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Assessment of Outcome of Encephalitis in Pediatric Intensive Care Unit

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Abstract

Introduction: Aim: To assess outcome of encephalitis in pediatric intensive care unit.
Methods: Eighty- four children age ranged 6-14 years of either gender with diagnosis of acute encephalitis admitted to pediatric intensive care unit were recruited. Patients' characteristics were recorded.
Results: Age group 6-8 years had 36, 9-11 years had 25 and 12-14 years had 23 children. There were 34 non- survivors and 50 survivors. Causative agent found to be Enterovirus in 12 and 35, Herpesvirus in 1 and 12, CMV in 2 and 4, Cosackie B1 in 1 and 3, Influenza & parainfluenza in 1 and 1 and Adenovirus in 0 and 5 in non- survivors and survivors respectively. Management done was inotropes in 34 and 7, IVIG in 20 and 10, anticonvulsant in 34 and 40, corticosteroid in 18 and 4. Mechanical ventilation was required in 34 and 25, PICU stay was 10 days and 4 days in non-survivors and survivors respectively. A significant difference was observed ($P< 0.05$).
Conclusion: A significant mortality was seen in pediatric encephalitis. Patients need extensive management to prevent deaths.

Key Words: Encephalitis, Intensive care unit, Influenza.

INTRODUCTION

Encephalitis is a serious acute infection/parainfection of the brain which is often associated with significant mortality and morbidity. Manifestations include fever, headache, altered/fluctuating mental state (confusion/drowsiness) and seizures.[1] Enteric, respiratory, herpes viruses and various endemic micro-organisms are the culprits. Even with extensive investigations, however, a viral or infectious etiology may not be identified.[2,3]

Encephalitis is challenging to manage given the diversity of clinical and epidemiologic features.[4] More than 100 infectious species have been identified as causative agents of meningoencephalitis, with a burgeoning of new infectious and autoimmune etiologies in the last decade. Despite advances in diagnosis, more than 50% of encephalitis cases remain cryptogenic, posing additional management challenges.[5]

Mortality in encephalitis patients is secondary to raised intracranial pressure (ICP), status epilepticus (SE), aspiration pneumonia and/or autonomic instability.[6] In dengue, chikungunya, malaria, leptospirosis and rickettsial infections, death may occur due to bleeding and refractory hypotension complicated by myocardial dysfunction.[7] The patients with dengue, JE and West Nile encephalitis may present with acute flaccid weakness that may contribute to respiratory failure. Infectious encephalitis is generally a monophasic illness, and many patients survive with variable outcome if they are supported by intensive care and mechanical ventilation (MV) whenever needed.[8]

Recent consensus statement of the International Encephalitis Consortium serves as a practical aid to clinicians evaluating patients with suspected encephalitis.[9] In the pediatric intensive care unit (PICU) setting, a significant number of patients with encephalitis die or result in residual neurologic impairments.[10] Considering this, the present study was aimed at assessing outcome of encephalitis in pediatric intensive care unit.

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MATERIALS AND METHODS

A total of eighty- four children age ranged 6-14 years of either gender with diagnosis of acute encephalitis admitted to pediatric intensive care unit were recruited. Parental consent was sorted with written permission. Ethical clearance committee approved the study.

Diagnosis of encephalitis was based on clinical evidence of cerebral dysfunction, inflammatory response or infection, or neuroimaging abnormalities according to recent international consensus. Cerebral dysfunction was defined as altered mental status or decreased or altered level of consciousness, lethargy or personality change lasting ≥24 hours without alternative cause identified, generalized or partial seizures not fully attributable to a pre-existing seizure disorder, and new onset of focal neurologic findings.

Isolation of etiologic agents from the CSF (if available), respiratory and stool specimens, neuroimaging (CT/MRI) and EEG studies. Etiologic agents were identified using conventional diagnostic methodology using antigen detection by immunofluorescence test, nucleic acid detection by PCR and isolation of organisms from cerebrospinal fluids was performed.

RESULTS

Table 1: Distribution of patients

Age group (Years)	Number	P value
6-8	36	>0.05
9-11	25	
12-14	23	

Age group 6-8 years had 36, 9-11 years had 25 and 12-14 years had 23 children [Table 1].

Table 2: Demographics of encephalitis patients

Variables	Parameters	Non-survivor (34)	Survivor (50)	P value
Causative agent	Enterovirus	12	35	<0.05
	Herpesvirus	1	12	
	CMV	2	4	
	Cosackie B1	1	3	
	Influenza & parainfluenza	1	1	
	Adenovirus	0	5	
Management	Inotropes	34	7	<0.05
	IVIG	20	10	
	Anticonvulsant	34	40	
	Corticosteroid	18	4	
Mechanical ventilation		34	25	
PICU stay (days)		10	4	<0.05

There were 34 non- survivors and 50 survivors. Causative agent found to be Enterovirus in 12 and 35, Herpesvirus in 1 and 12, CMV in 2 and 4, Cosackie B1 in 1 and 3, Influenza & parainfluenza in 1 and 1 and Adenovirus in 0 and 5 in non-survivors and survivors respectively. Management done was inotropes in 34 and 7, IVIG in 20 and 10, anticonvulsant in 34 and 40, corticosteroid in 18 and 4. Mechanical ventilation was required in 34 and 25, PICU stay was 10 days and 4 days in non- survivors and survivors respectively. A significant difference was observed (P< 0.05).

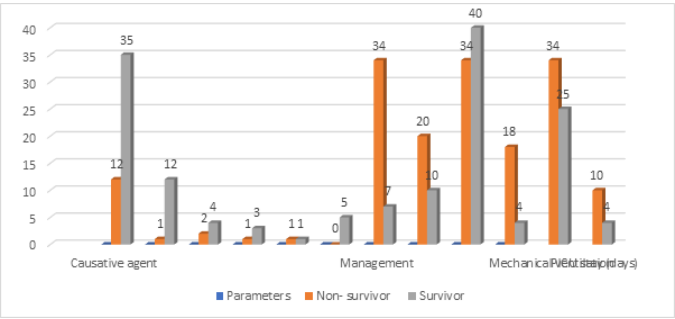

Figure 1: ?

DISCUSSION

The present study was aimed at assessing outcome of encephalitis in pediatric intensive care unit. We enrolled 84 children age ranged 6-14 years. Age group 6-8 years had 36, 9-11 years had 25 and 12-14 years had 23 children. Acute infectious encephalitis/encephalopathy syndrome (AIES) is characterized by fever with impaired consciousness ranging from confusion to coma.[11] This may be associated with seizures other than common febrile seizures. AIES may be due to viral, bacterial, fungal, rickettsial, spirochetal or parasitic infections. Autoimmune encephalitis may also simulate AIES but have different clinical presentation, different etiology and responds to immunotherapy.[12] The spectrum of AIES varies in different geographical regions because of differences in circulating organisms.[13] During the post-monsoon period, a number of arboviruses result in outbreaks of encephalitis in tropical countries. In South-East Asia, Japanese encephalitis (JE) is diagnosed in about 67 900 cases annually, and 50% of them die.[14]

Our study showed that There were 34 non- survivors and 50 survivors. Causative agent found to be Enterovirus in 12 and 35, Herpesvirus in 1 and 12, CMV in 2 and 4, Cosackie B1 in 1 and 3, Influenza & parainfluenza in 1 and 1 and Adenovirus in 0 and 5 in non- survivors and survivors respectively. Hon et al15 reviewed chart of all patients with encephalitis admitted to the PICU. Encephalitis (n = 46) accounted for 2.7 % of PICU admissions, but 11.8 % PICU mortality over a 12-y period. A microorganism (primarily virus) was identified in 59 % of encephalitis patients in the PICU. Enteroviruses and herpes viruses were isolated from the cerebrospinal fluid (CSF). Respiratory viruses [such as respiratory syncytial virus (RSV) and influenza viruses] and enteric viruses (such as rotavirus and norovirus) were obtained in the nasopharyngeal aspirate and stool respectively, but undetectable from the CSF. More than one-fourth patients with encephalitis died in the PICU. Boys accounted for 85 % of non-survivors and 52 % survivors (p = 0.038). Mechanical ventilation, inotrope, intravenous immunoglobulin (IVIG) and corticosteroid usage were significantly higher among non-survivors. Binomial logistic regression showed that patients who received corticosteroid had a lower chance of survival than those who did not after adjusting for gender, IVIG and mechanical ventilation. Eighteen (55 %) of the survivors had moderate-to severe neurodevelopmental impairment.

We found that management done was inotropes in 34 and 7, IVIG in 20 and 10, anticonvulsant in 34 and 40, corticosteroid in 18 and 4. Mechanical ventilation was required in 34 and 25, PICU stay was 10 days and 4 days in non- survivors and survivors respectively. Thakur et al,[16] investigated predictors of outcome in patients with all-cause encephalitis receiving care in the intensive care unit. They examined mortality and predictors of good outcome and poor outcome in those surviving to hospital discharge. In our cohort of 103 patients, the median age was 52 years (interquartile range 26), 52 patients (50.49%) were male, 28 patients (27.18%) had viral encephalitis, 19 (18.45%) developed status epilepticus (SE), 15 (14.56%) had cerebral edema, and 19 (18.45%) died. In our multivariate logistic regression analysis, death was associated with cerebral edema, SE and thrombocytopenia. Endotracheal intubation requirement with ventilator support was highly correlated with death (95%). In addition, in those patients who survived, viral, nonviral, and unknown causes of encephalitis were less likely to have a poor outcome at hospital discharge compared with an autoimmune etiology such as viral encephalitis, nonviral encephalitis, unknown etiology.

In a previous study evaluating SE in patients with encephalitis and response to antiepileptic drugs and mortality, 36.7% remained refractory to the second antiepileptic drug and approximately one-third of patients died.24 Studies have shown that encephalitis is a common cause of refractory SE.[17]

CONCLUSION

A significant mortality was seen in pediatric encephalitis. Patients need extensive management to prevent deaths.

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