

Research Article

Comparison of Viral and Non-Viral Acute Encephalitis Syndrome in Children: Etiological Profile and Clinical Outcomes

Dr. Swaroop Anand Bolleddu¹, Dr Hareesh², Dr B. Raghu Nandan Reddy³, Dr. Syed Shah Naseeruddin Sarmast^{4*}

¹Assistant Professor Department of Pediatrics Mahaveer Institute of Medical Sciences Vikarabad, Telangana. 501102

²Assistant Professor Department of Pediatrics Mahavir Institute of Medical Sciences, Vikarabad, Telangana. 501102

³Department of Pediatrics Assistant Professor Mahavir Institute of Medical Sciences, Shivareddypet, Vikarabad, Telangana. 501102

^{4*}Associate Professor Department of Pediatrics Mahaveer Institute of Medical Sciences, Shivareddypet, Vikarabad, Telangana. 501102

Corresponding Author: Dr. Syed Shah Naseeruddin Sarmast

Associate Professor Department of Pediatrics Mahaveer Institute of Medical Sciences, Shivareddypet, Vikarabad, Telangana. 501102

Received: 21.06.2026, Revised: 23.08.2026, Accepted: 25.09.2026

ABSTRACT

Background: Acute encephalitis syndrome (AES) in children comprises a heterogeneous group of infectious neurological disorders with considerable morbidity and mortality. Differentiating viral from non-viral AES may facilitate etiological evaluation and appropriate management.

Methods: This prospective cross-sectional study included 143 children aged 6 months to <12 years admitted with AES to the Paediatric Intensive Care Unit of Mahaveer Institute of Medical Sciences. Patients underwent detailed clinical evaluation and laboratory investigations, including cerebrospinal fluid analysis. Cases were categorized as viral or non-viral AES based on CSF characteristics and etiological assessment.

Results: Of 143 children, 80 (55.9%) had viral AES and 63 (44.1%) had non-viral AES. Viral AES was predominantly associated with rash, extrapyramidal manifestations, decreased total leukocyte count, and thrombocytopenia, whereas vomiting, undernutrition, meningeal signs, cranial nerve palsy, increased leukocyte count, and hyponatremia were more prominent in non-viral AES. Inotropic support was required in 67 (46.9%) patients and ventilatory support in 44 (30.8%). Overall, 28 (19.6%) children died. Dengue was the leading identified viral etiology, while pyogenic and tubercular meningitis were important non-viral causes.

Conclusion: Viral AES constituted the larger etiological group. Viral and non-viral AES demonstrated differing clinical and laboratory patterns, while substantial mortality was observed across the overall AES cohort. Early etiological evaluation and recognition of characteristic clinical patterns may support timely management.

Keywords: Acute Encephalitis Syndrome, Viral Encephalitis, Non-Viral Encephalitis, Dengue, Meningoencephalitis, Pediatric Intensive Care.

INTRODUCTION

Acute encephalitis syndrome (AES) is a clinically defined neurological syndrome characterized by acute onset of fever associated with altered mental status and/or new-onset seizures. It represents a major diagnostic and therapeutic challenge because several infectious agents can produce a similar initial clinical presentation. Encephalitis refers to inflammation of the brain parenchyma, whereas encephalopathy describes altered mental status that may result from infectious as well as metabolic, toxic,

hypoxic, vascular, or other systemic causes.[1,2,3]

The World Health Organization introduced a standardized clinical definition of AES for surveillance of Japanese encephalitis. According to this definition, a suspected case presents with acute fever accompanied by a change in mental status, including confusion, disorientation, coma, or inability to talk, and/or new-onset seizures other than simple febrile seizures.[4] The broad nature of this definition is useful for surveillance but encompasses multiple viral and non-viral infectious conditions

with differing clinical characteristics, investigations, treatment requirements, and outcomes.

Encephalitis and meningitis frequently represent overlapping clinical syndromes. Both viral and non-viral invasion of the central nervous system may produce varying degrees of meningeal and brain parenchymal inflammation, resulting in meningoencephalitis. Predominant alteration in sensorium generally suggests encephalitic involvement, whereas prominent meningeal irritation indicates greater meningeal involvement. Cerebrospinal fluid (CSF) examination is therefore an important component of etiological evaluation and can provide useful information regarding inflammatory patterns and possible infectious causes.[5,6]

The etiological spectrum of AES varies considerably across geographical regions and populations. In India, AES has historically included Japanese encephalitis as well as a broad range of other viral and non-viral infections, and its etiological landscape has changed over time.[3] Surveillance studies from northern India have further demonstrated the heterogeneous nature of AES and the difficulty of establishing a specific etiology in every patient.[7] This etiological diversity makes systematic evaluation essential, particularly among critically ill children.

The distinction between viral and non-viral AES may be clinically relevant because the two groups can demonstrate different patterns of presentation and laboratory abnormalities. Viral infections may produce characteristic systemic or neurological manifestations, whereas bacterial, tubercular, and other non-viral infections may be associated with different meningeal and CSF findings. However, substantial overlap can occur, and clinical findings alone may not reliably establish the underlying cause. Consequently, integration of clinical examination with hematological, biochemical, microbiological, and CSF investigations is required for etiological assessment.[5,6]

AES may also progress rapidly to severe neurological and systemic complications requiring intensive care support. Previous studies have identified low Glasgow Coma Scale scores, raised intracranial pressure, seizures, cerebral edema, thrombocytopenia, hypotension, and requirement for mechanical ventilation among factors associated with adverse outcomes in encephalitis.[8,9] The wide spectrum of etiologies and severity

therefore makes early recognition and appropriate supportive management particularly important in pediatric AES.

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 of less than 14 days duration accompanied by altered mental status and/or new-onset seizures, excluding simple febrile seizures, were included. Altered neurological status included altered consciousness, lethargy or irritability, changes in personality or behaviour, confusion, disorientation, coma, inability to talk, focal neurological deficits, and cranial nerve palsy.

Exclusion Criteria

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

Initial Assessment and Stabilization

Immediately after admission, patients underwent rapid assessment and stabilization according to the standard treatment protocol. Airway was secured when indicated, including in children 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 mechanical ventilation were provided when required.

Intravenous access was established and appropriate samples were collected. Children with circulatory failure received fluid resuscitation, and inotropic support was initiated when required. Patients were evaluated for raised intracranial pressure or cerebral herniation. Fever or hypothermia was managed appropriately, while ongoing seizures were treated with benzodiazepines followed by phenytoin loading according to the study protocol.

Clinical Evaluation

After stabilization, written informed consent was obtained from the parent or guardian. A detailed clinical history and physical examination were performed. Vital parameters, including heart rate, respiratory rate, blood pressure, and temperature, were recorded. General examination included assessment for pallor, icterus, cyanosis, clubbing, lymphadenopathy, and edema. A detailed systemic examination, with particular emphasis on the central nervous system, was performed. Findings were recorded using a predesigned study proforma.

Laboratory and Etiological Evaluation

Investigations for etiological diagnosis were performed as early as possible after admission. Laboratory evaluation included hematological and biochemical investigations and CSF examination. CSF parameters assessed in the study included pressure, appearance, sugar, protein, cell count, cytological predominance, culture, and CBNAAT.

The thesis also reports the use of blood serology for Japanese encephalitis virus, herpes simplex virus, measles virus, and scrub typhus using commercially available IgM ELISA kits. According to the study protocol, once a non-viral etiology was established, the patient was not further evaluated for viral etiologies.

Classification Of Viral and Non-Viral AES

Patients were categorized into viral and non-viral AES based on CSF characteristics and etiological assessment. Of the 143 children, 80

(55.9%) were classified as viral AES and 63 (44.1%) as non-viral AES.

The thesis identifies dengue as the leading detected viral cause. Among the non-viral etiologies, pyogenic meningitis, tubercular meningitis, and scrub typhus were reported. Japanese encephalitis was not detected in the study population.

Assessment of Supportive Care and Outcome

Requirement for intensive supportive therapy was recorded. Inotropic support was required in 67 (46.9%) children, while 44 (30.8%) required ventilatory support. Ventilatory support was required in 24 (30.0%) children with viral AES and 20 (31.7%) with non-viral AES.

Mortality was assessed as an outcome. Overall, 28 (19.6%) children died. The thesis reports deaths among children with dengue, pyogenic meningitis, tubercular meningitis, scrub typhus, measles, and cases in which a specific viral etiology could not be identified.

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 states that univariate and multivariate logistic regression were used for assessment of predictors. Data were entered into Microsoft Excel and analyzed using SPSS version 22.0.

RESULTS

Table 1. Distribution Of Viral and Non-Viral AES (N=143)

Type of AES	Number	Percentage
Viral AES	80	55.9
Non-viral AES	63	44.1
Total	143	100.0

Table 2. Major Etiological Categories Reported in the Study

Etiological category	n (%)
Viral AES	80 (55.9)
Pyogenic meningitis	29 (20.3)
Tubercular meningitis	26 (18.2)
Scrub typhus	6 (4.2)
Japanese encephalitis	0
Total study population	143 (100.0)

Table 3. Clinical And Laboratory Characteristics Reported as Predominant in Viral and Non-Viral AES

Viral AES	Non-viral AES
Rash	Vomiting
Extrapyramidal manifestations	Undernutrition
Edema	Meningeal signs
Decreased TLC	Cranial nerve palsy
Decreased platelet count	Increased TLC
	Hyponatremia

Table 4. Requirement For Supportive Care Among Children With AES

Support requirement	Viral AES (n=80)	Non-viral AES (n=63)	Overall (n=143)
Ventilatory support	24 (30.0%)	20 (31.7%)	44 (30.8%)
Inotropic support	NR	NR	67 (46.9%)

Table 5. Mortality According to Reported Etiological Categories

Etiology/category	Reported deaths
Dengue	8
Pyogenic meningitis	5
Tubercular meningitis	4
Unidentified specific viral etiology	7
Overall mortality	28 (19.6%)

DISCUSSION

The present study compared the etiological profile, clinical characteristics, supportive care requirements, and outcomes of viral and non-viral acute encephalitis syndrome (AES) in children. Among 143 children, viral AES accounted for 80 (55.9%) cases, while 63 (44.1%) were categorized as non-viral AES. This predominance of viral AES is consistent with earlier observations that viral infections constitute an important proportion of childhood AES. The thesis cites previous studies reporting viral meningoencephalitis in approximately 45% of cases and another study reporting viral encephalitis in 69%.[10,11]

The etiological spectrum in the present study was heterogeneous. Dengue was reported as the leading identified viral cause. Among non-viral etiologies, pyogenic meningitis was identified in 29 (20.3%) children and tubercular meningitis in 26 (18.2%), while scrub typhus was reported in 6 (4.2%). Japanese encephalitis was not detected in any patient. A substantial number of viral cases remained without identification of a specific viral agent, demonstrating the diagnostic difficulty associated with AES when testing is directed toward a limited panel of pathogens.

Clinical and laboratory patterns differed descriptively between viral and non-viral AES. Viral AES was characterized by greater predominance of rash, extrapyramidal manifestations, edema, decreased total leukocyte count, and decreased platelet count. Conversely, vomiting, undernutrition,

meningeal signs, cranial nerve palsy, increased leukocyte count, and hyponatremia were more prominent in non-viral AES. These differences suggest that a combination of systemic, neurological, and laboratory findings may assist in directing etiological investigations. However, considerable clinical overlap between different causes of AES remains possible, and the thesis does not provide group-specific numerical comparisons or statistical significance for these individual characteristics.

A considerable proportion of children required intensive supportive management. Inotropic support was required in 67 (46.9%) patients, and 44 (30.8%) required ventilatory support. Ventilation was required in 24 (30.0%) children with viral AES and 20 (31.7%) with non-viral AES, indicating a similar descriptive requirement in the two groups. Previous work cited in the thesis reported ventilatory support in 59.2% of encephalitis patients, while another study reported assisted ventilation in 35.3% of viral and 20.8% of non-viral AES cases.[9,12] Differences between studies may reflect variation in disease severity, etiology, referral patterns, and intensive care populations.

CONCLUSION

Viral acute encephalitis syndrome was more common than non-viral AES in the present pediatric cohort, accounting for 55.9% and 44.1% of cases, respectively. Dengue was the leading identified viral etiology, whereas pyogenic meningitis and tubercular meningitis were important non-viral causes. Viral and non-

viral AES demonstrated differing clinical and laboratory patterns. A substantial proportion of children required intensive supportive care, with 46.9% requiring inotropic support and 30.8% requiring ventilatory support. Overall mortality was 19.6%. These findings highlight the heterogeneous and potentially severe nature of childhood AES and emphasize the importance of early clinical recognition, systematic etiological evaluation, prompt stabilization, and appropriate supportive management.

REFERENCES

1. Prober CG, Srinivas NS. Viral meningoencephalitis. In: Kliegman RM, Stanton BF, St Geme JW, Schor NF, editors. *Nelson Textbook of Pediatrics*. Elsevier India; 2016. p. 2946-7.
2. Khetsuriani N, Holman RC, Anderson LJ. Burden of encephalitis-associated hospitalizations in the United States, 1988-1997. *Clin Infect Dis*. 2002;35(2):175-82.
3. Joshi R, Kalantri SP, Reingold A, Colford JM Jr. Changing landscape of acute encephalitis syndrome in India: a systematic review. *Natl Med J India*. 2012;25(4):212-20.
4. World Health Organization. Acute encephalitis syndrome. Japanese encephalitis surveillance standards. 2006.
5. Venkatesan A, Tunkel AR, Bloch KC, Luring AS, Sejvar J, Bitnun A, et al. Case definitions, diagnostic algorithms, and priorities in encephalitis: consensus statement of the International Encephalitis Consortium. *Clin Infect Dis*. 2013;57(8):1114-28.
6. Shukla B, Aguilera EA, Salazar L, Wootton SH, Kaewpoowat Q, Hasbun R. Aseptic meningitis in adults and children: diagnostic and management challenges. *J Clin Virol*. 2017;94:110-4.
7. Kakkar M, Rogawski ET, Abbas SS, Chaturvedi S, Dhole TN, Hossain SS, Krishnan SK. Acute encephalitis syndrome surveillance, Kushinagar district, Uttar Pradesh, India, 2011-2012. *Emerg Infect Dis*. 2013;19(9):1361-7.
8. Thakur KT, Motta M, Asemota AO, Kirsch HL, Benavides DR, Schneider EB, et al. Predictors of outcome in acute encephalitis. *Neurology*. 2013;81(9):793-800.
9. Karande S, Gupta V, Kulkarni M, Joshi A. Prognostic clinical variables in childhood tuberculous meningitis: an experience from Mumbai, India. *Neurol India*. 2005;53(2):191.
10. Potharaju NR, Reilly DP. Reducing case fatality rate of acute encephalitis syndrome in developing countries. *J Pediatr Neurol*. 2012;10:1-11.
11. Hrishikesh AP, Sethuraman M. Cerebrospinal fluid analysis and interpretation in neurocritical care for acute neurological conditions. *Indian J Crit Care Med*. 2019;23(Suppl 2):S115.
12. Singhi SC, Singhi PD, Srinivas B, Narakesri HP, Ganguli NK, Sally R, Walia BN. The fluid restriction does not improve the outcome acute meningitis. *Pediatr Infect Dis J*. 1995;14(6):495-503.