

Research Article

Clinical and Laboratory Profile of Acute Encephalitis Syndrome in Children: A Hospital-Based Study

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Received: 21.06.2026, Revised: 23.08.2026, Accepted: 25.09.2026

ABSTRACT

Background: Acute encephalitis syndrome (AES) is an important neurological emergency in children and is associated with considerable morbidity and mortality. Clinical presentation and laboratory abnormalities may vary with age and underlying etiology.

Methods: This prospective cross-sectional study included 143 children aged 6 months to <12 years admitted to the Paediatric Intensive Care Unit of Mahaveer Institute of Medical Sciences, who fulfilled the WHO criteria for AES. Clinical examination, hematological investigations, and cerebrospinal fluid (CSF) analysis were performed. Categorical variables were compared using the Chi-square test or Fisher's exact test, as appropriate.

Results: Among 143 children, 76 (53.1%) were male and 67 (46.9%) were female. Fever was present in all patients (100%), followed by seizures (95.8%), altered consciousness (92.3%), vomiting (42.7%), abnormal posturing (36.4%), rash (25.9%), and headache (20.3%). Significant age-related differences were observed for headache, seizures, loose stools, pulse rate, pallor, hemoglobin level, and CSF pressure. Viral AES accounted for 55.9% of cases and non-viral AES for 44.1%.

Conclusion: AES in children presents predominantly with fever, seizures, and altered consciousness, accompanied by important hematological and CSF abnormalities. Recognition of characteristic clinical and laboratory patterns may facilitate timely evaluation and management.

Keywords: Acute Encephalitis Syndrome, Children; Seizures, Cerebrospinal Fluid, Encephalitis, Clinical Profile.

INTRODUCTION

Acute encephalitis syndrome (AES) is an important neurological condition characterized by acute onset of fever associated with altered mental status and/or new-onset seizures. It encompasses a heterogeneous group of disorders involving inflammation of the brain and may result from several infectious causes. Encephalopathy, which commonly manifests as reduced consciousness or altered behaviour, may also occur due to systemic infections, metabolic disorders, toxins, hypoxia, trauma, vasculitis, and central nervous system infections.[1] Encephalitis, in contrast, specifically refers to inflammation of the brain, although in clinical practice the diagnosis is

usually supported by clinical, laboratory, and imaging findings.[2,3]

For surveillance purposes, the World Health Organization defined AES as the acute onset of fever accompanied by a change in mental status, such as confusion, disorientation, coma, or inability to talk, and/or new-onset seizures other than simple febrile seizures.[4] The illness generally has a duration of less than 14 days. Irritability, somnolence, and behavioural changes beyond those expected with an uncomplicated febrile illness may also be observed. This broad clinical definition facilitates identification of suspected cases, particularly in regions where laboratory confirmation of a specific etiological agent may not always be readily available.[4]

Encephalitis and meningitis may have overlapping clinical manifestations because infectious processes can involve both the meninges and brain parenchyma. Consequently, the term meningoencephalitis is often used when features of both conditions coexist. Patients with predominant alteration in sensorium are more likely to have an encephalitic presentation, whereas prominent signs of meningeal irritation suggest meningitis. Cerebrospinal fluid (CSF) examination therefore has an important role in evaluating children presenting with suspected central nervous system infection and in differentiating possible etiological patterns.[5,6]

The epidemiology and causes of AES vary considerably according to geographical region, age, season, and the availability of diagnostic facilities. Studies from India have demonstrated a changing etiological spectrum of AES, highlighting the need for systematic clinical and laboratory evaluation of affected children.[3] Surveillance studies have also demonstrated a substantial burden of AES among children, particularly in endemic regions.[7] Because AES represents a clinical syndrome rather than a single disease entity, establishing the underlying cause may remain challenging even after extensive investigation.

Children with AES can present with a wide spectrum of manifestations, ranging from fever, headache, vomiting, seizures, and altered consciousness to focal neurological deficits and severe neurological impairment. Previous pediatric studies have documented considerable variation in the clinical profile and outcomes of children admitted with AES.[8]

Therefore, the present study was undertaken to evaluate the clinical and laboratory profile of acute encephalitis syndrome in children aged 6 months to <12 years, with particular emphasis on presenting symptoms, vital parameters, general examination findings, hematological abnormalities, and CSF characteristics.

MATERIALS AND METHODS

Study Design and Setting

This prospective cross-sectional study was conducted in the Paediatric Intensive Care Unit (PICU) of Mahaveer Institute of Medical Sciences. The study was conducted over a period of 2 years.

Study Population

Children aged 6 months to <12 years presenting with acute onset of fever associated with altered mental status and/or new-onset

seizures were considered for inclusion. The clinical definition was based on the WHO criteria for AES. Altered neurological status included altered level of consciousness, lethargy or irritability, change in personality or behaviour, confusion, disorientation, coma, inability to talk, focal neurological deficit, or cranial nerve palsy.

Inclusion Criteria

Children aged 6 months to <12 years presenting with acute onset of fever of less than 14 days duration along with a change in mental status and/or new-onset seizures, excluding simple febrile seizures, were included. Neurological manifestations considered for enrolment included altered consciousness, convulsions, lethargy or irritability, behavioural changes, confusion or disorientation, coma, inability to talk, focal neurological deficit, and cranial nerve palsy.

Exclusion Criteria

Patients with non-infectious encephalopathy secondary to hypoxic, ischemic, vascular, toxic, or metabolic causes were excluded. Children with febrile convulsions, epilepsy, or known central nervous system malformations were also excluded. Patients whose parents or attendants did not provide consent were not enrolled.

Clinical Evaluation and Initial Management

On admission, each child underwent rapid assessment and stabilization according to the standard treatment protocol. Airway and breathing were assessed, and intubation was performed when indicated, including in patients with a Glasgow Coma Scale score <8, impaired airway reflexes, abnormal respiratory pattern, persistent oxygen saturation <92% despite high-flow oxygen, or fluid-refractory shock. Oxygenation and ventilatory support were provided as required.

Intravenous access was established, appropriate samples were collected, and fluid resuscitation was administered in children with circulatory failure. Inotropic support was provided when required. Patients were assessed for signs of raised intracranial pressure or cerebral herniation. Abnormal body temperature was managed, and ongoing seizures were treated with benzodiazepines followed by phenytoin loading as described in the study protocol.

History and Physical Examination

Following stabilization, written informed consent was obtained from the parent or

guardian. A detailed history was recorded, including presenting complaints and relevant clinical features. Vital parameters including heart rate, respiratory rate, blood pressure, and temperature were documented.

General physical examination included assessment for pallor, icterus, cyanosis, clubbing, lymphadenopathy, edema, and dehydration. A systemic examination with particular emphasis on detailed central nervous system examination was performed. All relevant clinical findings were recorded on a predesigned study proforma.

Laboratory Evaluation

Investigations were performed as early as possible following admission to establish the underlying etiology and characterize the laboratory profile. Hematological assessment included random blood sugar, hemoglobin, total leukocyte count, differential leukocyte count, and platelet count.

CSF evaluation included assessment of CSF pressure, appearance, sugar, protein, cell count, cytological predominance, culture, and

CBNAAT. According to the study methodology, when a non-viral etiology was established, further evaluation for viral etiologies was not performed.

Classification of AES

Based on CSF parameters and etiological assessment, AES cases were categorized into viral and non-viral AES. Of the 143 patients, 80 (55.9%) were classified as viral AES and 63 (44.1%) as non-viral AES.

Statistical Analysis

Categorical variables were expressed as frequencies and percentages. Qualitative variables were compared using the Chi-square test or Fisher's exact test, as appropriate. A p-value of <0.05 was considered statistically significant. The thesis also states that univariate and multivariate logistic regression were applied for assessment of predictors. Data were entered into Microsoft Excel and analyzed using Statistical Package for the Social Sciences (SPSS), version 22.0.

RESULTS

Table 1. Demographic Characteristics of the Study Population According To Age Group (N=143)

Characteristic	6 months to <5 years (n=77)	5–10 years (n=48)	>10 years (n=18)	p-value
Male	38 (49.4%)	29 (60.4%)	9 (50.0%)	0.464
Female	39 (50.6%)	19 (39.6%)	9 (50.0%)	
Total	77 (53.8%)	48 (33.6%)	18 (12.6%)	

Table 2. Clinical Manifestations According To Age Group

Clinical feature	6 months to <5 years (n=77)	5–10 years (n=48)	>10 years (n=18)	p-value
Fever	77 (100%)	48 (100%)	18 (100%)	NA
Headache	9 (11.7%)	15 (31.2%)	5 (27.8%)	0.021
Vomiting	28 (36.4%)	25 (52.1%)	8 (44.4%)	0.222
Seizures	76 (98.7%)	43 (89.6%)	18 (100%)	0.030
Abnormal posturing	25 (32.5%)	21 (43.8%)	6 (33.3%)	0.426
Altered consciousness	69 (89.6%)	45 (93.8%)	18 (100%)	0.297
Rash	15 (19.5%)	17 (35.4%)	5 (27.8%)	0.138
Extrapyramidal symptoms	1 (1.3%)	3 (6.2%)	0	0.196
Loose stools	16 (20.8%)	1 (2.1%)	2 (11.1%)	0.011

Table 3. Selected Vital and General Examination Findings According To Age Group

Parameter	6 months to <5 years	5–10 years	>10 years	p-value
Increased temperature	39 (50.6%)	24 (50.0%)	7 (38.9%)	0.356
Increased pulse rate	64 (83.1%)	30 (62.5%)	13 (72.2%)	0.034
Low blood pressure	28 (36.4%)	12 (25.0%)	7 (38.9%)	0.336
Increased respiratory rate	46 (59.7%)	24 (50.0%)	13 (72.2%)	0.495
Dehydration	25 (32.5%)	13 (27.1%)	4 (22.2%)	0.631
Pallor	29 (37.7%)	8 (16.7%)	5 (27.8%)	0.043
Icterus	4 (5.2%)	4 (8.3%)	0	0.412

Cyanosis	9 (11.7%)	4 (8.3%)	2 (11.1%)	0.834
Edema	12 (15.6%)	10 (20.8%)	3 (16.7%)	0.750

Table 4. Hematological Profile According To Age Group

Laboratory parameter	6 months to <5 years	5–10 years	>10 years	p-value
Hyperglycemia	11 (14.3%)	8 (16.7%)	3 (16.7%)	0.911*
Decreased hemoglobin	50 (64.9%)	11 (22.9%)	7 (38.9%)	<0.001
Increased TLC	45 (58.4%)	18 (37.5%)	9 (50.0%)	0.088
Increased neutrophil count	54 (70.1%)	36 (75.0%)	14 (77.8%)	0.734
Increased lymphocyte count	19 (24.7%)	11 (22.9%)	2 (11.1%)	0.459
Decreased platelet count	28 (36.4%)	19 (39.6%)	8 (44.4%)	0.605

Table 5. CSF Profile According To Age Group

CSF parameter	6 months to <5 years	5–10 years	>10 years	p-value
Increased CSF pressure	16 (20.8%)	22 (45.8%)	8 (44.4%)	0.007
Abnormal appearance	12 (15.6%)	12 (25.0%)	2 (11.1%)	0.293
Decreased sugar	21 (27.3%)	14 (29.2%)	3 (16.7%)	0.580
Increased protein	52 (67.5%)	33 (69.8%)	8 (44.5%)	0.358
Increased CSF cells	44 (57.1%)	28 (58.3%)	8 (44.4%)	0.571
Lymphocytic predominance	60 (77.9%)	41 (85.4%)	15 (83.3%)	0.563
Positive CSF culture	6 (7.8%)	2 (4.2%)	0	0.376
Positive CSF CBNAAT	2 (2.6%)	4 (8.3%)	1 (5.6%)	0.348

DISCUSSION

The present study evaluated the clinical and laboratory profile of 143 children with acute encephalitis syndrome (AES) aged 6 months to <12 years. The largest proportion of patients belonged to the younger age group, with 77 (53.8%) children aged 6 months to <5 years. The mean age reported in the thesis was 4.49 years. De et al. similarly reported a mean age of 5.32 years among children admitted with AES, while previous epidemiological observations have indicated a considerable burden of AES among younger children.[8,9] The clinical presentation was dominated by fever, seizures, and altered consciousness. Fever was present in all patients, while seizures occurred in 137 (95.8%) and altered consciousness in 132 (92.3%). Other manifestations included vomiting (42.7%), abnormal posturing (36.4%), rash (25.9%), headache (20.3%), and extrapyramidal symptoms (2.8%). Kumar et al. also described fever, convulsions, headache, loose stools, and vomiting among children with AES.[10] These findings emphasize that although fever with neurological dysfunction defines the syndrome, the accompanying clinical manifestations can be diverse.

Significant age-related differences were observed for headache, seizures, and loose stools. Headache was more frequently documented among older children, whereas seizures were particularly frequent in the youngest and oldest age groups. This variation

may partly reflect differences in clinical presentation and the ability of younger children to report subjective symptoms such as headache. Among vital parameters, increased pulse rate was common and differed significantly across age groups ($p=0.034$), whereas temperature, blood pressure, and respiratory rate did not demonstrate significant age-related differences. Pallor was the only general examination finding significantly associated with age ($p=0.043$).

Hematological abnormalities were frequently observed. Decreased hemoglobin was particularly common among children aged 6 months to <5 years (64.9%) and showed a significant association with age ($p<0.001$). Leukocytosis was observed across all age groups, while thrombocytopenia affected 36.4%–44.4% of children depending on age. Thrombocytopenia has also been identified in previous studies as an important abnormality among patients with severe encephalitis and has been associated with adverse outcomes in some clinical settings.[11]

CSF examination demonstrated several abnormalities. Increased CSF protein was present in 67.5% of children aged 6 months to <5 years and 69.8% of those aged 5–10 years. Lymphocytic predominance was the most frequent cytological pattern in all age groups. Increased CSF pressure was significantly associated with age ($p=0.007$), occurring in 20.8% of younger children compared with 45.8% of those aged 5–10 years and 44.4% of

those aged >10 years. CSF analysis remains an important component of the diagnostic evaluation of acute neurological infections and contributes to differentiation of infectious patterns.[12]

Although the primary objective of this paper was to characterize the clinical and laboratory profile, etiological classification showed that viral AES accounted for 80 (55.9%) cases and non-viral AES for 63 (44.1%). This predominance of viral AES is consistent with the pattern described in the thesis discussion, where previous studies also reported viral meningoencephalitis as an important contributor to AES.[13]

CONCLUSION

Acute encephalitis syndrome in children was characterized by a broad spectrum of clinical and laboratory abnormalities. Fever, seizures, and altered consciousness were the predominant presenting features. Significant age-related differences were observed in headache, seizures, loose stools, pulse rate, pallor, hemoglobin levels, and CSF pressure. Hematological abnormalities, particularly decreased hemoglobin, leukocytosis, and thrombocytopenia, were frequently observed, while CSF examination commonly demonstrated increased protein and lymphocytic predominance. Viral AES constituted a greater proportion of cases than non-viral AES. These findings emphasize the importance of systematic clinical assessment combined with hematological and CSF evaluation in children presenting with AES. Early recognition of characteristic abnormalities may facilitate appropriate investigation and timely management.

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