[1].

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In Asia alone (~4.5 billion people) there are an estimated 23 million epilepsy patients. Children bear a disproportionate share of this burden: worldwide over 1.2 million new cases of epilepsy occur in children each year, and children account for more than 90% of newly diagnosed epilepsy[2].

DRE in children is associated with significant morbidity: recurrent seizures in this population are linked to neurocognitive decline, developmental delay, physical injury, psychosocial dysfunction, and increased risk of early mortality[2]. Moreover, in resource-limited settings epilepsy carries stigma and treatment gaps: up to 90% of epilepsy patients in parts of Asia and Africa do not receive adequate therapy. Thus, understanding factors that distinguish DRE from drug-responsive epilepsy in children is of critical clinical importance.

Electroencephalography (EEG) and neuroimaging (primarily MRI) are key diagnostic tools in pediatric epilepsy, often revealing underlying etiologies. EEG recordings frequently show epileptiform discharges even in new-onset epilepsy, and certain EEG patterns have been associated with higher risk of intractability[3]. For example, studies have found that diffuse background slowing, high spike-wave frequency, multifocal discharges, or paroxysmal fast activities on EEG are more common in drug-resistant cases[3]. Brain MRI often identifies structural lesions that cause epilepsy. In many pediatric epilepsy series, children with DRE have a much higher prevalence of imaging abnormalities (e.g. cortical malformations, encephalomalacia, atrophy) compared to drug-responsive cases[4]. In a Tanzanian cohort, for example, about 80% of children with DRE had identifiable MRI lesions (most commonly cystic encephalomalacia) versus only ~25% of drug-responsive peers[4]. Likewise, an Egyptian pediatric study reported abnormal MRI in ~88% of drug-resistant versus 27% of controlled epilepsy cases. Such findings underscore that many children with refractory seizures have an identifiable cause, often from perinatal hypoxic-ischemic injury, malformations of cortical development (MCD), or other insults[5].

Despite these insights, data on EEG and imaging correlates of pediatric DRE in South Asia and other low-resource regions are sparse. Most published series come from Europe, North America, or higher-resource settings[3], with few reports from Bangladesh or neighboring countries. In Bangladesh, available studies indicate that childhood epilepsy outcomes are often poor: for example, one Dhaka series found poor seizure remission in half of children over time, with multiple seizure types and EEG abnormalities predicting worse control. Yet there is limited systematic information about the EEG patterns or neuroimaging findings that differentiate DRE from drug-responsive epilepsy among Bangladeshi children. Likewise, South Asian pediatric neurologists have noted challenges: many children present late with advanced disease, and few have ready access to MRI or advanced care. Moreover, newer therapies (surgery, ketogenic diet, neuromodulation) are rarely used in this region[6], making it all the more important to identify early which children may become drug-resistant so that interventions can be considered.

Given these gaps, our study aimed to characterize and compare EEG and radiological findings in children with DRE versus drug-responsive epilepsy in Dhaka, Bangladesh. We conducted a case-control study of 60 children (age 1–18 years) at BSMMU, including 30 with DRE and 30 with well-controlled epilepsy. We analyzed EEG tracings for epileptiform and background abnormalities, and MRI (or CT) scans for structural lesions. By comparing the prevalence and types of EEG and imaging abnormalities between groups, we sought to identify features that may predict or accompany drug resistance in our setting. This work addresses a critical need for local data on pediatric DRE: it may help clinicians recognize high-risk children early, guide further investigations, and ultimately improve management in resource-limited South Asian contexts.

Materials and methods

This case-control study was conducted in the Department of Pediatric Neurology (outpatient and inpatient services), Institute of Paediatric Neurodisorder and Autism (IPNA), Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka. The study period extended over twelve months, from April 2022 to March 2023. A total of 60 children with epilepsy were enrolled, comprising 30 children with drug-resistant epilepsy (cases) and 30 children with drug-responsive epilepsy (controls).

Children aged 1–18 years who met the diagnostic criteria for drug-resistant epilepsy, as defined by the International League Against Epilepsy (ILAE) 2017, were included in the case group. Age-matched children with drug-responsive epilepsy were recruited as controls. Patients with incomplete clinical information or poor treatment compliance were excluded. For each participant, detailed data were collected regarding demographic characteristics, age at seizure onset, seizure semiology and treatment history. All children underwent comprehensive neurological examination, developmental assessment, electroencephalography (EEG), and neuroimaging where indicated.

Data were collected using a structured questionnaire and supplemented with information from parents or primary caregivers. The collected data were coded, checked, and analyzed using SPSS version 24. Quantitative variables were expressed as mean ± standard deviation (SD) and compared between groups using the Student's t-test. Categorical variables were presented as frequencies and percentages, analyzed with Chi-square test or Fisher's exact test where appropriate. A p-value < 0.05 was considered statistically significant.

Ethical approval was obtained from the Ethical Review Committee of BSMMU prior to commencement of the study. Written informed consent was obtained from parents or legal guardians of all participants, ensuring voluntary participation, confidentiality, and the right to withdraw at any stage.

Result

A total of 60 children with epilepsy were included, with 30 in the drug-resistant group (case) and 30 in the drug-responsive group (control). The mean age at presentation was 4.83 years in the drug-resistant group and 6.97 years in the drug-responsive group. The mean age of onset was 1.91 years in the drug-resistant group compared to 4.31 years in the drug-responsive group. Males constituted 56.7% of the drug-resistant and 63.3% of the drug-responsive group. A family history of epilepsy was present in 10% of drug-resistant and 6.7% of drug-responsive cases, while consanguinity was observed in 16.7% and 6.7% respectively (Table I).

All children in the drug-resistant group had abnormal EEG findings compared to 70% in the drug-responsive group. Abnormal background activity was seen in 86.7% of drug-resistant children and 10% of drug-responsive children, with generalized slowing being the most frequent abnormality. Multifocal epileptiform discharges (30% vs 3.3%) and paroxysmal fast activity (20% vs 3.3%) were more frequent in drug-resistant cases, while generalized epileptiform discharges were more frequent in the drug-responsive group (46.7% vs 20%) (Table II).

Neuroimaging showed abnormal findings in 73.3% of the drug-resistant group compared to 16.7% of the drug-responsive group. Cortical atrophy (54.5%), cystic encephalomalacia (18.2%), infarction (13.6%), cerebral hemiatrophy (13.6%), ventricular dilatation (13.6%), and other structural abnormalities such as lissencephaly, tuberous sclerosis, and corpus callosal agenesis were observed among drug-resistant children. In contrast, the majority of drug-responsive children (83.3%) had normal neuroimaging (Table III).

Table I : Demographic profile of children with Drug resistant and responsive epilepsies

	Characteristics	Frequency in Drug resistant epilepsy group (n=30) (%)	Frequency in Drug responsive epilepsy group (n=30) (%)	P value
Age at presentation (Years)	Mean age	4.83	6.97	0.023*
	Standard Deviation	±3.12	±3.93	
Age of onset (Years)	Mean age	1.91	4.31	0.005*
	Standard Deviation	±2.31	±3.86	
Sex	Male	17 (56.7%)	19 (63.3%)	0.598**
	Female	13 (43.3%)	11 (36.7%)	
Family History	Epilepsy	3 (10%)	2 (6.7%)	0.640**
	Consanguinity	5 (16.7%)	2 (6.7%)	0.228**

** Chi-squared Test (χ^2) was performed.
* Student t-test was performed.

Table II: EEG findings in children with Drug resistant and responsive Epilepsies

Findings		Frequency in Drug resistant epilepsy group (n=30) (%)	Frequency in Drug responsive epilepsy group (n=30) (%)	P value
Abnormal EEG		30 (100%)	21 (70%)	0.002*
Abnormal Background		26 (86.66%)	3 (10%)	<0.001*
Abnormal Background	Generalized slowing	16 (61.5%)	2 (66.7%)	0.027*
	Focal slowing	4 (15.4%)	1 (33.3%)	0.349*
	Others (asymmetry, asynchrony, giant delta activity)	6 (23.1%)	0 (0%)	0.025*
Epileptiform discharges	Focal epileptiform discharge	11(36.7%)	5(16.7%)	0.143*
	Multifocal epileptiform discharge	9 (30%)	1 (3.3%)	0.012*
	Generalized epileptiform discharge	6(20%)	14 (46.7%)	0.054*
	Burst suppression pattern	3 (10%)	0 (0%)	0.782*
	ESES	3 (10%)	0 (0%)	0.782*
	Paroxysmal fast activity	6 (20%)	1 (3.3%)	0.043*

*p value was determined by Chi-squared Test (χ^2) and Fisher-exact test

Table III: Neuroimaging findings in children with Drug resistant and responsive Epilepsy

	Findings	Frequency in Drug resistant epilepsy group (n=30) (%)	Frequency in Drug responsive epilepsy group (n=30) (%)	P value
Normal		8 (26.7%)	25 (83.33%)	0.003*
Abnormal	Cortical atrophy	12(54.54%)	2 (40%)	0.008*
	Cystic encephalomalacia	4(18.18%)	0(0%)	
	Infarction	3 (13.63%)	1 (20%)	
	Calcification	2 (9.1%)	0(0%)	
	Lissencephaly	2 (9.1%)	0(0%)	
	Corpus callosal agenesis	2 (9.1%)	0(0%)	
	Arachnoid cyst	1 (12.5%)	0(0%)	
	Tuberous sclerosis	2 (9.1%)	0(0%)	
	Ventricular dilatation	3 (13.63%)	2 (40%)	
	Basal ganglia hyperintensity	2 (9.1%)	0(0%)	
	Cerebral hemiatrophy	3(13.63%)	0(0%)	

*p value was determined by Chi-squared Test (χ^2) and Fisher-exact test

Discussion:

In this case-control study, Bangladeshi children with drug-resistant epilepsy (DRE) differed markedly from drug-responsive group in clinical onset, EEG, and imaging. The DRE group had a much younger mean age at seizure onset (1.9 vs. 4.3 years, p=0.005), all had abnormal interictal EEGs (100% vs. 70%, p=0.002), and showed far more diffuse background slowing (86.7% vs. 10%, p<0.001), multifocal epileptiform discharges (30% vs. 3.3%, p=0.015), and paroxysmal fast activity (20% vs. 3.3%, p=0.043) on EEG. Neuroimaging was also strikingly different: 73.3% of DRE children had abnormalities on MRI versus only 16.7% of controls (p=0.003), predominantly cortical atrophy and focal structural lesions (e.g. hypoxic-ischemic encephalopathy, malformations). These findings are summarized in Table III and suggest more widespread and severe brain pathology in DRE.

Our results align well with prior literature on pediatric DRE. Early age of onset has long been recognized as associated with refractory epilepsy; in our data mean onset was under 2 years in the DRE group. Similarly, Yıldırım et al. found that over one-third of children with epilepsy onset under age 2 developed medical intractability[7]. Symptomatic (structural/ metabolic) etiologies are repeatedly linked to DRE. For example, Mangunatmadja et al. reported that symptomatic etiology (e.g. encephalomalacia, cortical malformations) was a very strong predictor of resistance (adjusted OR ≈84)[8]. Early structural brain injury or dysplasia likely underpins both the early onset and poor response. In congenital or acquired brain lesions, epileptogenesis is more diffuse and aggressive, favoring refractoriness[8,9] . This is consistent with our finding of a very young DRE cohort and high rate of structural MRI lesions.

We observed 100% abnormal EEG in DRE vs 70% in responsive. Meta-analyses have identified EEG abnormalities as a major risk factor for DRE – studies report that any interictal slowing or epileptiform EEG confers an ~2.8-fold increased risk of resistance[10]. Abnormal EEG is thus a validated predictor of medical intractability. In DRE the nature of EEG findings also differs: diffuse slowing and multifocal discharges are much more common than in responders. Abokrysha et al. found diffuse background slowing in 61.5% of DRE children vs only 1.9% of controls, and multifocal spikes in 25% vs 1%[11]. In our study, diffuse slowing occurred in 86.7% of DRE vs 10% of controls, and multifocal discharges in 30% vs 3.3%. These striking differences parallel the Egyptian data, underscoring that background slowing – a marker of diffuse cerebral dysfunction – is strongly linked to refractory epilepsy. Diffuse slowing reflects global encephalopathy or multifocal pathology, and it is rarely seen in benign, drug-responsive cases[12].

Paroxysmal fast activity (PFA) was significantly more frequent in our DRE group (20% vs 3.3%, p=0.043). PFA – rapid beta-frequency bursts on the EEG – is a recognized marker of active epileptogenesis. In children, Wu et al. reported PFA in about 16% of all EEGs and in 28% of those with epilepsy, and found it highly predictive of true epilepsy (97% positive predictive value)[13]. The presence of PFA likely indicates an epileptic network that is deeply embedded and difficult to suppress with medication.

Neuroimaging findings similarly distinguished the groups. We found abnormal MRI in 73.3% of DRE children vs 16.7% of controls

(p=0.003). Prior pediatric studies report comparable or even larger contrasts. Abokrysha et al. noted an 88.5% abnormal MRI rate in DRE vs 27.2% in well-controlled epilepsy[11]. Khosronejad et al. (Iran) found abnormal MRI features (including cortical dysplasia, porencephaly, atrophy, hippocampal sclerosis) in 70.8% of intractable cases vs only 7.5% of responsive cases[14]. Likewise, Flores-Sobrecueva et al. reported 87% abnormal MRI in pediatric DRE versus 52% in controls[15]. Our result of is fully consistent with these. The types of lesions also match: cortical atrophy (often cerebellar or diffuse) and encephalomalacic scars were particularly common in DRE. This pattern suggests that longstanding, diffuse brain injury (e.g. hypoxic-ischemic damage) contributes to refractoriness. By contrast, focal lesions localized to one hemisphere (e.g. mesial temporal sclerosis) may be more amenable to resection and thus associated with potential seizure freedom.

In sum, our findings reinforce known EEG and imaging correlates of drug resistance. Patients with DRE tend to be younger at onset, have structural etiologies, and show EEG markers of widespread dysfunction. Abnormal EEG features – especially background slowing and multifocal epileptiform discharges – have repeatedly been associated with resistance.

Study Limitations

The sample size was relatively small (n=60) and drawn from a single tertiary center, so findings may not generalize to all Bangladeshi children. The retrospective design means we depended on chart data and a single EEG report per patient; subtle or evolving EEG features may have been missed. Selection bias is also possible, since children referred to a specialty clinic likely have more severe or unusual epilepsy, inflating differences. Genetic causes were not assessed, and advanced imaging (e.g. 3T MRI) was not uniformly available, so the true prevalence of subtle lesions may be higher. Despite these limitations, the strong statistical differences and consistency with international data lend confidence to our conclusions.

Conclusion

DRE cases have distinct clinical, EEG and MRI features: younger onset, diffuse background slowing, multifocal discharges, fast activity bursts, and frequent cortical lesions. These findings echo prior studies and emphasize that early developmental or structural brain insults underlie drug resistance. Recognizing these hallmarks in clinical practice should prompt expedited referral and tailored management. Ultimately, improving access to EEG and imaging and applying these predictive markers may help close the treatment gap and improve outcomes for children with epilepsy in low-resource settings.

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