Section: Paediatrics

Original Research Article

STUDY OF ACUTE ENCEPHALITIS SYNDROME IN CHILDREN ADMITTED AT PICU IN GUJARAT

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Corresponding Author:
Dr. Nilesh T,
Email: dr.nilesh27@yahoo.com

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Shivendra Varshney[1], Nilesh T[2], Umang Patel[3]

[1]Consultant Paediatrician, Subhanpura Hospital Vadodara, Gujarat, India.
[2]Assistant Professor, Department of Paediatrics, Gujarat Medical Education and Research Society Medical College Gotri Vadodara, Gujarat, India.
[3]Professor, Department of Forensic Medicine and Toxicology, Smt Bhikhiben Kanjibhai Shah Medical College and Research Centre Sumandeep Vidyapeeth Deemed to be University Vadodara, Gujarat, India.

Abstract

Background: Acute encephalitis syndrome is a medical emergency and needs to be admitted to the NICU. Acute onset of fever and changes in mental status, which include confusion, disorientation, coma, or inability to talk, create phobia among parents and challenge the paediatrician. **Materials and Methods:** 50 children aged between 1 month to 12 years admitted to the PICU were studied. Blood examinations included CBC, electrolyte, PS study, and CSF for cytological study. The biochemical analysis involved the use of PFB and gram stain. Viral analysis was done in CSF, i.e., HSV-I and II, JEV, dengue, and enterovirus, a neuroimaging study was carried out to find out the aetiology of AES. **Results:** The clinical manifestation included 50 (100%) fever, 36 (72%) altered sensorium, 35 (70%) convulsion, 13 (26%) headache, 3 (6%) excessive crying, 21 (42%) vomiting, 16 (32%) had pupillodema, 3 (6%) had CVS, 3 (6%) had R.S., and 4 (8%) abdominal complications, 25 (50%) viral aetiologies, 3 (6%) pyogenic, 4 (8%) tuberculosis, 12 (24%) dengue encephalitis, 3 (6%) cerebral malaria, 3 (6%) cerebral malaria, 2 (4%) other, 1 (2%) unknown. Out of 50, 5 deaths were recorded. **Conclusion:** Out of 50, 5 (10%) deaths occurred; hence, AES should be treated as a medical emergency; prompt identification and proper treatment prevent the morbidity and mortality in children with AES.

INTRODUCTION

Acute Encephalitic Syndrome (AES) is an intracranial infection that is life-threatening. Infections in the central nervous system are caused by various microorganisms with clinical manifestations such as meningitis, encephalitis, and pynogenic infections such as emphysema and brain abscess. Encephalitis is a medical emergency that needs to be admitted at PICUC.[1] Viruses are one of the main causes of acute encephalitis in India. Apart from viral encephalitis, severe forms of leptospirosis and toxoplasmosis can cause AEC. The causative agent of AES varies with season and geographical location and predominantly affects population below 15 years.[2]

Japanese encephalitis and dengue virus are the chief causative agents of AES. The Etiology of AES is an ongoing puzzle. Recently, an association was found between ingestion of litchi fruit and encephalitis and death. A virologist found that death may be because of hypoglycaemia.[3] Another unconfirmed report mentioned that alpha-cypermethrin was present in litchi fruit.

The complications of AES include motor incordination, convulsive disorders, total or partial deafness, and behavioral disturbances, which become a clinical challenge to the pediatrician; hence, an attempt is made to rule out the etiology, virology, and clinical manifestations in the children below 15 years.[4]

MATERIALS AND METHODS

50 Children 1 month to 12 years of age admitted at the Gujarat Medical Education and Research Society Medical College, Goitri Vadodhara, were studied.

Inclusive Criteria: The cases admitted with symptoms of AES In cases below 12 years of age, with the onset of fever, patients' mental status changes, such as confusion, disorientation, or coma, and they have an inability to talk. Their parents or guardians gave written consent for admittance and treatment and were included in the study.

Exclusion Criteria: Patients with febrile seizures Toxic encephalopathy children with central nervous

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system (CNS) malformations and CSF rhinorrhea and meningiocele were excluded from the study.

Method: A detailed history was taken and relevant factors were recorded. Demographic details were taken into consideration to find out the endemicity of any particular etiological agent.

Immunization status was recorded in the proforma outcome, which mainly includes mortality, and any sequelae were noted. Blood investigations included CBC electrolytes, peripheral smears were analyzed, neuroimaging was done, and abnormalities were analyzed and noted. CSF was sent for cytological study. Biochemical analysis, AFB, and gram staining. Viral analysis was done in CSF for HSV-I and II, JEV, dengue, and enteroviruses.

Serum was also tested for IgM Elisa for HIV, JE, CMV, and dengue. The survivors were followed up for any neurological sequelae. Out of 60 patients, 6 (10%) died.

The duration of the study was from January 2023 to December 2023.

The statistical analysis: Various clinical features and distribution of AES patients according to aetiology detection virus in AES patients were classified with percentage laboratory findings of AES viral; AES (other than dengue) were compared with normal AES with a t test, and significant results were noted. The statistical analysis was carried out in SPSS software. The ratio of male children with female children was 2:1.

RESULTS

Table 1: Clinical manifestations of AES patients: -

50 (100%) fever, 36 (72%) altered sensorium, 35 (70%) convulsion, 13 (26%) headache, 3 (6%) excessive cry, 12 (24%) altered behaviors, 21 (42%) vomiting, 3 (6%) neurodeficiency, 2 (4%) extra pyramid palsy, 34 (68%) were normal fundoscopy, 16 (32%) had papillodema, 3 (6%) had CVS abnormality, 3 (6%) had RS abnormalities, and 4 (8%) had abdomen abnormalities.

Table 2: Study to Aetiology of AES Patients:

25 (50%) viral aetiology (other than dengue fever), 3 (6%) pyogenic, 4 (8%) Tuberculosis, 12 (24%) dengue encephalitis, 3 (6%) cerebral malaria, 2 (4%) other, 1 (2%) unknown

Table 3: Comparative Study of Laboratory Findings:

- 10 (±2.6) in viral AES, (other than dengue), 10.8 (± 2.4) in normal AES, t test was 1.1 and p > 0.026
- TLC: 12.6 (± 4.6) in viral AES, 12.4 (±4.2) in normal AES, t test was 0.16 and p > 0.87.
- Platelet lakh/cun: 1.6 (± 0.9) in viral AES, 1.8 (±0.5) in normal AES, t test was 0.96 and p > 0.34.
- Total Serum Bilirubin (mg/dl): 0.8 (± 0.2) in viral AES, 1.8 (±0.4) in normal AES, t test was 11.3 and p<0.01 (p value is highly significant).
- Total serum protein (g/dl): 7.6 (± 1.5) in viral AES, 7.5 (±1.2) in normal AES, t test was 0.25 and p > 0.79.
- Serum albumin: 37 (± 0.4) in viral, 3.8 (±0.5) in normal AES, t test was 0.78 and p > 0.43.
- SGOT: 75 (± 18) in viral AES, 84 (±16) in normal AES, t test was 1.86 and p > 0.68.
- SGPT: 65 (± 20) in viral AES, 68 (±24) in normal AES, t test was 0.48 and p > 0.62.
- In the CSF study, cell count was 80.4 (± 24.2) in viral AES and 108 (±30.2) in normal AES, t test was 3.57 and p<0.001.
- Sugar level: 56.2 (± 15.2) in viral AES, 44.4 (±12.6) in normal AES, t test was 2.97 and p<0.001.
- Protein level: 98.6 (± 12.6) in viral AES, 118.5 (±20.5) in normal AES, t test was 4.17 and p<0.001.

Table 4: Study of virus detected from CSF causing AES: -

9 (34.6%) HSV-I, 5 (19.2%) HSV-II, 4 (15.3%) HSC (I and II), 3 (11.5%) measles, 2 (7.4%) JEV, 1 (3.8%) dengue, 1 (3.8%) VAV, 1 (3.8%) enterovirus.

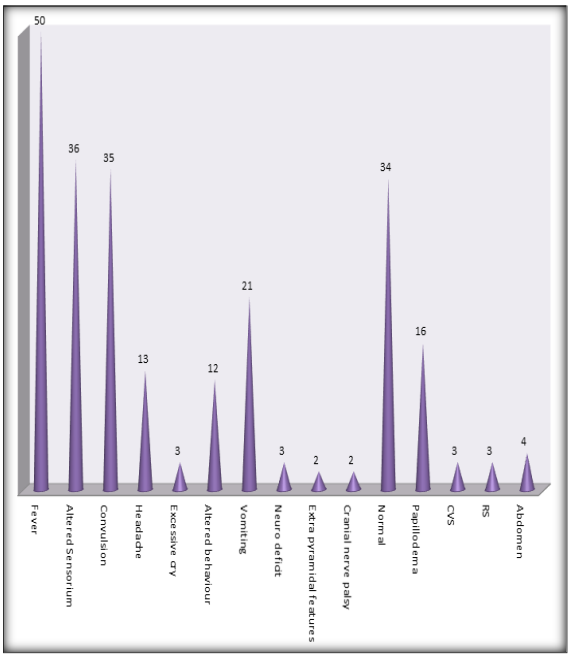

Figure 1: Clinical Manifestations of AES patients

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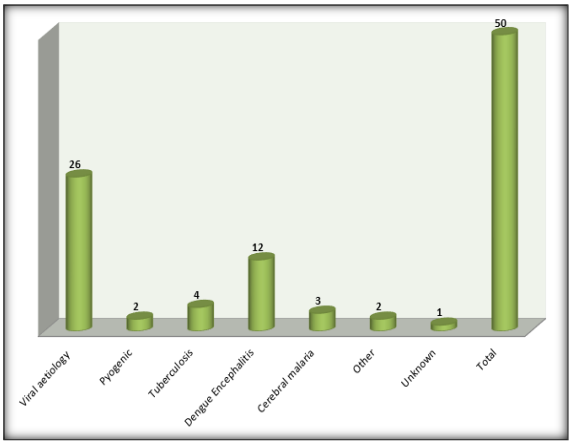

Figure 2: Study of Aetiology of patients

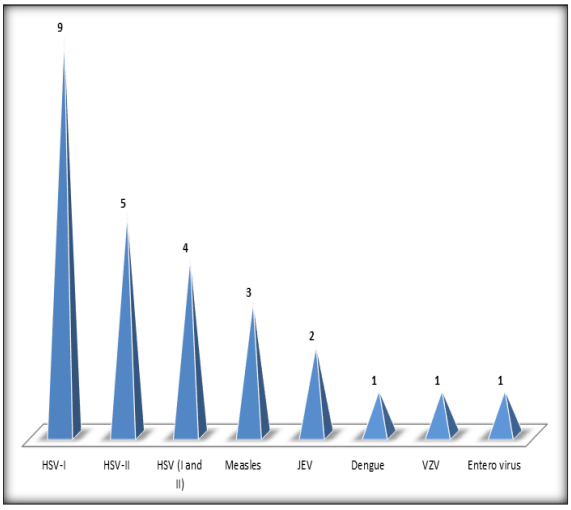

Figure 4: Defection of viruses from CSF causing AES

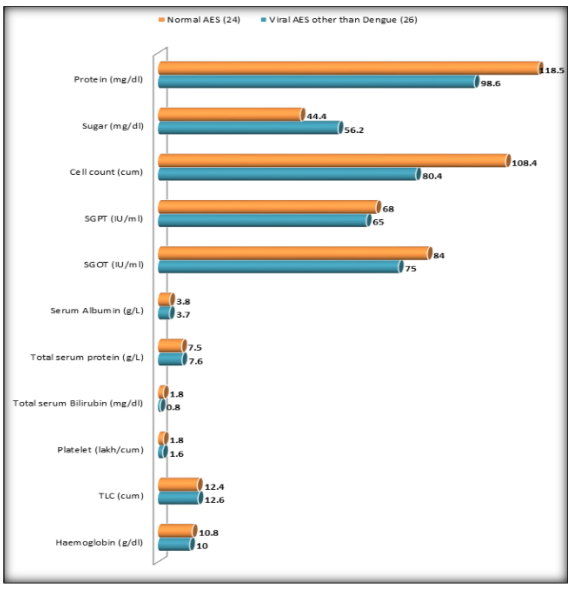

Figure 3: Comparative study of Laboratory findings of viral AES and normal AES

Table 1: Clinical Manifestations of AES patients **No of patients: 50**

Clinical Manifestations	Frequency	Percentage (%)
Fever	50	100
Altered Sensorium	36	72
Convulsion	35	70
Headache	13	26
Excessive cry	3	6
Altered behaviour	12	24
Vomiting	21	42
Neuro deficit	3	6
Extra pyramidal features	2	4
Cranial nerve palsy	2	4
Funduscopy		
a) Normal	34	68
b) Papillodema	16	32
Other system abnormality		
a) CVS	3	6
b) RS	3	6
c) Abdomen	4	8

Table 2: Study of Aetiology of patients **No of patients: 50**

Aetiology of patients	Number of patients	Percentage (%)
Viral aetiology (other than Dengue)	26	52
Pyogenic	2	4
Tuberculosis	4	8
Dengue Encephalitis	12	24

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Cerebral malaria	3	6
Other	2	4
Unknown	1	2
Total	50	100

Table 3: Comparative study of Laboratory findings of viral AES and normal AES

Variable	Viral AES other than Dengue (26)	Normal AES (24)	t test	p value
Haemoglobin (g/dl)	10.0 (± 2.6)	10.8 (± 2.4)	1.1	p>0.26
TLC (cum)	12.6 (±4.6)	12.4 (± 4.2)	0.16	p>0.87
Platelet (lakh/cum)	1.6 (±0.9)	1.8 (±0.5)	0.96	P<0.01
Total serum Bilirubin (mg/dl)	0.8 (±0.2)	1.8 (±0.4)	11.3	P<0.01 *
Total serum protein (g/L)	7.6 (±1.5)	7.5 (±1.2)	0.25	P>0.79
Serum Albumin (g/L)	3.7 (±0.4)	3.8 (±0.5)	0.78	p>0.1430
SGOT (IU/ml)	75 (±18)	84 (±16)	1.86	P>0.68
SGPT (IU/ml)	65 (±20)	68 (± 24)	0.48	p>0.62
CSF				
a) Cell count (cum)	80.4 (±24.2)	108.4 (±30.2)	3.57	P<0.001 *
b) Sugar (mg/dl)	56.2 (±15.2)	44.4 (±12.6)	2.97	P<0.001 *
c) Protein (mg/dl)	98.6 (±12.6)	118.5 (±20.5)	4.17	P<0.001 *

*** p<0.001 is highly significant**

Table 4: Defection of viruses from CSF causing AES

Viral agent	Number of cases (26)
HSV-I	9 (34.6%)
HSV-II	5 (19.2%)
HSV (I and II)	4 (15.3%)
Measles	3 (11.5%)
JEV	2 (7.4%)
Dengue	1 (3.8%)
VZV	1 (3.8%)
Entero virus	1 (3.8%)

Dual infection was detected in 10 cases (HSV-I, HSV-II 5 cases, HSV-I and Measles in 4 cases, JEV with dengue in 1case)

HSV = Herpes simplex Virus, JEV = Japanese Encephalitis virus

VZV = Vircella zoster virus

DISCUSSION

Present study of AES in children admitted at PICU in Gujarat. The clinical features were 50 (100%) fever, 36 (72%) altered sensorium, 35 (70%) convulsion, 13 (26%) headache, 3 (6%) excessive cry, 12 (24%) altered behaviours, 21 (24.1%) vomiting, 3 (6%) neurodeficiency, 2 (4%) extra pyramidal features, 16 (32%) had papillodema, 3 (6%) CVS, 3 (6%) had RS, and 4 (8%) abdomen complications (Table 1). The etiology of patients was 26 (52%) viral etiology, and the remaining major etiologies were 12 (24%) dengue encephalitis and 4 (8%) tuberculosis (Table 2). In the comparison of laboratory findings of viral AES and normal AES study, CSF findings had a significant p value (p<0.001) (Table 3). In the detection of viruses from SCF causing AES, the majority of viruses were 9 (34.6%) HSV-I, 5 (19.2%) HCV-II, 4 (15.3%) HSV (I and II), 3 (11.5%) were measles, and 2 (7.4%) JEV, followed by dengue, VZV, and enterovirus one cases (Table 4). These findings are more or less in agreement with previous studies.[5,6,7]

It is known that the etiological diagnosis of AES cases represents a diagnostic challenge in many previous studies worldwide. Aetiology was not found in > 50% of cases in the present study, with viruses being the most common aetiology. This may be because of some difficulty in isolating the organism, delayed presentation of the cases to a tertiary health care facility, prior antiviral use (particularly in herpes virus cases), auto-immune phenomena leading to encephalitis, and no investigation of outbreak cases separately.

Although viruses are the major cause of AES, the type of virus also markedly differs in different parts of the world. For example, HSV is the most common viral agent for AES in China, the UK, Norway, Spain, and France.[8] JES is the predominant cause of AES in north and north-east India and Cambodia.[9] Although enterovirus has been increasingly recognized as an important pathogen in AES cases, This could be because of the fragility of these viruses or the inadequate diagnostic modality employed. Certain epidemiological factors, such as the presence of paddy fields post-monsoon and low socioeconomic status, are shown to promote transmission of JEV.[10]

It is reported that in acute febrile encephalopathy, or AES, the mortality is higher in viral cases. This could be due to non-specific manifestations leading to delayed presentation, no specific treatment for viruses, some viruses difficulty in establishing diagnoses early, leading to delayed treatment, and the common occurrence of complications (shock, respiratory failure, and bleeding).[11] Most of the

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specific etiological agents of encephalitis remain unknown due to the higher cost of viral markers in CSF and serum. Follow-up is lacking in our study, which may help find out long-term neurological deficits and other sequences in AES patients.

CONCLUSION

AES is an important cause of morbidity and mortality, especially during the monsoon and postmonsoon periods. Fever, altered sensorium, and convulsion were the important presenting features in AES cases. Viral encephalitis, along with dengue encephalitis, are important causes of AES, early stabilization, and the institution of supportive measures.

Limitation of Study: Owing to the tertiary location of the research centre, the small number of patients, and the lack of the latest technologies, we have limited findings and results.

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