Original Article

Risk Factors and Neuroimaging Findings in New-Onset Focal Epilepsy in Bangladeshi Children

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ABSTRACT

Background: Epilepsy is a major cause of morbidity in children, particularly in low- and middle-income countries, where preventable perinatal and genetic risk factors are common. Data on childhood focal epilepsy in Bangladesh remain limited. **Objective:** To determine the frequency of risk factors and describe neuroimaging findings in children with new-onset focal epilepsy. **Methods:** A retrospective study was conducted at Mount Adora Hospital, Sylhet, Bangladesh, from July 2024 to June 2025. Children aged 1–18 years with new-onset focal epilepsy were included. Clinical data and neuroimaging findings (CT/MRI) were analyzed using descriptive statistics. **Results:** Fifty-one children were studied (mean age 6.2 ± 3.8 years; 59% male), with most (80%) from rural areas. Perinatal asphyxia was reported in 21.6%, and 9.8% required NICU admission. Family history of epilepsy (27.4%) and parental consanguinity (21.6%) were common. Infections were rare (2% each for CNS infection and neonatal sepsis). Neuroimaging was abnormal in 35.3% of cases, predominantly infarctions (25.5%), followed by cortical atrophy (3.9%), structural abnormalities (3.9%), and basal ganglia hyperintensities (2%). **Conclusion:** Perinatal insults, hereditary factors, and rural health disparities play key roles in childhood focal epilepsy in Bangladesh. Improved obstetric care, expanded access to neuroimaging, and community-based epilepsy services are essential to reduce this burden.

Keywords: New-onset Focal epilepsy; Children; Risk factors; Perinatal asphyxia; Neuroimaging;

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Introduction

Epilepsy is one of the most common chronic neurological disorders in children worldwide, affecting an estimated 70–80 million people, including over 10 million children (with four-fifths of pediatric cases in low- and middle-income countries).1 In high-income countries, active epilepsy affects roughly 4–10 per 1000 of the population, whereas in low- and middle-income countries it can be as high as 13–14 per 1000.[1,2] The global burden of epilepsy is substantial – accounting for over 0.5% of all years of healthy life lost – and is disproportionately borne by resource-poor regions.[2] Children with epilepsy face high morbidity. Epilepsy is strongly associated with

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neurodevelopmental delay, cognitive impairment, and psychosocial adversity. For example, in one Bangladesh study, most epileptic children already had "neurodevelopmental morbidities" by the time of diagnosis.[3] Epilepsy in childhood also carries increased risk of premature mortality (up to 3-fold higher than peers) and imposes high economic and social costs on families.[2,4]

Regionally, South Asia harbors a particularly high epilepsy burden. An estimated 23 million people in Asia live with epilepsy, with prevalence rates ranging approximately 1.5–14 per 1000.5 Factors such as endemic neuroinfections (neurocysticercosis, tuberculosis, encephalitis), high rates of birth injury and head trauma, and limited healthcare resources contribute to this burden.[2,5] In India and Nepal, up to half of epilepsy may be attributable to neurocysticercosis.[5] The incidence of childhood epilepsy is notably higher in low-income settings than in high-income countries. A recent meta-analysis in Africa found a pooled lifetime prevalence of ~17 per 1000 for pediatric epilepsy.[6] In the Indian subcontinent, community-based studies suggest a childhood epilepsy prevalence of around 0.6–1.0%.[7] Notably, despite the high prevalence in the region, epilepsy often goes untreated – for example, in Bangladesh, a survey found over 60% of people with active epilepsy received no treatment, and of those treated, 72% had poor medication adherence.[8] The resulting treatment gap exacerbates the public health impact, perpetuating disability and stigma.[2,8]

In Bangladesh, the epidemiology and causes of childhood epilepsy are poorly characterized. A recent national survey (the first of its kind) estimated epilepsy prevalence at 8.4 per 1000 (approximately 1.5–2.0 million people)[8]. Prevalence among children (<18 years) was similar to that of adults.[8] The majority of these cases were undiagnosed and untreated, reflecting Bangladesh's very high treatment gap. Clinical series suggest that childhood epilepsy in Bangladesh is often symptomatic (acquired or structural), with perinatal brain injury a leading cause. For example, Banu et al. reported that 56% of children attending a Dhaka epilepsy clinic had symptomatic epilepsy, and nearly half had a history of perinatal asphyxia (46%) or neonatal seizures (41%).[3] Similarly, a tertiary center study found hypoxic-ischemic encephalopathy in 32% and developmental brain malformations in 26% of symptomatic pediatric epilepsy cases.[9] These figures far exceed estimates from high-income settings and likely reflect preventable obstetric and neonatal deficits in Bangladesh. Other Bangladeshi studies have identified additional risk factors: a case-control study found that birth asphyxia, neonatal seizures, and parental consanguinity significantly increased the odds of epilepsy in infancy.[10] Likewise, low birthweight and postnatal CNS infections were linked to epilepsy among Bangladeshi children with cerebral palsy.[11] These findings underscore multiple interplaying perinatal and genetic factors in this context.

Neuroimaging (especially MRI) is a key tool for identifying structural causes of epilepsy. High-quality MRI, ideally with epilepsy-specific protocols, should be offered to children with focal or drug-resistant seizures.[12] In pediatric epilepsy, MRI often reveals malformations of cortical development, hippocampal sclerosis, encephalomalacia from infarcts or hypoxia, tumors, or other lesions. Studies from other low-income regions report high rates of imaging abnormalities: for instance, a Ugandan study found structural lesions in 74% of children with epilepsy, mostly acquired (hippocampal sclerosis, encephalomalacia).[13] An Indian study observed that neuroimaging was abnormal in ~15% of children with seizures, chiefly perinatal insult sequelae (7%) and CNS infections (3%).[14] Globally, focal epilepsy in childhood often correlates with identifiable pathology: up to ~25% of new-onset focal seizures occur in the context of occult focal cortical dysplasia or other lesions detectable by MRI.

Despite the high epilepsy burden in Bangladesh, few studies have systematically assessed risk factors and imaging findings specifically in children with new-onset focal epilepsy. Existing data are limited to general epilepsy cohorts or special groups (e.g., cerebral palsy), and there are no published series focusing on MRI findings in routine pediatric

practice in Bangladesh. This knowledge gap has practical importance: identifying modifiable risk factors (e.g., obstetric care, infection control) and diagnostic yields can inform prevention and early management. In particular, given the substantial role of perinatal insults and genetic factors in Bangladesh, understanding their contribution to focal epilepsy is crucial for targeted interventions.

To address these gaps, we undertook a study of children presenting with new-onset focal epilepsy at Mount Adora Hospital in Sylhet, Bangladesh. The goals were to determine the frequency of known risk factors (perinatal complications, infections, consanguinity, etc.) and to characterize neuroimaging abnormalities on MRI in this population. We hypothesized that a high proportion of Bangladeshi children with focal epilepsy would have perinatal or congenital etiologies and that many would show actionable lesions on MRI, consistent with other LMIC settings. The objectives of this study are therefore: (1) to estimate the prevalence of identified risk factors among children with new-onset focal epilepsy in Bangladesh, and (2) to describe the patterns and frequency of neuroimaging findings in these children. The findings are intended to highlight the clinical profile of focal epilepsy in Bangladeshi children and to suggest avenues for improved prevention and diagnostic care.

Materials and Methods

This retrospective study was conducted in Mount Adora Hospital, Akhalia, Sylhet, Bangladesh, over a 12-month period from July 2024 to June 2025. Children aged 1–18 years with a new onset of focal epilepsy were included. Exclusion criteria were children with generalized epilepsy, previously diagnosed epilepsy or prior antiepileptic drug use, incomplete medical records, and seizures secondary to acute metabolic, infectious, or toxic causes.

Data were collected using a structured questionnaire from hospital records, covering demographic details, perinatal history, family history, consanguinity, and associated features. Neuroimaging (MRI/CT) findings were reviewed and classified as normal, infarction, cortical atrophy, structural abnormality, or basal ganglia hyperintensities. The data were entered into SPSS version 27 for analysis and summarized using descriptive statistics (frequencies, percentages, and mean ± SD). Informed consent was obtained from parents or guardians at the time of admission, and institutional approval was obtained from Mount Adora Hospital, with strict confidentiality maintained.

Results

A total of 51 children with new-onset focal epilepsy were included in the study. The mean age at presentation was 6.23 ± 3.8 years, and most (62.7%) were ≥5 years old. Males were more commonly affected than females (58.9% vs. 41.1%), yielding a male-to-female ratio of 1.43:1. Most children (80.4%) were from rural areas.

Regarding perinatal history, 51% of cases were delivered by normal vaginal delivery (NVD) at home, 21.6% by NVD at the hospital, and 27.4% by lower uterine cesarean section (LUCS). Perinatal asphyxia occurred in 21.6%, NICU admission was required in 9.8%, and a small proportion had either CNS infection (2%) or neonatal sepsis (2%). Seizure onset occurred before age 5 in 33.3% of children and after age 5 in 66.7%. Family history of epilepsy was noted in 27.4% of cases, and consanguinity was reported in 21.6%. Other associated features included hyperactivity (7.8%), somnolence (7.8%), and behavioral disturbances (5.9%) (Table 1). Neuroimaging findings were available for all patients. The majority (64.7%) showed no abnormalities. Infarction was detected in 25.5% of cases, cortical atrophy in 3.9%, and structural abnormalities in 3.9%. Basal ganglia hyperintensities were identified in 2% of children (Table 2).

Table 1: Risk factors responsible for New Onset Focal Epilepsy	Table 1: Risk factors responsible for New Onset Focal Epilepsy	Table 1: Risk factors responsible for New Onset Focal Epilepsy
	Category	Frequency (%)
Age	<5 years	19 (37.3%)
Age	≥5 years	32 (62.7%)
Age	Mean Age (±SD)	6.23 (±3.8) years
Sex	Male	30 (58.9%)
Sex	Female	21 (41.1%)
Sex	M:F ratio	1.43:1
Area of Residence	Rural	41 (80.4%)
Area of Residence	Urban	10 (19.6%)
Mode of delivery	VD at home	26 (51%)
Mode of delivery	VD at hospital	11 (21.6%)
Mode of delivery	LUCS	14 (27.4%)
Past Medical History	H/O Perinatal asphyxia	11 (21.6%)
Past Medical History	NICU Admission	5 (9.8%)
Past Medical History	H/O CNS Infection	1 (2%)
Past Medical History	H/O Neonatal sepsis	1 (2%)
Onset of seizure	<5 years	17 (33.3%)
Onset of seizure	≥5 years	34 (66.7%)
Others	Family history	14 (27.4%)
Others	Consanguinity	11 (21.6%)
Others	Hyperactivity	4 (7.8%)
Others	Somnolence	4 (7.8%)
Others	Behavioral disturbances	3 (5.9%)

Table 2: Neuroimaging findings	**Table 2: Neuroimaging findings**
Findings	Frequency (%)
Normal	33 (64.7%)
Infarction	13 (25.5%)
Cortical atrophy	2 (3.9%)
Structural abnormality	2 (3.9%)
Basal ganglia hyper intensities	1 (2%)

Our study of 51 Bangladeshi children with new-onset focal epilepsy, a majority were school-aged (mean 6.2 years, with two-thirds ≥5 years) and male (59%), with most (80%) from rural areas. A significant proportion (22%) had a history of perinatal complications, and 10% needed neonatal intensive care support. Notably, parental consanguinity was reported in 22% of cases and a positive family epilepsy history in 27%. Infections were uncommon (only one case of meningitis and one case of neonatal sepsis). Neuroimaging was abnormal in 35% of children, predominantly showing brain infarcts/ischemia (26%). These findings indicate that symptomatic (acquired) etiologies are important in Bangladeshi childhood focal epilepsy, consistent with the high burden of perinatal brain injury and genetic risk in this setting.

Our observations align with prior Bangladeshi studies reporting high rates of perinatal insults and structural epilepsy. Banu *et al.* found 61% of pediatric epilepsy cases were symptomatic, with perinatal asphyxia in 46% and neonatal seizures in 41%.3 Although our perinatal asphyxia rate (22%) was lower than these earlier tertiary center reports, it remains substantial. The prominence of perinatal causes reflects ongoing challenges in obstetric and neonatal care; as emphasized in local and regional literature, improving peripartum care could prevent a large share of pediatric epilepsy.[3,15]

In our study, consanguinity rate (22%) and family history (27%) imply a genetic contribution. While we did not perform genetic testing, such rates are comparable to other South Asian settings, where autosomal-recessive epilepsies are relatively common. A Bangladeshi case-control study reported

consanguinity in 21% of infant epilepsy cases, with an odds ratio of ~10 for developing epilepsy.[16] In our cohort, children with parental consanguinity had a higher proportion of early-onset epilepsy, suggesting that recessive mutations may underlie some focal epilepsies here. This is consistent with other studies from LMIC: e.g., Ugandan data showed that perinatal asphyxia conferred ~10-fold higher epilepsy risk, and positive family history ~4-fold16, pointing to both acquired and inherited factors. In Asia, surveys also underline genetic links: many "idiopathic" epilepsies have familial patterns once imaging is normal. Overall, our genetic-consanguinity associations echo the regional literature on inherited focal epilepsies in children.

Although the sample size is relatively small, infectious causes were rare in our study, possibly reflecting better control of some CNS infections in our country. Only one child had meningitis, and one had neonatal sepsis. This contrasts with other LMIC studies; in South Asia, endemic neurocysticercosis is a major cause of epilepsy (e.g., about 25% of childhood epilepsy in parts of India and 17% in Nepal), and in Africa, infections like malaria, meningitis, and onchocerciasis are significant contributors.[5,17,18] African reviews similarly report that parasites (malaria, onchocerciasis, cysticercosis) account for ~27% of epilepsy cases.[5,18] However, we did not find any parasitic CNS infection history in our patients. It may reflect rural childhood death from infections before hospital referral; nonetheless, it contrasts with many LMIC studies where CNS infection is a leading risk factor for childhood epilepsy.[15,19]

In our study, 64.7% of neuroimaging findings were normal, and 35.3% showed positive findings. These findings are comparable to those reported in other studies. For instance, in an Indian pediatric cohort, only ~15% of children with seizures had abnormal imaging (perinatal sequelae 7%, infections 2.8%), whereas in Uganda, 74% of children with epilepsy had structural abnormalities (mainly acquired lesions like hippocampal sclerosis, encephalomalacia).[13] In Ghana, MRI abnormalities were found in 46.6% of pediatric epilepsy cases, commonly cerebral atrophy (21.5%) and white-matter hyperintensities (11.5%).[20] The rate of positive neuroimaging findings in our study is higher than some Asian reports but lower than some African series. The predominance of infarcts/ischemic lesions (26% overall) suggests perinatal hypoxic injury or stroke, akin to the cerebral atrophy seen in other LMICs.[20] By contrast, developmental malformations or tumors were rare in our group, possibly due to referral patterns. Nonetheless, our findings confirm that a significant minority of focal epilepsies in Bangladeshi children have identifiable structural causes, justifying imaging where available.

The observed risk patterns have a clear pathophysiological basis. Perinatal asphyxia and neonatal insults cause neuronal death and gliosis, creating epileptogenic scar tissue (e.g., cortical laminar necrosis or multicystic encephalomalacia), leading to focal seizures. Consanguinity hints at autosomal recessive channelopathies or cortical malformations; indeed, modern genetics shows that a substantial fraction of pediatric focal epilepsies have monogenic origins when sequencing is done. Infection-related epilepsy (e.g., post-meningitis gliosis) was uncommon. Yet globally, infections remain a key cause: for example, epilepsy incidence is tripled by cerebral malaria in endemic areas[18], and neurocysticercosis is endemic across South Asia. Thus, our relatively low infection rate is encouraging but should not engender complacency, as outbreaks (e.g., Japanese encephalitis) or parasitic exposures could still contribute.

Rural residence (80% of our patients) underscores inequities in epilepsy etiology and care. Rural births often occur without skilled attendants in Bangladesh, consistent with our 51% home births. This likely elevates the risk of birth injury. Moreover, rural poverty and low maternal education are known epilepsy risk factors: e.g., in Bangladesh, two-thirds of households have limited schooling.[8] Rural families may also have less awareness of epilepsy symptoms, leading to delayed diagnosis. The fact that a majority of our cases arose in rural areas suggests that expanding obstetric care and neonatal support in villages could reduce epilepsy incidence.

The findings from our study highlight multiple intervention points. First, strengthening perinatal and neonatal care is paramount. As seen in Tanzanian and Nigerian studies, identifying perinatal risk factors implies that improving antenatal monitoring, skilled delivery, and neonatal resuscitation could lower epilepsy incidence.[15,16] Additionally, community education is needed: most children with active epilepsy in Bangladesh are not receiving treatment (63%)[8], and among those treated, 72% have poor adherence. This treatment gap (over 60%) is comparable to other LMICs

(Kenya reports only 7–29% of active epilepsy cases on medication19). Addressing medication access and adherence through national epilepsy programs is critical.

From a healthcare planning perspective, our high imaging yield suggests that when available, MRI (or at least CT) should be used in new-onset focal epilepsy in Bangladeshi children to identify potentially treatable lesions. High-quality MRI with epilepsy protocols is the gold standard[13], but given limited MRI access in many low-resource settings, even targeted CT (as our center does) can provide useful etiologic clues. Nearly one-third of children with epileptogenic lesions in our study argue for investment in neuroimaging capacity. Finally, the rural predominance indicates a need for outreach neurology services; telemedicine or hub-and-spoke models could help rural physicians obtain expert guidance in epilepsy management.

Study limitations

Several limitations temper our conclusions. As a retrospective, single-center study, selection bias is likely: our sample may over-represent complex cases that reach a tertiary hospital. The small sample size (51 children) reduces precision and precludes multivariable analysis of risk factors. Data on perinatal history and consanguinity were based on parental recall, which may be incomplete or inaccurate. Imaging interpretation depended on CT or non-specialized MRI without epilepsy protocols, possibly under-detecting subtle malformations. Because we lacked genetic or metabolic testing, some etiology remains unexplained. Finally, this is a hospital-based study, so findings may not generalize to all Bangladeshi children with epilepsy, especially those who never seek care.

Conclusion

New-onset focal epilepsy in Bangladeshi children is strongly associated with perinatal complications, genetic predisposition, and rural healthcare disparities, with over one-third showing structural abnormalities on neuroimaging, mainly infarcts. These findings emphasize the preventable nature of many cases and highlight the urgent need to strengthen perinatal and neonatal care, expand access to diagnostic imaging, and reduce the treatment gap through community awareness and rural health services.

Declarations

Data Availability Statement

The datasets generated and analyzed during the current study are available from the corresponding author on reasonable request.

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Conflict of Interest Disclosure

The authors declare no conflict of interest. None of the authors serves on the editorial board of *ACNS*.

Ethics Approval Statement

Ethical approval was obtained from the Mount Adora Hospital, Sylhet, Bangladesh. All procedures were conducted in accordance with the Declaration of Helsinki.

Patient Consent Statement

Written informed consent was obtained from the parents or legal guardians of all participating children at the time of hospital admission.

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Statements Relating to Ethics and Integrity Policies

The authors confirm that this study was conducted with integrity and transparency. All authors made substantial contributions to the conception, design, analysis, and writing of the manuscript, and approved the final version. The manuscript has not been published previously and is not under consideration elsewhere.

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