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Original Research Article

Study of Encephalitis Syndrome in Children Admitted in PICU in North Karnataka

Vijay Kolhar[1], Srinivasrao G Shinde[2], Samruddhapoorn[3*], Pradeep R Reddy[4]

[1]Assistant Professor, Department of Paediatrics, ESIC Medical College and hospital Gulbarga, Karnataka-585106

[2]Assistant Professor, Department of Paediatrics, ESIC Medical College and hospital Gulbarga, Karnataka-585106

[3]Assistant Professor, Department of Paediatrics, ESIC Medical College and hospital Gulbarga, Karnataka-585106

[4]Assistant Professor, Department of Paediatrics, ESIC Medical College and hospital Gulbarga, Karnataka-585106

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Corresponding author: Dr. Samruddhapoorn
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Abstract:

Background: Encephalitis is a serious neurological condition that can result in admission to intensive care. The clinical course is variable, and severe disease can occur, necessitating admission to intensive care. However, there are no studies reporting data for children with all-cause encephalitis admitted to the PICU.

Method: 60 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 Biochemical analysis of PFB and gram stain. Viral analysis was done in CST, i.e., HSV-I and II, JEV, dengue, and enterovirus, A neuroimaging study was carried out to find out the aetiology of AES.

Results: Clinical features were 60 (100%) fever, 44 (73%) altered sensorium, 42 (70%) convulsion, 16 (26.6%) headache, 3 (5%) excessive crying, 14 (23%) altered behaviour, 25 (41.6%) vomiting, and 3 (5%) neuro deficiency. 2 (3.3%) extrapyramidal features, 2 (3.3%) cranial nerve palsy funduscopy studies had 19 (31.6%) papillodema, 4 (6.6%) CVS, 4 (6.6%) RS, and 5 (8.3%) abdomen. The aetiology was 31 (51.6%) viral (other than dengue), 3 (5%) pyogenic, 4 (6.6%) tuberculosis, 14 (23.3%) dengue encephalitis, 4 (6.6%) cerebral malaria The CSF study had 11 (35.4%) HSV-I, 7 (22.5%) HSV-II, 6 (19.3%) HSV (I and II), 4 (12.9%) measles, 2 (6.4%) JEV, 2 (6.4%) dengue, 1 (3.2%) VZV, and 1 (3.2%) enterovirus, and out of 60, 6 (10%) deaths occurred.

Conclusion: The present pragmatic data suggests that, encephalitis places a significant burden on the health care system. More work is needed to improve outcomes for children with encephalitis.

Keywords: Acute Encephalitis Syndrome, cerebra spinal fluid, seizures, morbidity and mortality, HSV-I and II.

Introduction

Infections of the central nervous system are one of the most common neurological emergencies in children, accounting for significant mortality and morbidity. Delay or inadequate treatment can cause serious sequelae or even death [1]. CNS infections causing inflammation of the meninges are termed "meningitis," and inflammation of the brain parenchyma is called “encephalitis," and most of them coexist and are termed "meningoencephalitis”. Many microbes can cause infection but are also influenced by the age and immune status of the host and the epidemiology of the pathogen. In general, viral infections of the CNS are more common than bacterial infections, which in turn are more common than fungal and parasitic infections [2].

Acute Encephalitis Syndrome (AES) poses a great public health problem in the Diaspora. It has been estimated to have around 50,000 cases and 10,000 deaths annually [3]. It occurs in both epidemics and sporadically. There is a seasonal and geographical variation in the causative organisms too. Viruses are the most common cause of acute encephalitis syndrome worldwide [4].

Hence, an attempt is made to determine the profile of children admitted with acute encephalitis syndrome (AES), which includes aetiology, clinical presentation, outcome, and CSF analysis in different age groups of children.

Material and Method

Children 1 month to 12 years of age admitted at the PICU of ESIC Medical College Hospital, Gulbarga-585106, Karnataka, 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, were included in the study.

Exclusion Criteria: Patients with febrile seizures Toxic encephalopathy children with central nervous system (CNS) malformations and CSF Rhinorrhea, meningocele 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.

Immunisation status was recorded in the proforma outcome, which mainly includes mortality, and any sequelae were noted. Blood investigations included CBC electrolytes, peripheral smears were analysed, neuroimaging was done, and abnormalities were analysed 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 May 2022 to April 2023.

The Statistical analysis: Various clinical features, distribution 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 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.

Observation and Results

Table 1: Distribution of AES patients according to clinical features 60 (100%) fever, 44 (73.1%) altered sensorium, 42 (70%) convulsion, 16 (26.6%) headache, 3 (5%) excessive cry, 14 (23%) altered behaviours, 25 (41.6%) vomiting, 3 (5%)

neuro deficits, and 2 (3.3%) extra pyramid palsy. In a fundoscopic study, 41 (68.3%) were normal, 19 (31.6%) had papilloedema or other abnormalities – 4 (6.6%) CVS, 4 (6.6%) RS, and 5 (8.3%) abdomen.

Table 2: Distribution of AES patients according to aetiology 31 (51.6%) viral aetiology, 3 (5%) pyogenic, 4 (6.6%) Tuberculosis, 14 (23.3%) dengue encephalitis, 4 (6.6%) cerebral malaria, 3 (5%) other, 1 (1.6%) unknown

Table-3: Laboratory findings of AES were compared between viral AES, (other than dengue) and normal AES. Total serum bilirubin was 0.6 (± 0.5) in viral AES, 1.8 (±0.4) in normal AES, t test was 10.2 and p<0.001

In CSF study –

80. Cell count (cum) was 80.2 (± 25.2) in viral AES and 108.2 (±30.2) in normal AES, test was 3.8 and p<0.001

81. Sugar (mg/dl) 56.4 (± 14.2) in viral AES and 44.2 (±12.7) in Normal AES, test was 3.51 and p<0.001

82. Protein level (mg/dl) was 98.5 (± 12.8) in viral AES and 119.6 (±20.6) in Normal AES, test was 4.70 and p<0.001

Table-4: Detection of viruses from CSF causing AES

11 (35.4%) HSV-I, 7 (22.5%) HSV-II, 6 (19.3%) HSC I and II, 4 (19.3%) measles, 2 (6.4%) JEV, 1 (3.2%) dengue, 1 (3.2%) VAV, 1 (3.2%) Enterovirus.

Table 1: Distribution of AES patients according to clinical features

Clinical features	Frequency	Percentage
Fever	60	100
Altered Sensorium	44	73
Convulsion	42	70
Headache	16	26.6
Excessive cry	3	5
Altered behaviour	14	23
Vomiting	25	41.6
Neuro deficit	3	5
Extra pyramidal features	2	3.3
Cranial nerve palsy	2	3.3
Funduscopy		
a) Normal	41	68.3
b) Papillodema	19	31.6
Other system abnormality		
a) CVS	4	6.6
b) RS	4	6.6
c) Abdomen	5	8.3

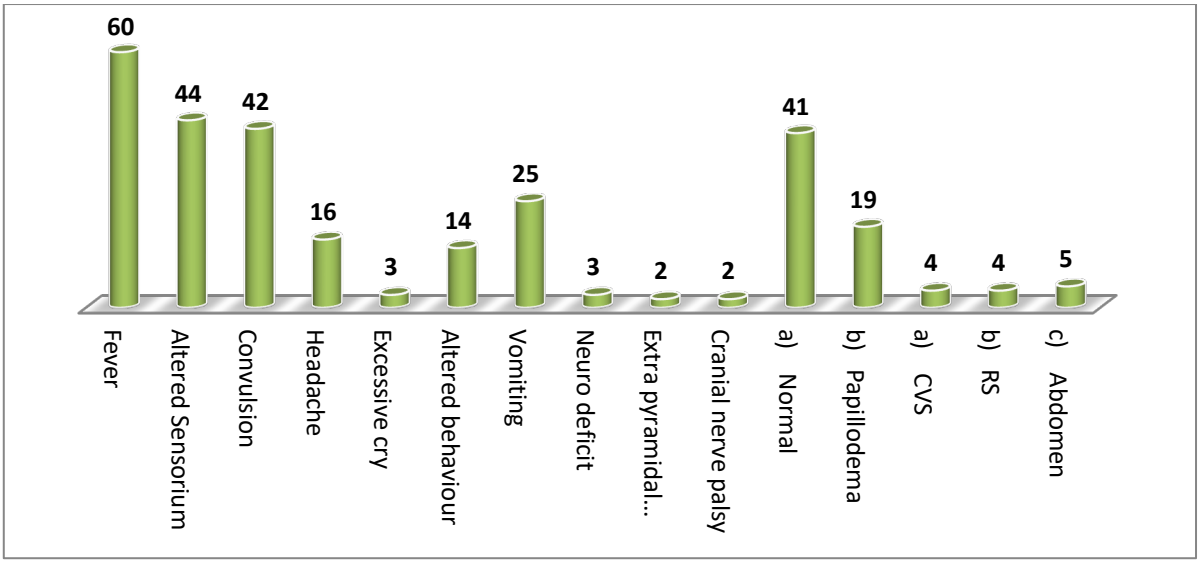

Figure 1: Distribution of AES patients according to clinical features

Table 2: Distribution of AES patients according to Aetiology

Aetiology of AES patients	Number of patients	Percentage
Viral aetiology (other than Dengue)	31	51.6
Pyogenic	3	5
Tuberculosis	4	6.6
Dengue Encephalitis	14	23.3
Cerebral malaria	4	6.6
Other	3	5
Unknown	1	1.6
Total	60	

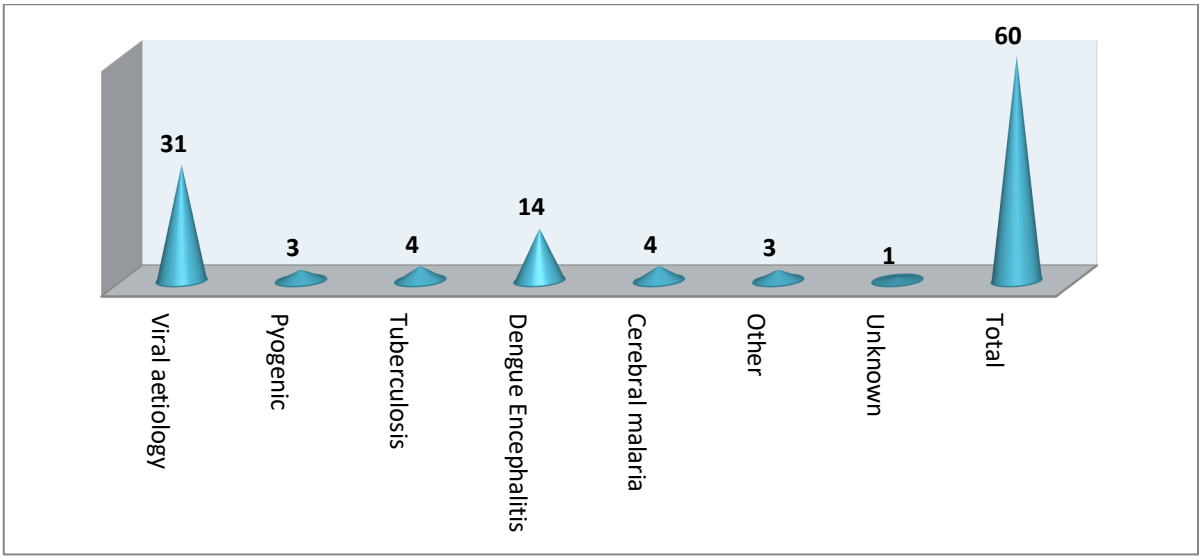

Figure 2: Distribution of AES patients according to Aetiology

Table 3: Laboratory findings of AES

Variable	Viral AES other than Dengue (31)	Normal AES (29)	t test	p value
Haemoglobin (g/dl)	10.0 (± 2.4)	10.6 (± 2.2)	- 0.01	0.31
TLC (cum)	12.5 (±4.4)	12.2 (± 4.2)	0.27	0.78

Platelet (lakh/cum)	1.5 (±0.8)	1.8 (±0.4)	2.57	0.01
Total serum Bilirubin (mg/dl)	0.6 (±2.5)	1.8 (±0.4)	10.2	P<0.001 *
Total serum protein (g/L)	7.7 (±1.4)	7.5 (±1.2)	0.59	P<0.55
Serum Albumin (g/L)	3.6 (±0.2)	3.7 (±0.6)	0.85	p>0.4
SGOT (IU/ml)	74 (±20)	82 (±18)	1.63	P<0.10
SGPT (IU/ml)	64 (±22)	69 (± 24)	0.83	p>0.40
CSF				
a) Cell count (cum)	80.2 (±25.2)	108.2 (±30.2)	3.88	P<0.001 *
b) Sugar (mg/dl)	56.4 (±14.2)	44.2 (±12.7)	3.51	P<0.001 *
c) Protein (mg/dl)	98.5 (±12.8)	119.6 (±20.6)	4.70	P<0.001 *

* = p<0.001 is highly significant

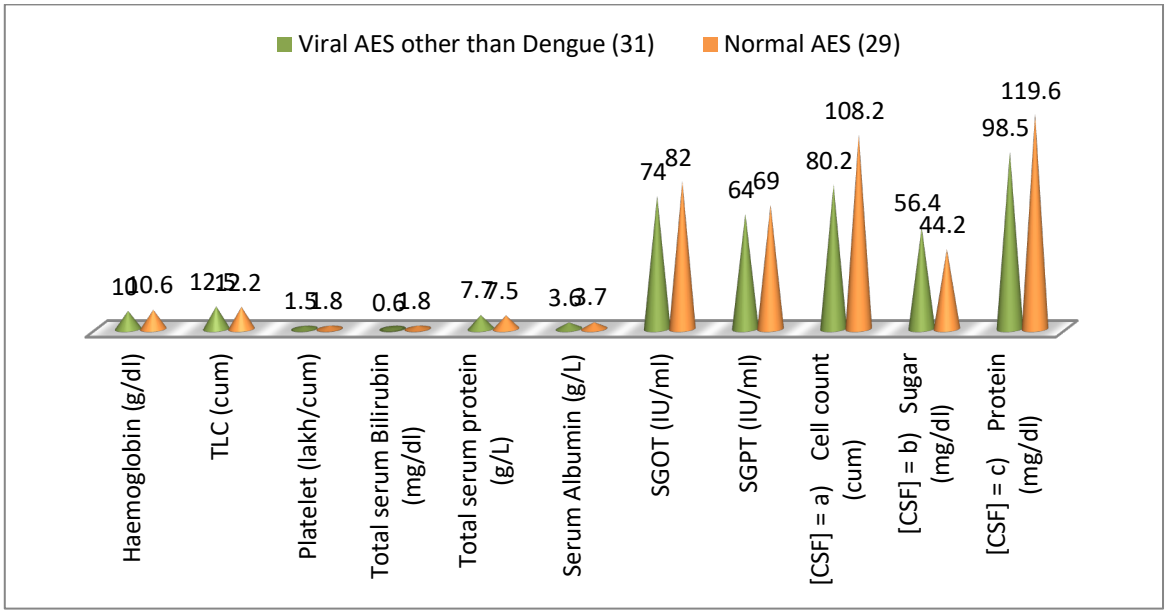

Figure 3: Laboratory findings of AES

Table 4: Defection of viruses from CSF causing AES

Viral agent	Number of cases (31)
HSV-I	11 (35.4%)
HSV-II	7 (22.5%)
HSV (I and II)	6 (19.3%)
Measles	4 (12.9%)
JEV	2 (6.4%)
Dengue	1 (3.2%)
VZV	1 (3.2%)
Entero virus	1 (3.2%)

Dual infection was detected in 12 cases (HSV-I, HSV-II 7 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

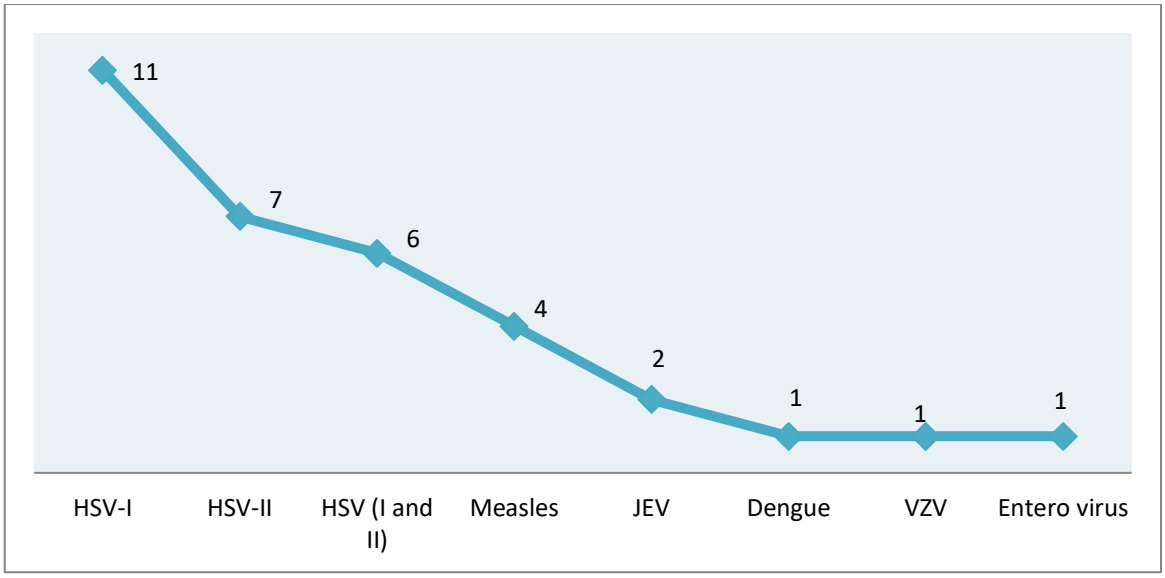

Figure 4: Defection of viruses from CSF causing AES

Discussion

Present study of encephalitis syndrome in children admitted to the PICU in North Karnataka. The clinical features were 60 (100%) fever, 44 (73%) altered sensorium, 42 (70%) convulsion, 16 (26.6%) headache, 3 (5%) excessive cry, 14 (23%) altered behaviours, 25 (41.6%) vomiting, 3 (5%) neuro deficit, 2 (5%) extra pyramidal features, 2 (3.3%) cranial nerve palsy, 19 (31.6%) papillodema, 4 (6.6%) CVS, 4 (6.6%) RS, and 5 (8.3%) abdomen (Table-1). The aetiology of AES was 31 (51.6%) viral aetiology (other than dengue), 3 (5%) pyogenic, 4 (6.6%) tuberculosis, 14 (2.3%) dengue encephalitis, 4 (6.6%) cerebral malaria, 3 (5%) other viruses, 1 (1.6%) unknown (Table-2). In a comparative study of viral AES and normal AES Total serum bilirubin (mg/dl) 0.6 (± 0.5) in viral AES 1.8 (± 0.4) normal AES, t test was 10.2 and p<0.001 and CSF study of cell count cum 80.2 (± 25.2) in viral AES, 108 (± 30.2) in normal AES, t test was 3.88 and p<0.001, sugar level in CSF 56.4 (±14.2) in viral AES, 44.2 (± 12.7%), t test was 3.51 and p<0.001. Protein level in CSF 98.5 (±12.8) in viral AES 119 (±20.6) in normal AES, t test was 4.70 and p<0.001 (Table-3). In CSF 11 (35.4%) HSV-I, 7 (22.5%) HCV-II, 6 (19.3%) HSV (I and II), 4 (12.9%) measles, 2 (6.4%) JEV, 1 (3.2%) dengue, 1 (3.2%) VZV, 1 (3.2%) enterovirus (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 recognised 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 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.

Summary and 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 stabilisation 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.

This research was approved by the ethical committee of AES Medical College and Hospital, Gulbarga, Karnataka, 585101.

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