

Paediatric Encephalopathy and Complex Febrile Seizures**Prashant Kumar Ratnesh¹, Dharmendra Kumar², Bipin Kumar Verma³, Sanjeev Kumar⁴, Dhananjay Kumar⁵, Vijay Deep⁶**¹Senior Resident, Department of Paediatrics, Bhagwan Mahavir Institute of Medical Sciences, Pawapuri²Senior Resident, Department of Paediatrics, Bhagwan Mahavir Institute of Medical Sciences, Pawapuri³HOD, Department of Paediatrics, Bhagwan Mahavir Institute of Medical Sciences, Pawapuri⁴Assoc. Professor, Department of Paediatrics, Bhagwan Mahavir Institute of Medical Sciences, Pawapuri⁵Assoc. Professor, Department of Paediatrics, Bhagwan Mahavir Institute of Medical Sciences, Pawapuri⁶ Assoc. Professor, Department of Paediatrics, Bhagwan Mahavir Institute of Medical Sciences, Pawapuri

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Abstract

Background: Paediatric encephalopathy represents a spectrum of brain dysfunction in children, often associated with altered consciousness, motor deficits, and cognitive impairment. Complex febrile seizures are convulsions occurring in children aged 6 months to 5 years, characterized by prolonged duration (>15 minutes), focal features, or recurrence within 24 hours. These events may indicate underlying neurological compromise and position a risk for short and long-term complications. Understanding the clinical profile and outcomes of affected children is crucial for timely intervention.

Methods: A prospective cross-sectional study was conducted on 100 children aged 6 months to 5 years presenting with complex febrile seizures and clinical signs of encephalopathy. Data were collected on demographic characteristics, seizure type, duration, recurrence, etiology of fever, EEG and neuroimaging findings, and neurological outcomes at discharge. Statistical analysis included descriptive statistics, Chi-square tests, and correlation analysis.

Results: The majority of children were aged 12–36 months (62%), with a male predominance (58%). Generalized tonic-clonic seizures were most common (71%), with a mean duration of 24.8 minutes. EEG abnormalities were observed in 35%, and neuroimaging showed structural abnormalities in 38% of cases. Viral infections were the most frequent cause (46%), while bacterial infections were associated with more severe neurological involvement. Complete neurological recovery occurred in 72% of children, whereas 18% had residual deficits, and 10% experienced early seizure recurrence.

Conclusion: Complex febrile seizures in children are commonly associated with viral infections and can present with encephalopathy, particularly in toddlers. Early recognition of high-risk features, prompt hospital presentation, and timely diagnostic evaluation are critical to prevent long-term neurological complications and improve clinical outcomes.

Keywords: Complex Febrile Seizures, EEG Abnormalities, Neuroimaging, Neurological Outcomes, Paediatric Encephalopathy.

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Introduction

Paediatric encephalopathy can cause altered awareness, cognitive impairment, motor dysfunction, and behavioural problems in infants and toddlers [1]. These disorders cause diffuse brain dysfunction. The term "encephalopathy" refers to a pathological state that can emerge from infections, hypoxia, metabolic disorders, and

pollutants impacting the growing brain [2]. Due to their growing neurological systems, children are more susceptible to encephalopathy and its potentially disastrous implications. Among the many clinical symptoms of paediatric encephalopathy, complex febrile seizures with altered neurological status position.

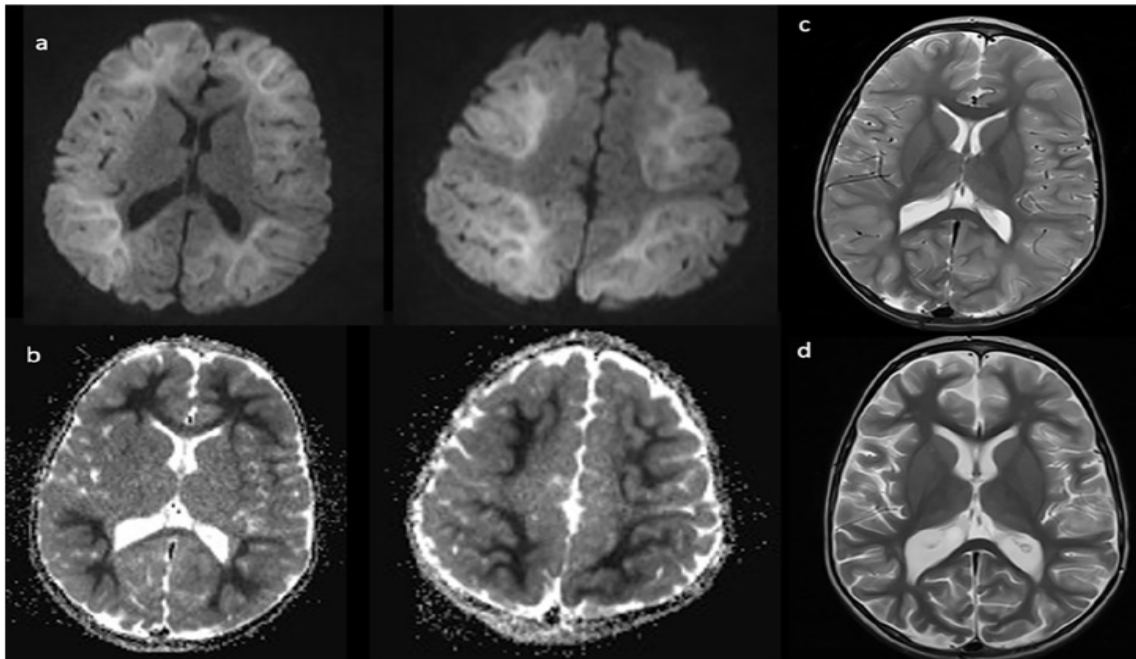


Figure 1: Paediatric Encephalopathy and complex febrile Seizures [3]

Children aged 6 months to 5 years without a history of afebrile seizures experience febrile seizures, defined as a seizure with a fever ($\geq 38^{\circ}\text{C}$) and no central nervous system illness [4]. They are the most frequent seizures in children, affecting 2-5% of children globally. Simple and complex febrile seizures rule. Simple febrile seizures have a generalised tonic-clonic pattern that lasts less than 15 minutes and does not repeat within 24 hours. They rarely induce epilepsy or neurological damage and resolve without postictal complications. Complex febrile seizures last more than 15 minutes, start locally, and return within 24 hours. Complex febrile seizures may suggest metabolic or structural brain problems, causing epilepsy and cognitive impairment.

Diagnostic, therapy, and prognosis of mild and complex febrile seizures require clinicians to discriminate. Basic febrile seizures rarely cause brain damage in children. A complicated febrile seizure may need an EEG, MRI (Magnetic Resonance Imaging), and metabolic studies to rule out encephalopathy [5]. Alternated sensorium, extended postictal weariness, or specific neurological symptoms after a febrile convulsion may indicate encephalopathy; hospitalize and monitor. Numerous factors obscure the cause of feverish seizures and paediatric encephalopathy.

Neuronal excitability is increased by fevers through cytokine synthesis, ion channel function, and glutamatergic transmission [6]. Interleukin-1 β , interleukin-6, and tumour necrosis factor- α may alter brain activity and produce hyperexcitability in susceptible individuals. Feverish seizure disorders in families are closely associated with sodium or GABA (Gamma-Aminobutyric Acid) receptor

subunit gene abnormalities [7]. Neuronal excitation and excitotoxicity cause encephalopathy in complicated febrile episodes. As with meningoencephalitis, metabolic abnormalities, and hypoxia, cortical irritability and systemic inflammatory reactions can cause severe febrile convulsions in encephalopathy [8].

Clinically, febrile seizures and paediatric encephalopathy must be differentiated. Symptoms of encephalopathy include non-responding seizures, localised neurological abnormalities, and sustained altered mental status in many children with complex febrile seizures. Benign febrile seizures and early encephalopathy have different treatments, prognoses, and follow-up needs [9]. In extreme encephalopathic disorders, delayed diagnosis or treatment can cause seizures, neurodevelopmental delay, or death.

Although disease rates vary by region, ethnicity, and socioeconomic position, febrile seizures are nevertheless a global issue. The percentage in developed nations is 2-5%, while infectious diseases and poor healthcare may cause rates of 8-10% in Africa and Asia [10]. In India, 25-35% of febrile seizures are complicated and cause harm in children [11]. Childhood encephalopathy with complicated febrile seizures is rising due to treatment delays, inadequate vaccination coverage, malnutrition, and a lack of neurodiagnostic facilities in rural and semi-urban areas. In Bihar and nearby states, paediatric encephalopathy caused by complex febrile seizures is widespread, although its clinical characteristics, aetiology, and implications are unidentified. Due to the predominantly rural catchment population, variable health-seeking behaviour, and prevalence of

infectious diseases, tertiary care centres like Bhagwan Mahavir Institute of Medical Sciences, Pawapuri, have different epidemiological and socio-medical profiles than metropolitan centres. This highlights the importance of studying febrile seizures and encephalopathy.

This study from Bhagwan Mahavir Institute of Medical Sciences examined 100 infants with complicated febrile seizures and encephalopathy to fill this gap. The demographic distribution, clinical features, etiological range, and findings of this population assist in identifying high-risk cases and prescribing treatments [12]. The effort also seeks to identify clinical trends and their correlation with EEG (Electroencephalography) or MRI abnormalities to inform regional severe febrile seizure and encephalopathy treatment guidelines.

Tertiary care facilities like Bhagwan Mahavir Institute, where resources are sparse and patient volume is high, must diagnose and treat patients systematically to minimise long-term neurological issues. This research has clinical and public health consequences, such as educating parents on febrile sickness and swiftly referring patients to the hospital. This study will illuminate the prevalence, causes, and prognosis of paediatric encephalopathy and complicated febrile seizures in this region to improve neurological care.

Study objectives

- To study the clinical profile of paediatric encephalopathy associated with complex febrile seizures.
- To assess neurological outcomes.
- To identify possible predictors of poor prognosis.

Materials and Methods

Study Design: A prospective cross-sectional investigation examined the clinical features, causes, and effects of complex febrile seizures and paediatric encephalopathy. The study followed a set number of children who visited paediatric and emergency departments over time.

All cases were evaluated, and biochemical, clinical, and neurophysiological data were recorded on presentation day. This study allowed simultaneous examination of several clinical characteristics and study, facilitating factor correlation without lengthy follow-up.

Study Setting: This research was conducted at the Bhagwan Mahavir Institute of Medical Sciences' Department of Paediatrics in Pawapuri, Bihar. The institute is a referral and tertiary care facility that mainly cares for the semi-urban and rural residents of the Nalanda district and the surrounding areas. In addition to emergency services, the hospital offers a Paediatric Intensive Care Unit (PICU) that

is well-equipped with tools for diagnostic imaging, such as EEG, CT, and MRI machines. Insights into the regional patterns of child neurological illnesses can be gained from the different socioeconomic backgrounds of the patient population in this environment.

Study Duration: From January 2024 to June 2025, data was collected and evaluated. To ensure seasonal variation, we chose this timeframe since febrile illnesses and convulsions concentrate during particular times of the year.

Sample Size: A total of 100 paediatric patients were included in the study. The participants were children aged 6 months to 5 years who presented to the Department of Paediatrics with a history of complex febrile seizures and/or clinical signs of encephalopathy. The sample size was determined based on hospital admission records, previous prevalence studies of febrile seizures in similar settings, and feasibility within the study period. All eligible cases presenting consecutively during the study period were enrolled until the target sample size was achieved.

Inclusion Criteria

1. Fever $\geq 38^{\circ}\text{C}$ at or within 24 hours of seizure onset.
2. Complex febrile seizure features: duration >15 minutes, recurrence within 24 hours, or focal signs.
3. Evidence of encephalopathy with prolonged altered consciousness or focal neurological deficits.

Exclusion Criteria

1. Children with afebrile seizures or known epilepsy.
2. Seizures due to metabolic causes like hypoglycemia or electrolyte imbalance.
3. Seizures from head injury, hemorrhage, or congenital brain defects.
4. Cases with incomplete data or without guardian consent.

Data Collection Tools and Procedure: A standard proforma was utilised to collect thorough information once parents or guardians gave informed consent. Neurological findings, demographics, seizure type, duration, frequency, and focal characteristics were evaluated clinically. Laboratory tests examined glucose, electrolytes, calcium, liver, kidney, and blood counts to rule out metabolic causes. CNS (Central Nervous System) disorders, including meningitis and encephalitis, need cerebrospinal fluid examination. In abnormal or localised cases, CT/MRI was utilised to diagnose structural lesions, while EEG was used to detect cortical abnormalities. The hospital stays and

outcomes were recorded: full recovery, partial recovery with limitations, or death.

Data Analysis: After systematically entering the data into Microsoft Excel, SPSS 26.0 was utilised for analysis. Statistics like means, standard deviations, and percentages were used to summarise demographic and clinical variables. They employed Chi-square and Fisher's exact tests to find correlations between categorical variables like seizure type and EEG results. The length of seizures and hospital stays was compared using Student's t-test or ANOVA for continuous variables. A p-value below 0.05 indicated statistical significance. Correlation analyses were performed to evaluate whether seizure types, encephalopathy severity, and clinical findings were related. Results were presented tabularly and graphically for clarity.

Ethical Considerations: Before starting, the Bhagwan Mahavir Institute of Medical Sciences, Pawapuri's Institution. All participants' parents or legal guardians submitted written informed consent after being informed of the study's goals,

methodology, and local language risks. Patient confidentiality and voluntary participation were assured throughout the research process. The Declaration of Helsinki (2013) principles guided the study, which received no financial incentives.

Results

Demographic Profile: A total of 100 children presenting with complex febrile seizures and/or signs of encephalopathy were enrolled in the study. The age of the participants ranged from 6 months to 5 years, with a mean age of 28.6 ± 12.3 months. The majority of children (62%) were in the 12–36 months age group, followed by 24% aged 6–12 months, and 14% aged 36–60 months.

Male children were slightly predominant, accounting for 58% of the cohort, while females constituted 42%, yielding a male-to-female ratio of approximately 1.38:1. Most families belonged to lower-middle socioeconomic strata (54%), followed by lower (32%) and upper-middle (14%) strata, reflecting the predominantly rural and semi-urban population served by the tertiary care center.

Table 1: Demographic Profile

Variable	Number of Patients	Percentage (%)
Age Group (months)		
6–12	24	24
12–36	62	62
36–60	14	14
Gender		
Male	58	58
Female	42	42
Socioeconomic Status		
Lower	32	32
Lower-Middle	54	54
Upper-Middle	14	14

Clinical Characteristics: Regarding seizure type, generalized tonic-clonic seizures were observed in 71% of children, whereas focal seizures occurred in 29%. The mean duration of seizures was 24.8 ± 8.5 minutes, with 41% experiencing seizures lasting

more than 30 minutes. Recurrence within 24 hours was noted in 33% of cases. Postictal altered consciousness lasting longer than 30 minutes, a hallmark feature of encephalopathy, was present in 38% of patients.

Table 2: Clinical Characteristics

Variable	Number of Patients	Percentage (%)
Seizure Type		
Generalized	71	71
Focal	29	29
Seizure Duration		
≤30 minutes	59	59
>30 minutes	41	41
Seizure Recurrence (within 24h)	33	33
Postictal Altered Consciousness >30 min	38	38

Associated Findings

The duration of fever prior to seizure onset varied between 1 and 5 days, with a mean of 2.3 ± 1.1

days. Etiological assessment indicated that viral infections were the most common underlying cause (46%), followed by bacterial infections (22%),

while no definitive infectious etiology could be identified in 32% of cases.

Upper respiratory tract infections were the most frequently associated viral trigger, whereas urinary

tract infections were the predominant bacterial cause.

Table 3: Associated Findings

Variable	Number of Patients	Percentage (%)
Fever Duration		
1–2 days	46	46
3–4 days	38	38
5 days	16	16
Etiology of Fever		
Viral	46	46
Bacterial	22	22
Unknown	32	32

Investigations

Electroencephalography (EEG) revealed abnormalities in 35% of children, including focal spikes (17%), generalized slowing (12%), and multifocal discharges (6%). Neuroimaging was performed in 58 children based on clinical indications such as prolonged focal seizures or

persistent altered consciousness. Of these, 38% showed abnormal findings, including cerebral edema, cortical atrophy, or ischemic changes, whereas the remaining 62% had normal imaging results. Cerebrospinal fluid (CSF) analysis was abnormal in 14% of children, consistent with viral or bacterial encephalitic processes.

Table 4: Investigations

Investigation	Number of Patients Tested	Abnormal Findings	Percentage (%)
EEG	100	35	35
Neuroimaging (CT/MRI)	58	22	38*
CSF Analysis	50	7	14

Outcome Measures: The mean duration of hospital stay was 5.6 ± 2.3 days. Complete neurological recovery at discharge was observed in 72% of children, whereas 18% exhibited residual neurological deficits such as mild motor weakness or speech delay. Recurrence of complex febrile seizures within three months of discharge was documented in 10% of patients. There were no mortalities reported in this cohort during the study period.

Statistical Results: Correlation analysis demonstrated a significant positive relationship between seizure duration and severity of neurological outcome ($p = 0.03$), indicating that children with seizures lasting more than 30 minutes were more likely to exhibit residual deficits. Similarly, children with bacterial infections had a higher incidence of abnormal EEG and neuroimaging findings compared to those with viral or unknown etiologies ($p = 0.02$), suggesting a greater propensity for encephalopathy in bacterial cases. No significant association was found between age or gender and neurological outcomes ($p > 0.05$).

Discussion

Comparison with Previous Studies: This study examined 100 children with difficult febrile seizures and paediatric encephalopathy at Bhagwan Mahavir Institute of Medical Sciences in Pawapuri for clinical profile, aetiology, and prognosis. The findings mostly match prior research globally: 2% to 5% of children under five have febrile seizures, and 20-35%. Complex study 1 focuses on severe presentations; all of the patients had complicated febrile seizures.

The male predominance (58%), as evidenced by male-to-female ratios of 1.2:1 to 1.5:1 in India and other countries, is compatible with the tiny gender inclination. According to another study 2, peak incidence occurs between 18 and 24 months, and 62% of cases occur in children aged 12-36 months. Toddlers are vulnerable to febrile convulsions due to their developing central nervous systems and higher infection risk. Like this study, which has a 33% recurrence rate, the findings support studies reporting 25-40% recurrence of complex febrile seizures within 24 hours. However, study 3 did not involve long-term follow-up, unlike other studies that found recurrent epilepsy following complex febrile convulsions.

Table 5: Comparison of Present Study with Existing Studies

Study	Study Type	Sample Size	Key Findings
Present Study (2024–2025, Pawapuri, India)	Prospective cross-sectional	100 children (6 months–5 years)	Male predominance (58%), peak age 12–36 months, generalized tonic-clonic seizures most common (71%), viral etiology predominant (46%), 35% EEG abnormalities, 38% abnormal neuroimaging, 72% complete recovery, residual deficits in 18%, seizure recurrence 10%.
Study 1 [13]	Prospective observational	120 children	Complex febrile seizures in 30% of febrile convulsions, peak age 18–24 months, male-to-female ratio 1.4:1, EEG abnormalities in 33%, focal seizures associated with higher risk of recurrence, favorable short-term outcomes in majority.
Study 2 [14]	Multicenter prospective cohort	249 children	Complex febrile seizures associated with prolonged duration (>15 min) and focal features, 30–40% recurrence, risk of subsequent epilepsy 8–12%, EEG abnormalities predictive of recurrence, viral infections common trigger.
Study 3 [15]	Longitudinal cohort	1,050 children	Majority of febrile seizures were simple (70%), complex seizures 30%, male predominance 1.3:1, children with prolonged seizures had higher risk of neurological deficits, long-term cognitive outcomes were generally favorable, and early intervention was recommended.

Common Etiologies: Viruses (46%), bacterial infections (22%), and unknown causes produced fever before seizures. These findings confirm national research suggesting respiratory and gastrointestinal viruses cause most childhood febrile seizures. Rare bacterial infections caused worse symptoms, aberrant EEGs, and neuroimaging in our sample. In severe febrile seizures with encephalopathic symptoms, systemic inflammatory reactions and direct CNS involvement in bacterial meningitis or sepsis have been connected. Supports literature. Children in undernourished communities can have seizures from metabolic abnormalities such as hypoglycemia and electrolyte imbalance. The study eliminated metabolically unwell children for infectious and neurological reasons. Identifying complex febrile seizures from encephalopathy requires metabolic monitoring.

Age and Gender Trends: The observed age distribution shows that toddlers have more complex febrile seizures and encephalopathy. The brain develops rapidly in the first 12–36 months, making it susceptible to fever-induced neuronal excitability. This study's sample's male predominance may be due to sex-specific immune response, genetic predisposition, and healthcare-seeking behaviour. Despite not being statistically significant in neurological outcomes, gender variations may affect the population's disease burden. Knowing these demographic trends helps clinicians identify high-risk groups for early surveillance and intervention.

Limitations: Despite limitations, the study exposes complex febrile seizures and paediatric encephalopathy in tertiary care. The 100-patient sample size limits generalisation. Second, the study's single tertiary care centre may create selection bias. Because hospital patients may have more serious illnesses or repercussions than primary care clinic patients. Short-term follow-up hindered investigation of prolonged neurological effects, seizure recurrence, and epilepsy progression beyond three months. Clinically indicated MRIs may have underestimated structural abnormality frequency. This study did not explore nutritional and socioeconomic aspects that may affect seizure vulnerability and outcome. More multi-center trials with larger populations and longer follow-ups are needed to confirm these findings and build strong outcome prediction models.

Conclusion

According to Bhagwan Mahavir Institute of Medical Sciences in Pawapuri, complex febrile seizures and paediatric encephalopathy disproportionately strike 12–36-month-olds, with few males. The longest, most common seizure was generalised tonic-clonic, which altered consciousness and happened again within 24 hours. The most prevalent cause was viral infections, while bacterial infections caused more serious neurological disorders, such as aberrant EEG and MRI readings 72% of children rebounded. 10% had early seizure recurrence, and 18% had neurological problems. These findings emphasise the clinical relevance of early diagnosis of high-risk variables

like focal onset, persistent seizures, and aberrant neurophysiological or radiological findings. To avoid long-term neurological disorders, go to the hospital, get diagnosed, and start therapy. To reduce morbidity, community-based education may highlight fever control, seizure warning indicators, and quick tertiary referral. Public health educators and clinicians can aid children at risk of complicated febrile seizures and encephalopathy.

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