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# CLINICAL PRESENTATION AND ETIOLOGICAL SPECTRUM OF PAEDIATRIC ACUTE ENCEPHALITIS SYNDROME IN A TERTIARY CARE HOSPITAL

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ABSTRACT

**Background:** Acute Encephalitis Syndrome (AES) is a major cause of pediatric neurological morbidity and mortality in India, with diverse infectious etiologies and variable clinical outcomes. Understanding region-specific clinical and etiological patterns is essential for early diagnosis and effective management.

**Materials and Methods:** This hospital-based observational study was conducted in a tertiary care center and included 127 children aged 1 month to 18 years diagnosed with AES. Detailed demographic, clinical, laboratory, cerebrospinal fluid, neuroimaging, and etiological data were collected. Patients were followed until discharge to assess outcomes. Statistical analysis was performed to identify factors associated with mortality.

**Results:** The mean age of presentation was 6.8 ± 4.1 years, with a male predominance (58.3%). Most children presented during the monsoon season (54.3%). Viral encephalitis was the most common etiology (48.0%), with Japanese encephalitis accounting for 16.5% of cases. Scrub typhus (11.8%) and bacterial meningoencephalitis (22.8%) were important non-viral causes. Complete recovery was observed in 53.5% of children, neurological sequelae in 29.1%, and mortality in 17.3%. Younger age, low Glasgow Coma Scale score (≤8), refractory seizures, shock, and raised intracranial pressure were significantly associated with mortality ($p < 0.05$).

**Conclusion:** Pediatric AES continues to be associated with substantial mortality and neurological sequelae. While viral etiologies predominate, emerging treatable infections such as scrub typhus contribute significantly. Early identification of high-risk children and strengthening of diagnostic, preventive, and critical care services are essential to improve outcomes.

**Keywords:** Acute encephalitis syndrome; Pediatrics; Etiology; Japanese encephalitis; Scrub typhus; Neurological outcomes.

## INTRODUCTION

Acute Encephalitis Syndrome (AES) is a major public health problem in the pediatric population, particularly in low- and middle-income countries, where it contributes substantially to childhood morbidity, mortality, and long-term neurological disability.[1] AES is clinically defined by the acute onset of fever accompanied by altered mental status (such as confusion, disorientation, coma) and/or new-onset seizures, excluding simple febrile seizures.[2] This broad syndromic definition encompasses a wide spectrum of infectious and non-infectious etiologies, making early diagnosis and targeted management particularly challenging in children.[2]

Globally, encephalitis accounts for significant pediatric hospital admissions, with reported incidence ranging from 3.5 to 7.4 per 100,000 children per year, and higher rates in children under 15 years of age.[3] Mortality rates vary widely depending on etiology and access to intensive care, ranging from 5% to over 30%, while up to 50% of survivors may be left with persistent neurocognitive or motor sequelae.[4] The burden is disproportionately

higher in South and Southeast Asia, where recurrent outbreaks of AES have been reported, often with seasonal clustering.[5]

In India, AES remains a major cause of pediatric neurological illness, particularly in rural and peri-urban regions. Although Japanese encephalitis (JE) virus has historically been recognized as a leading cause of AES outbreaks, especially in endemic belts, recent epidemiological data indicate a changing etiological spectrum.[6] A large proportion of AES cases are now attributed to non-JE viral pathogens such as enteroviruses, herpes simplex virus, dengue virus, scrub typhus, and other emerging or re-emerging infections, while a significant number of cases remain etiologically undetermined despite extensive investigations.[7] This evolving pattern underscores the complexity of AES and highlights the limitations of surveillance systems that focus predominantly on JE.

Children are particularly vulnerable to AES due to their developing immune and nervous systems, and the clinical presentation in pediatric age groups is often non-specific. Symptoms such as fever, vomiting, seizures, irritability, and altered sensorium may overlap with other common pediatric illnesses, leading to delays in recognition and referral.[8] Moreover, the clinical profile, severity, and outcomes of AES can vary considerably depending on the underlying etiology, age of the child, nutritional status, and timeliness of supportive and specific therapy.[8]

Understanding the clinical manifestations and etiological distribution of AES in children is crucial for improving early diagnosis, rational use of antimicrobials and antivirals, prioritization of laboratory investigations, and implementation of appropriate public health interventions.[9] Region-specific data are particularly important, as the etiological agents and clinical patterns of AES show marked geographical and temporal variation.[10.11] Despite the significant disease burden, there is a relative paucity of comprehensive hospital-based observational studies focusing exclusively on the pediatric age group that correlate clinical features with etiological findings and outcomes.[11]

In this context, the present observational study was aimed to describe the clinical and etiological profile of Acute Encephalitis Syndrome among children. By delineating demographic characteristics, presenting features, laboratory and etiological patterns, and outcomes, this study aims to contribute to a better understanding of AES in the pediatric population and to support evidence-based clinical and preventive strategies tailored to local epidemiology.

## MATERIALS AND METHODS

**Study design and setting:** This study was conducted as a hospital-based observational study in the Department of Pediatrics of a tertiary care teaching hospital, in North India. The hospital serves as a major referral center for pediatric neurological and infectious diseases, catering to both urban and rural populations. The study was carried out over a defined study period of 24 months, from August 2023 to August 2025, during which all eligible pediatric patients presenting with features suggestive of Acute Encephalitis Syndrome (AES) were prospectively evaluated and enrolled.

**Study population:** The study population comprised children aged 1 month to 18 years who were admitted with a clinical diagnosis of AES during the study period. AES was defined in accordance with standard case definitions as an acute onset of fever associated with altered mental status (including confusion, disorientation, altered sensorium, or coma) and/or new-onset seizures, excluding simple febrile seizures. Children who fulfilled the clinical criteria at the time of admission or during the course of hospitalization were considered for inclusion.

**Inclusion and exclusion criteria**

All consecutive pediatric patients fulfilling the AES case definition and whose parents or legal guardians provided informed consent were included in the study. Neonates with perinatal asphyxia, children with known epilepsy presenting with breakthrough seizures without altered sensorium, cases of metabolic encephalopathy (such as hypoglycemia, dyselectrolytemia, hepatic or uremic encephalopathy), traumatic brain injury, and children with central nervous system malformations or neurodegenerative disorders were excluded to minimize diagnostic ambiguity.

**Clinical evaluation and data collection:** A detailed clinical evaluation was performed for each enrolled child using a structured proforma. Information regarding demographic details, seasonal distribution, immunization status (including vaccination against Japanese encephalitis where applicable), history of preceding illness, duration of fever, seizures, altered sensorium, vomiting, headache, and other neurological or systemic symptoms was recorded. A thorough general physical examination and detailed neurological examination were carried out at admission and repeated during hospitalization to document disease progression and complications. Level of consciousness was assessed using age-appropriate pediatric coma scales.

**Laboratory investigations:** All patients underwent baseline laboratory investigations, including complete blood count, random blood glucose, serum electrolytes, renal and liver function tests, and inflammatory markers as clinically indicated. Lumbar puncture was performed in all patients without contraindications, and cerebrospinal fluid (CSF) was analyzed for cell count, differential count, protein, glucose, and microbiological parameters. CSF samples were subjected to Gram staining, bacterial culture, and relevant antigen or molecular tests depending on clinical suspicion and availability. Blood cultures were obtained prior to initiation of antibiotics whenever feasible.

**Etiological evaluation:** Etiological workup was guided by clinical presentation, epidemiological context, and seasonal trends. Serum and/or CSF samples were tested for common viral and bacterial causes of AES, including Japanese encephalitis virus, herpes simplex virus, enteroviruses, dengue virus, scrub typhus, and other regionally prevalent pathogens, using appropriate serological and molecular assays. Cases were categorized as confirmed etiological diagnoses when laboratory evidence was obtained, probable when supported by clinical and epidemiological features, or of unknown etiology when no specific cause could be identified despite investigations.

**Neuroimaging and electrophysiological studies:** Neuroimaging was performed in patients with focal neurological deficits, persistent altered sensorium, refractory seizures, or clinical deterioration. Computed tomography (CT) and/or magnetic resonance imaging (MRI) of the brain were undertaken as per institutional protocol to identify structural lesions, cerebral edema, or characteristic patterns suggestive of specific etiologies. Electroencephalography (EEG) was performed in selected cases with recurrent seizures or suspected encephalopathy to assess cerebral electrical activity and support diagnosis.

**Treatment and outcome assessment:** All enrolled children received standard supportive care as per institutional guidelines, including airway protection, hemodynamic stabilization, seizure control, and management of raised intracranial pressure when present. Empirical antimicrobial and antiviral therapy was initiated at admission and modified according to laboratory results and clinical response. Patients were monitored throughout hospitalization, and outcomes were documented at discharge in terms of recovery, presence of neurological deficits, or death. Neurological sequelae were assessed clinically before discharge.

**Statistical analysis:** Data were entered into a standardized database and analyzed using appropriate statistical software. Continuous variables were expressed as mean ± standard deviation or median with interquartile range, while categorical variables were presented as frequencies and percentages. Associations between clinical features, etiological categories, and outcomes were analyzed using suitable statistical tests, with a p-value of less than 0.05 considered statistically significant.

**Ethical considerations:** The study was conducted after obtaining approval from the Institutional Ethics Committee. Written informed consent was obtained from parents or legal guardians prior to enrollment, and assent was obtained from older children when appropriate. Patient confidentiality was maintained throughout the study, and all procedures were performed in accordance with ethical principles outlined in the Declaration of Helsinki.

## RESULTS

The study included 127 children diagnosed with Acute Encephalitis Syndrome. The mean age of presentation was 6.8 ± 4.1 years, with the highest proportion of cases observed in children below 5 years of age (38.6%), followed by those aged 5–10 years (36.2%). Male children predominated, accounting for 58.3% of cases. A majority of patients belonged to rural areas (63.8%). Seasonal clustering was evident, with more than half of the cases presenting during the monsoon months (54.3%). Only 30.7% of children had received Japanese encephalitis vaccination. The mean duration of fever prior to admission was 4.6 ± 2.1 days [Table 1].

**Table 1: Baseline Demographic and Clinical Characteristics of Children with Acute Encephalitis Syndrome (n = 127).**

| Variable | Frequency / Mean ± SD | % |
|---|---|---|
| Age (years) | 6.8 ± 4.1 | — |
| <5 years | 49 | 38.6 |
| 5–10 years | 46 | 36.2 |
| >10 years | 32 | 25.2 |
| Gender | | |
| Male | 74 | 58.3 |
| Female | 53 | 41.7 |
| Residence | | |
| Rural | 81 | 63.8 |
| Urban | 46 | 36.2 |
| Season of presentation | | |
| Monsoon (Jun–Sep) | 69 | 54.3 |
| Post-monsoon (Oct–Nov) | 32 | 25.2 |
| Winter & Summer | 26 | 20.5 |
| Immunization (JE vaccine received) | 39 | 30.7 |
| Mean duration of fever (days) | 4.6 ± 2.1 | — |

JE – Japanese encephalitis.

Fever was a universal presenting symptom, observed in all children. Altered sensorium was present in 92.9% of cases, while seizures were noted in 70.1%. Nearly half of the children experienced vomiting (48.0%), and headache was reported in 29.1% of older children. Meningeal signs were elicited in one-third of cases (33.1%). Focal neurological deficits were documented in 24.4% of patients. Hemodynamic instability in the form of shock was present in 15.0%, and 45.7% of children required admission to the PICU, reflecting significant disease severity [Table 2].

**Table 2: Clinical Profile of Pediatric Acute Encephalitis Syndrome (n = 127).**

| Clinical Feature | Frequency | % |
|---|---|---|
| Fever | 127 | 100 |
| Altered sensorium | 118 | 92.9 |
| Seizures | 89 | 70.1 |
| Vomiting | 61 | 48 |
| Headache (older children) | 37 | 29.1 |
| Meningeal signs | 42 | 33.1 |
| Focal neurological deficits | 31 | 24.4 |
| Shock / hypotension | 19 | 15 |
| Need for PICU admission | 58 | 45.7 |

AES – Acute encephalitis syndrome; PICU – Pediatric Intensive Care Unit.

Lumbar puncture could be performed in 118 children, while it was deferred in 9 cases due to raised intracranial pressure. CSF pleocytosis was observed in 70.3% of samples, with lymphocytic predominance seen in 54.2% and neutrophilic predominance in 16.1%. Elevated CSF protein levels were noted in 64.4% of children, while hypoglycorrhachia was present in 23.7%. Overall CSF findings were suggestive of viral etiology in 51.7% of cases and bacterial etiology in 18.6%, aiding in etiological categorization (Table 3).

**Table 3: Cerebrospinal Fluid Analysis Findings in Children with Acute Encephalitis Syndrome (n = 118).**

| CSF Parameter | Frequency (%) |
|---|---|
| Pleocytosis (>5 cells/mm³) | 83 (70.3%) |
| Predominant lymphocytes | 64 (54.2%) |
| Predominant neutrophils | 19 (16.1%) |
| Elevated protein (>45 mg/dL) | 76 (64.4%) |
| Low glucose (<40 mg/dL) | 28 (23.7%) |
| CSF suggestive of viral etiology | 61 (51.7%) |
| CSF suggestive of bacterial etiology | 22 (18.6%) |

CSF – Cerebrospinal fluid; LP – lumbar puncture; ICP – intracranial pressure.

A definite or probable etiological diagnosis was established in 89.8% of cases. Viral encephalitis constituted the largest etiological group (48.0%), with Japanese encephalitis identified in 16.5% of cases, followed by herpes simplex virus (11.0%), dengue virus (10.2%), and enteroviruses (10.2%). Bacterial meningoencephalitis accounted for 22.8% of cases. Scrub typhus encephalitis emerged as an important etiology, contributing to 11.8% of cases. Tubercular meningoencephalitis was diagnosed in 7.1%, while 10.2% of cases remained etiologically undetermined despite investigations. Neuroimaging was performed in 82 children based on clinical indications. Abnormal findings were observed in 74.4% of scans. Cerebral edema was the most common abnormality, seen in 31.7% of cases. Thalamic and basal ganglia involvement was noted in 20.7%, while temporal lobe involvement was observed in 13.4%. Hydrocephalus was identified in 8.6% of children. A normal imaging study was reported in 25.6% of cases (Table 4).

**Table 4: Etiological Distribution of Acute Encephalitis Syndrome in the Study Population (n = 127); and Neuroimaging Findings in Children with Acute Encephalitis Syndrome (n = 82).**

| Variables | Frequency | % |
|---|---|---|
| Etiology | | |
| Viral encephalitis (total) | 61 | 48 |
| – Japanese encephalitis | 21 | 16.5 |
| – Herpes simplex virus | 14 | 11 |
| – Dengue encephalitis | 13 | 10.2 |
| – Enteroviral | 13 | 10.2 |
| Bacterial meningoencephalitis | 29 | 22.8 |
| Scrub typhus encephalitis | 15 | 11.8 |
| Tubercular meningoencephalitis | 9 | 7.1 |
| Unknown / undetermined etiology | 13 | 10.2 |
| Imaging Finding | | |
| Normal study | 21 | 25.6 |
| Cerebral edema | 26 | 31.7 |
| Thalamic/basal ganglia involvement | 17 | 20.7 |
| Temporal lobe involvement | 11 | 13.4 |
| Hydrocephalus | 7 | 8.6 |

HSV – Herpes simplex virus; JE – Japanese encephalitis; CT – Computed tomography; MRI – Magnetic resonance imaging.

At discharge, complete neurological recovery was observed in 53.5% of children. Neurological sequelae were documented in 29.1%, while the overall mortality rate was 17.3%. Children with viral

encephalitis demonstrated a significantly higher rate of complete recovery compared to those with non-viral etiologies (62.3% vs 45.5%, p = 0.048). Mortality was significantly higher in the non-viral group compared to viral etiologies (21.2% vs 13.1%, p = 0.041) [Table 5].

**Table 5: Outcome of Acute Encephalitis Syndrome and Its Association with Etiology (n = 127).**

| Outcome | Total | Viral (n=61) | Non-viral (n=66) | p-value |
|---|---|---|---|---|
| | Frequency (%) | | | |
| Complete recovery | 68 (53.5) | 38 (62.3) | 30 (45.5) | 0.048 |
| Neurological sequelae | 37 (29.1) | 15 (24.6) | 22 (33.3) | 0.27 |
| Death | 22 (17.3) | 8 (13.1) | 14 (21.2) | 0.041 |

Outcomes assessed at discharge; p-value <0.05 considered statistically significant.

Mortality analysis revealed that children younger than 5 years had a significantly higher risk of death compared to older children (68.2% vs 32.4%, p = 0.002). A Glasgow Coma Scale score of ≤8 at admission was strongly associated with mortality (77.3% vs 18.1%, p < 0.001). Refractory seizures, presence of shock at admission, and raised intracranial pressure were all significantly more common among non-survivors, with p-values <0.001, indicating their strong predictive value for poor outcome (Table 6).

**Table 6: Factors Associated with Mortality in Pediatric Acute Encephalitis Syndrome (n = 127).**

| Variable | Survivors (n=105) | Non-survivors (n=22) | p-value |
|---|---|---|---|
| | Frequency (%) | | |
| Age <5 years | 34 (32.4%) | 15 (68.2%) | 0.002 |
| Glasgow Coma Scale ≤8 | 19 (18.1%) | 17 (77.3%) | <0.001 |
| Refractory seizures | 21 (20.0%) | 13 (59.1%) | 0.001 |
| Shock at admission | 9 (8.6%) | 10 (45.5%) | <0.001 |
| Raised intracranial pressure | 17 (16.2%) | 14 (63.6%) | <0.001 |

GCS – Glasgow Coma Scale; ICP – intracranial pressure.

## DISCUSSION

Acute Encephalitis Syndrome (AES) continues to pose a substantial clinical and public health challenge in the pediatric population, particularly in developing countries such as India where the disease burden is amplified by seasonal outbreaks, diverse etiological agents, and limitations in diagnostic capacity.[12] The present observational study provides a comprehensive overview of the clinical spectrum, etiological profile, and outcomes of pediatric AES in a tertiary care setting and highlights important trends relevant to contemporary Indian and global scenarios. In the present study, AES predominantly affected younger children, with nearly two-thirds of cases occurring below 10 years of age and a mean age of 6.8 years. This age distribution is consistent with reports from Indian studies by Susmitha et al., and Ravi et al., which demonstrate increased susceptibility among younger children due to immature immune responses and greater exposure to environmental and vector-borne pathogens.[13,14] Male predominance observed in this cohort has been widely reported in Indian studies by Susmitha et al., and Arunkumar et al., and is often attributed to increased outdoor exposure and possible gender-based healthcare-seeking behavior.[13,15] The predominance of rural children and the marked seasonal clustering during monsoon and post-monsoon months further reinforce the role of environmental, vector-related, and socio-demographic factors in the epidemiology of AES in India.[16]

Clinically, fever and altered sensorium were near-universal features, while seizures were observed in over two-thirds of patients, reflecting the classical presentation of pediatric AES. Similar frequencies of seizures and altered consciousness have been reported from studies across India by Suma et al., and Ary et al.[17,18] The relatively high requirement for pediatric intensive care admission underscores the severity of illness at presentation and highlights the need for early recognition and timely referral.[18] The presence of focal neurological deficits and shock in a substantial proportion of children suggests advanced disease at admission, which has been consistently linked to poorer outcomes in previous studies by Ary et al., and Sneha et al.[18,19]

Cerebrospinal fluid analysis in the present study predominantly showed lymphocytic pleocytosis with elevated protein levels, suggestive of viral encephalitis in more than half of the cases. This pattern aligns with contemporary Indian studies by Susmitha et al., and Ravi et al., that report a shift from classical bacterial causes toward viral and rickettsial etiologies.[13,14] However, the presence of neutrophilic pleocytosis and low CSF glucose in a significant subset highlights the ongoing contribution of bacterial and tubercular meningoencephalitis in the pediatric population, particularly in resource-limited settings.[16]

Etiologically, viral encephalitis constituted the largest group, accounting for nearly half of all AES cases. Japanese encephalitis (JE), though historically the dominant cause of AES outbreaks in India, accounted for only about one-sixth of cases in this study. This finding mirrors recent Indian studies by

Vasanthapuram et al., and Kabilan et al., demonstrating a declining proportion of JE following the introduction and expansion of JE vaccination programs.[20,21] Nevertheless, the persistence of JE cases emphasizes the need for sustained immunization coverage and vector control measures. The notable contribution of herpes simplex virus and dengue virus reflects the expanding etiological spectrum of AES, a trend also observed globally.[22] Importantly, scrub typhus emerged as a significant and potentially treatable cause of AES, corroborating reports from various parts of India that describe rickettsial infections as an increasingly recognized etiology of pediatric encephalitis.[23] Despite extensive evaluation, approximately 10% of cases remained etiologically undetermined, a finding consistent with Indian studies by Kaliban et al., and Rohani et al., and indicative of diagnostic limitations and the possibility of emerging or unidentified pathogens.[21,22]

Neuroimaging findings revealed abnormalities in nearly three-fourths of the children who underwent imaging, with cerebral edema being the most common finding. Thalamic and basal ganglia involvement, observed in a notable proportion, is classically associated with JE and other viral encephalitides and has been frequently reported in Indian studies by Suma et al., and Kaliban et al.[17,21] Temporal lobe involvement in a subset of children further supports the contribution of herpes simplex virus encephalitis.[22] These imaging patterns are valuable adjuncts in guiding etiological suspicion and management, especially when laboratory confirmation is delayed or unavailable.[23]

Outcome analysis demonstrated that just over half of the children achieved complete recovery, while nearly one-third suffered from neurological sequelae, underscoring the long-term burden of AES beyond acute mortality. The overall mortality rate of 17.3% observed in this study is comparable to rates reported from other tertiary care centers in India by Kaliban et al., and Ravi et al., and remains higher than those reported from high-income countries, reflecting disparities in early diagnosis, access to intensive care, and availability of advanced diagnostic and therapeutic modalities.[21,24] Viral etiologies were associated with significantly better recovery and lower mortality compared to non-viral causes, a finding supported by previous Indian studies by Swetapadma et al., and Shah et al.[25,26] Bacterial, tubercular, and rickettsial encephalitis often present with more severe systemic involvement and delayed diagnosis, contributing to poorer outcomes.[25,26]

Analysis of mortality predictors identified younger age, low Glasgow Coma Scale score at admission, refractory seizures, shock, and raised intracranial pressure as strong determinants of poor outcome. These factors have been consistently recognized across multiple Indian studies by Bokade et al., and Adhikari et al., and emphasize the importance of early identification of high-risk children.[27,28] Prompt stabilization, aggressive supportive care, and early initiation of appropriate antimicrobial or antiviral therapy remain critical to improving survival and reducing long-term neurological disability.[29]

**Limitations**

This study has certain limitations that should be acknowledged while interpreting the findings. Being a single-center, hospital-based observational study, the results may not be fully generalizable to the wider community or to regions with different epidemiological patterns. Although a comprehensive etiological workup was attempted, advanced molecular diagnostic techniques were not universally available, which may have contributed to the proportion of cases with undetermined etiology. Neuroimaging and electroencephalography could not be performed in all patients due to clinical instability or resource constraints. Long-term neurodevelopmental follow-up was not undertaken, and neurological sequelae were assessed only at discharge, which may underestimate the true burden of persistent deficits. Despite these limitations, the study provides valuable region-specific insights into the evolving clinical and etiological profile of pediatric Acute Encephalitis Syndrome.

## CONCLUSION

Acute Encephalitis Syndrome remains a significant cause of morbidity and mortality among children, with a heterogeneous and evolving etiological spectrum. Viral encephalitis continues to be the predominant cause, although the relative contribution of Japanese encephalitis has declined in the post-vaccination era. Emerging and treatable infections such as scrub typhus constitute an important proportion of cases and warrant heightened clinical suspicion. Younger age, severe neurological impairment at presentation, refractory seizures, shock, and raised intracranial pressure are key predictors of mortality. Early recognition, prompt referral, aggressive supportive care, and timely initiation of appropriate antimicrobial or antiviral therapy are crucial to improving outcomes. Strengthening diagnostic facilities, expanding vaccination coverage, and conducting ongoing surveillance are essential to reduce the burden of pediatric Acute Encephalitis Syndrome and its long-term neurological consequences.

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