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Clinical Study of acute encephalitis syndrome in hospitalized children

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Abstract

Background: Acute encephalitis is the clinical diagnosis of children with acute onset of symptoms and signs of inflammatory lesions in the brain. It must be diagnosed promptly for saving life and preserving brain functions. Objectives of this study was to determine the profile and outcome of children admitted with Acute Encephalitis Syndrome (AES)

Methods: Study consist of a prospective cohort study analysis of hospital records of children up to 18 years of age admitted with a diagnosis of AES in the Pediatric ward, J L N medical college, Ajmer from may 2019 to april2020.

Results: In a total of 96 patients of AES, clinical features like fever (100%), altered sensorium (100%), convulsion (52.08%), headache (60.4%) and vomiting (83%). The average Glasgow coma scale at admission was 8. Encephalitis (52.08%) were documented significantly more in males as compared to females. seventy-one cases were discharged, twenty five expired.

Conclusions: To conclude AES is not an uncommon reason for hospital admission of children. Clinically fever and altered sensorium were universal symptoms. Factor associated with poor prognosis were young age group, male gender and CSF pleocytosis.

Keywords: Acute encephalitis syndrome, Glasgow coma scale.

Introduction

Acute Encephalitis Syndrome (AES) is a leading cause of mortality and morbidity in children characterized as acute onset of fever and change in the mental status (mental condition, disorientation, delirium, or coma) and new onset of seizures in the person of any age at any time of the year.1 AES may present with encephalitis, meningoencephalitis, or meningitis and may be caused by viruses, bacteria, mycobacteria, rickettsia, and rarely by toxoplasma.

Japanese B encephalitis (JE) is the major cause in India. Knowing the wide range of causal agents and the rapid rate of neurological deterioration due to pathogenesis, clinicians face the challenge of small window period between diagnosis and treatment. As a step to control JE, the World Health Organization (WHO) is maintaining a set of standards for JE surveillance, which requires identification of patients with AES(2 to 5)

Paediatricians who treat these children should be aware of how to manage a child with suspected encephalitis, as specific antiviral therapy is lifesaving in some diseases, and these should be diagnosed without delay.

These guidelines have been developed to aid the paediatrician in the management of children with suspected viral encephalitis, in both sporadic and epidemic settings in India.

These guidelines do not cover viral encephalitis in the neonatal period and in immuno-compromised children, Rabies encephalitis, and chronic viral encephalitis such as Sub-acute sclerosing pan encephalitis(SSPE).

The present study was carried out to evaluate the clinical profile of Pediatric AES cases, to determine both prevalence and outcome of encephalitis.

Methods

The current study was carried out in children with a diagnosis of AES admitted in the Pediatric ward, JLN Medical College, Ajmer, Rajasthan, India.

This prospective cohort study (hospital records) on 96 children up to 18 years of age from may2019 to April 2020. AES diagnosed according to the WHO case definition.

Case records of each child were analyzed in detail and data recorded for history, examination, investigation, and outcome.

Patients were categorized based on predominant clinical-investigational picture suggestive of encephalitis.

Inclusion criteria

• All Pediatric patients up to 18 years of age, fulfilling the following symptoms were included in the study.

1. Pyrexia-temperature >37.5*C
2. altered sensorium-GCS

Exclusion criteria

1. Tubercular meningitis
2. Aseptic meningitis
3. Bacterial meningitis
4. Intra cranial hemorrhage
5. Intra cranial trauma
6. Metabolic disorder/poisoning/any other recognisable cause of neurologic illness.
7. Those patient not giving consent

• Data on prevalence and clinical parameters were expressed as a percentage, and a student's t-test performed to identify significant change deriving p-value using SPSS version 16.0software.

Evaluation and management of children suffering from acute Encephalitis syndrome

Step I: Rapid assessment and stabilization.

• Establish and maintain airway: Intubate if GCS score 8, impaired airway reflexes, abnormal respiratory pattern, signs of raised Intracranial pressure, oxygen saturation <92 % despite high flow oxygen, and fluid refractory shock

• adequate Ventilation, Oxygenation

• Circulation: Secure IV access, take samples (CBC, Blood sugar, KFT, LFT, electrolytes, blood gas, lactate, PS and RDT for a malarial parasite, serology for viruses), Fluid bolus if in circulatory failure (20 mL/kg NS), inotropes if required

• Identify signs of cerebral herniation or raised ICP

• Temperature: treat fever and hypothermia

• Treat ongoing seizures- Benzodiazepine, followed by a phenytoin loading dose

Step II: Clinical evaluation: History and examination

History and examination

Step III: Investigation/ Samples to be collected

• CSF

• Blood/serum, Urine

• CT/MRI, avoid sedation

• Throat swab, nasopharyngeal swab.

Step IV: Empirical treatment (must be started if CSF cannot be done/report will take time and patient sick)

• Ceftriaxone.

• Acyclovir (use in all suspected sporadic viral encephalitis)

• Artesunate (stop if peripheral smear and RDT are negative)

Step V: Supportive care and treatment.

• Maintain euglycemia, Control fever, Maintain hydration.

• Treat raised intracranial pressure, mild head-end elevation15-30°.

• Treat seizures; Give anticonvulsant if the history of seizures or if GCS <8 , or if the child had features of raised ICT.

• Steroids: Pulse steroids (methylprednisolone or dexamethasone) should be given in children with suspected ADEM.

Step VI: Prevention and treatment of complications and rehabilitation.

• Physiotherapy, posture change, Prevent bed sores, and exposure keratitis.

• Complications: Aspiration pneumonia, no socomial infections. Important Points in the History of a Child with AES

• Fever, headache, vomiting, seizures, abnormal posturing

• Altered behaviour, cognition, personality changes, altered consciousness

• Prodromal symptoms- flu-like illness, diarrhea

• Rash, vesicles, past history of chickenpox

• Residence of a child: Rural/urban, endemic for cerebral malaria, any epidemic of AES in neighbourhood

• History of animal contact, insect bite, dog bite

• Drug or toxin exposure- enquire for the presence of any drugs at home

• Recent history of travel

• History of trauma

• Personal or family history of seizure disorder

• Recent immunizations

• H/o recurrent episodes of encephalopathy: These are characteristic of some inborn errors of metabolism (urea cycle defects, organic acidemias, and fatty acid oxidation defects), but may also be present in migraine, epilepsy, substance abuse, and Munchausen syndrome by proxy

• Other concurrent systemic illness, e.g., jaundice (hepatic failure), pneumonia (hypoxic encephalopathy), diarrhea (dy selector lytemia), dysentery (shigella encephalopathy)

• Past h/o medical illness: Diabetes, congenital heart disease, chronic kidney or liver disease

• Family h/o previous infant/child deaths

• Pre-morbid developmental and neurological status of the child

• Risk factors for immunodeficiency- HIV risk factors, cancer treatment, steroid/ immuno suppressant treatment.

Treatment-Empirical treatment must be started, pending the results of investigations. A broad spectrum antibiotic such as ceftriaxone must be given, which can be stopped if no evidence of bacterial meningitis is forthcoming.

Table 1: MRI Findings in viral encephalitis and some mimickers.

Etiology	MRI Finding
Herpes simplex encephalitis	Abnormal signal intensity in medial temporal lobe, cingulate gyrus, and orbital surface of frontal lobes
Japanese B encephalitis	Abnormal signal intensity in thalami (87-94%), substantianigra, and basal Ganglia
EV 71	Abnormal signal intensity in the dorsal pons, medulla, midbrain, and dentate nuclei of the cerebellum; gigh-signal lesions can also be found in the anterior horn cells of spinal cord in patients with acute flaccid paralysis
Chandipura virus	Normal
Nipah virus	Focal subcortical and deep white matter and gray matter lesions; small hyperintense lesions in the white matter, cortex, pons and cerebral peduncles have also been seen.
Varicella	Multifocal abnormalities in cortex, associated cerebellitis, vasculitis and vasculopathy
Acute disseminated encephalomyelitis	Multifocal abnormalities in subcortical white matter; involvement of thalami, basal ganglia, and brainstem also seen
West Nile virus	Abnormalities in deep grey matter and brainstem (50%); white matter lesions mimicking demyelination may also be seen; meningeal involvement on contrast enhanced images.

The table shows suggestive MRI findings present in some etiologist of viral encephalitis such as Herpes simplex encephalitis, JE, enterovirus encephalitis. MRI may show non-specific features of viral encephalitis, such as cortical hyperintensities and cerebral edema. MRI is also useful for diagnosing alternative etiologist such as Acute disseminated encephalomyelitis, and antibody associated encephalopathies.

Acyclovir should be stopped if an alternative diagnosis has been made, or HSV PCR in the CSF is negative and MRI is normal. However,

if the CSF PCR for HSV or MRI have been performed very early after symptom onset (within 48 hours), these may be falsely negative. Hence, these studies should be repeated before stopping acyclovir if the clinical suspicion of HSE continues to be high.

Dose and duration of acyclovir in children with encephalitis- 3 months to 12 y (500mg/m2 8 hourly)<12 yr.: 10mg/Kg 8 hourly.

Confirmed cases

14-21 days intravenous treatment; Minimum 21 day for those aged 3 mon-12yr.

Where therapy was started empirically, stop acyclovir, if an alternative diagnosis is confirmed, or if HSV PCR in the CSF is negative on two occasions (24-48 hr apart) and MRI imaging does not suggest HSE.

Results

There were 96 cases of AES Pediatric patients up to 18 years of age during the study period fulfilling the WHO definition. Male and female patients were 50 and46 there was no statistical significance observed between them (Table 2).

Table 2: Distribution of total cases of meningitis/ encephalitis according to sex.

Diagnosis			Total (AES)
	Male	Female	
1. Encephalitis	50	46	96
p value	p<0.015		

Majority (45.83%) patients were from age group 0-5 years followed by 26.04% and21.87% patients from 6-10 years and 11-15 years age group.

Out of total 96 cases of AES, 71 were discharged, 25 expired. Eleven out 25 patients died in 0-5 years age group followed by 10 out 25 patients died in 6-10 years age group. 29 patients were discharged in 0-5 years followed by 16 patients in 11-15 years age group. 4 out of 7 patients were discharged with sequlae in 0-5 years age group. Ten male and fifteen female patients died. 31 female patients and 33 male patients were discharged. While 7 boys discharged with sequlae.

Discussion

WHO has given clinical case definition of AES so that these cases are subjected for confirmative diagnosis by IgM captured ELISA in blood and or CSF. JE is the single largest cause of viral encephalitis in the world.6 Clinical profile of AES patients in this study included vomiting, seizures, Glasgow coma scale (GCS) <8 meningeal irritation signs, neurological deficit . In our study most common complaints were fever (100%), altered sensorium (100%) followed by vomiting (83.33%), headache (60.41%), convulsion (52.08%), blurred vision (18.75%). In consistent with our results Gupta N et al, study observed vomiting in 41.4%, seizures in 79.3%, altered sensorium in 51.7%, signs of meningeal irritation in 17.2% and neurological deficit in 34.5% of their cases in the study done in hospitalized patients suspected of JE (7) .These findings are comparable with our present study Kumar et al, study described vomiting in 6.5%, meningeal signs in 35.1%, GCS<7 in 44.1%, extrapyramidal features in 31.1% and convulsions in 98.7% in JE epidemic in Eastern Uttar Pradesh(8).

In present study, majority (45.83%) patients were from age group 0-5 years followed by 26.04 % and 21.87%patients from6-10 years and 11-15 years age group respectively. The mean age was 7.25 years. In concordance with our results BasuRetal9foundthat out of142 children from age group(1month to 12 years and maximum) 62(43.66%)patients were from age group of 1year to 5 years In our study there were 52.08% Male and 47.91% Female patients. Similarly Basu R et al 9reported that82 (57.74%) cases were boys and 60(42.26%) cases were girls.

We found that 55.28% patients were from rural area 44.79% patients were from urban area. This can be attributed to the fact that Our centre caters to medical need of both urban as well as rural population. Similarly Kemble S et al 10found that Among 136 study subjects more than half were residing in rural area 80(58.8%) and 56 (41.2%) of them were from urban areas.

CSF Biochemical finding - CSF examination was done in 81patient, on CSF biochemical examination mean CSF Protein (mean=119.06, SD=192.63) level was raised while CSF Chloride (mean=120.40, SD= 19.55) level was normal . In our study 91.35% patients had normal CSF glucose (>2/3 CSF blood sugar ratio) and 8.64% patients had low CSF glucose (>2/3 CSF blood sugar ratio) and 8.64% patients had low CSF glucose 3 CSF blood sugar ratio) and 8.64% patients had low CSF glucose(<2/3 CSF blood sugar ratio).

A study by Ranjan A et al 11found that the mean CSF protein and glucose level were 35.38 and 65.5 mg/dl respectively. Similar study by Avabratha et al 12observed elevated protein in 74.67% study patient.

The protein concentration in CSF tend to be elevated, and concentration may be very high if brain destruction is extensive, as with HSV encephalitis. The glucose level is typically normal, although hypoglycorrhachia can occur with certain virus.

In present study CSF pleocytes was observed in 12 (14.8%) while it was normal in 69 (85.18%) patients. We found that 16 patients with normal CSF Cells died. 49 patients with normal CSF Cells were discharged and 4 such patients were discharged with sequelae. There is significant difference among these groups (p value is 0.005). Similar finding were also found by Khinchi Y R et al 13, Bandyopadhyay Bhaswati et al14, Avabratha et al 12and DongolS et al 15.

The CSF findings in Menin go encephalitis are characterized by a pleocytosis of leucocytosis with count typically <1000/mm3.Very early in the disease ,the cells are often polymorphonuclear ,whereas mononuclear cells predominant for the remainder of the duration of illness. This change in cell type often occur over 8-12 hr.

Neuroimaging was done in 24 patients .In half of patients it was normal, whereas temporal lobe signal changes were seen in 8(33.3%) patients in coronal T2 weighted MRI. In axial FLAIR MRI 2 (8.3%) patients were having thalamic involvement,1 (4.1%) of each was having basal ganglia with midbrain involvement and subcortical and periventricular white matter changes.

When SGPT/SGOT and PT-INR values were correlated with outcome then statically no significant difference between deranged and normal range was found.

Conclusion

To conclude AES is not an uncommon reason for hospital admission of children .In present study it accounted for about 1% of total admission during 1 year of study period. Demographically maximum number of cases belonged to 0-5 yr. age groups ,were boys, belonging to rural background and lower socioeconomic status. Clinically fever and altered sensorium were universal symptoms, and other symptoms and neurological signs were quite variable. Routine haematology and biochemical investigations were within normal limits in the most of these cases. Case fatality rate of 26.04% was observed .Factors associated with poor prognosis were younger age group male gender and CSF pleocytosis. In half of cases out of 24 patient in whom MRI could be done it was normal, Rest patients had variable neuroimaging findings.

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