

Research Article



INTERNATIONAL RESEARCH JOURNAL OF PHARMACY

www.irjponline.com

ISSN 2230-8407 [LINKING]

CLINICO-LABORATORY PROFILE AND ACUTE ENCEPHALOPATHY SYNDROME IN CHILDREN

Dr. Sukanta Nandi,¹ Dr. Subhashish Paul^{2*}

¹Assistant Professor, Department of Paediatrics, ICARE Institute of Medical Science and Research & Dr. Bidhan Chandra Roy Hospital, Haldia, West Bengal

^{2*}Assistant Professor, Department of Paediatrics, Gouri Devi Institute of Medical Sciences & Hospital, Durgapur, West Bengal

Corresponding Address

Email id: dr.subhasish2017@gmail.com

How to cite: Nandi S, Paul S. Clinico-laboratory profile and acute encephalopathy syndrome in children. International Research Journal of Pharmacy, 2023,14:2:45-49.

Doi: 10.7897/2230-8407.1303212

ABSTRACT

Background: Children with acute encephalopathy syndrome (AES) typically require hospitalization to special care units and intensive care units (ICUs). Data from the literature is limited, there is no specific treatment for AES, and the main line of management is supportive.

Aim: The purpose of this study was to evaluate the relationship between the outcomes of acute encephalopathy syndrome in children with AES and their clinical and analytical characteristics.

Methods: 98 participants between the ages of one month and twelve years who were admitted to the Institute during the specified study period and had a confirmed diagnosis of acute encephalopathy syndrome were evaluated in this study. Clinical and laboratory characteristics were evaluated and linked with AES in each of the enrolled individuals. The collected data were statistically examined.

Results: According to the study's findings, the most frequent clinical manifestation of AES in all participants was fever and altered sensorium, which was observed in 100% of them. Seizures, headaches, vomiting, and diarrhea were present in 63%, 34.7%, 26.5%, and 22.4% of the participants, respectively. In 28.6% of research participants, Japanese encephalitis (JE) was the most common cause of AES. Herpes simplex virus, pyogenic meningitis, and tubercular meningitis were the next most common causes, accounting for 4.1%, 4.1%, and 2% of study participants, respectively. In 61.2% of individuals, the etiology was unknown. Death and recovery with neurological sequelae were observed in 28.6% (n = 28), 16.8% (n = 16), and 55.1% (n = 54) of the 98 cases, respectively.

Conclusions: According to the current study, AES typically manifests clinically as fever, altered sensorium, headache, vomiting, seizures, and meningeal irritation symptoms. One of the main causes of AES in Indian children is Japanese encephalitis. The GCS results at the time of admission and during the hospital stay are significantly correlated. The necessity for international attention to help preserve the lives of children with AES is illustrated by neurological consequences.

Keywords: Acute encephalitis syndrome, AES, AES outcomes, Glasgow coma scale, meningitis,

INTRODUCTION

Acute encephalitis syndrome, or AES, is a prevalent clinical illness that typically affects children and is typically brought to the pediatric practice by children. Because of its high fatality rates, which are typically characterized by abrupt presentation and fast cerebral deterioration, AES presents serious complications for doctors. AES is characterized by an acute onset of fever, a change in mental status, and/or the development of new seizures in a person of any age, at any time of year.

The cause of acute encephalitis syndrome varies greatly depending on the season and geographic area. Viruses are the primary causative agent of acute encephalitis syndrome, especially in those living in India. However, there have also been reports linking other poisons and bacteria to the etiology of acute encephalitis syndrome.³

In addition to requiring specialized care and treatment, child subjects with acute encephalitis syndrome typically need to be admitted to an intensive care unit (ICU). Acute encephalitis syndrome does not, however, have a specific treatment; instead, supportive care is the mainstay of treatment.⁴ The purpose of the current study was to evaluate the relationship between the clinical and laboratory characteristics of child patients with AES and the outcomes of acute encephalopathy syndrome.

MATERIALS AND METHODS

The goal of the current prospective clinical investigation was to evaluate the relationship between the clinical and laboratory characteristics of child participants with AES and the outcomes of acute encephalopathy syndrome. The Institute's Department of Pediatrics provided the study participants. Prior to their involvement in the study, all subjects gave their written and verbal informed consent.

During the designated study period, 98 child participants of both sexes who had been hospitalized to pediatric wards with a history of AES were evaluated. The study's inclusion criteria included children ages 1 month to 12 years, parents who provided informed consent for study participation, and individuals having an AES diagnosis according to the WHO case definition. Participants having a history of mild febrile seizures, those with neurological impairments prior to the beginning of AES, and neonates aged 0–28 days were excluded from the study.

Every research participant was evaluated methodically using pre-made, standardized proforma. Involuntary movements, seizure type, pupillary reaction to light, motor pattern, posture, temperature, blood pressure, breathing patterns and rate, and/or heart rate were among the clinical characteristics evaluated in each study participant.

Following a thorough clinical examination, 2 milliliters of blood and 1 milliliter of CSF (cerebrospinal fluid) were taken from each subject under strict aseptic and sterile conditions. These samples were then sent to the clinical laboratory for the necessary laboratory tests, such as the immunoglobulin M enzyme-linked immunosorbent assay for Japanese encephalitis (JE) and CBNAAT for tubercular meningitis (Cartridge-based). Based on clinical presentation, peripheral blood smears and rapid diagnostic tests for malaria parasites were performed.

Japanese encephalitis, cerebral malaria, tubercular meningitis, bacterial meningitis, and AES of uncertain origin were the classifications given to the causes of acute encephalitis syndrome. Recovery without neurological sequelae at discharge, death, or recovery with neurological sequelae at discharge were the three outcomes evaluated.

SPSS (Statistical Package for the Social Sciences) software version 24.0 (IBM Corp., Armonk, NY, USA) was used to statistically analyze the collected data. This included chi-square testing, multiple logistic regression, Kaplan-Meier survival analysis, and descriptive measure evaluation. The findings were presented as frequency, percentages, mean, and standard deviation. Statistical significance was defined as a p-value of less than 0.05.

RESULTS

The goal of the current prospective clinical investigation was to evaluate the relationship between the clinical and laboratory characteristics of child participants with AES and the outcomes of acute encephalopathy syndrome. The current study evaluated 98 participants, ages 1 month to 12 years, who were admitted to the Institute during the specified study period and had a confirmed diagnosis of acute encephalopathy syndrome.

Among the study participants, 61.2% (n=60), 2.04% (n=2), 4.1% (n=4), 4.1% (n=4), and 28.6% (n=28) had tubercular meningitis, pyogenic meningitis, HSV, and JE, respectively, with the disease etiology being unclear. The following clinical characteristics were present in 22.4% (n=22), 26.5% (n=26), 34.7% (n=34), 63.3% (n=62), 100% (n=98), and (n=98) of the participants, respectively: diarrhea, vomiting, headache, seizures, and fever. In 25% (n=4), 37.5% (n=6), 12.5% (n=2), and 25% (n=4) of the individuals, respectively, behavioral abnormalities, cranial nerve palsy, motor impairment, and aphasia/incomprehensible sound were the neurological sequelae. In 55.1% (n=54), 16.3% (n=16), and 28.6% (n=28) of the individuals, respectively, the study outcomes were recovered without neurological sequelae, recovered with neurological sequelae, and death (Table 1).

GCS 3-8 was shown to be correlated with AES outcomes in study participants in 8.3% (n=2) of individuals who recovered without neurological sequelae, 9-12 in 25% (n=6) of subjects who recovered with neurological sequelae, and 66.7% (n=16) of subjects who died. 42.9% (n=12) of the individuals who died had a GCS of 13–15, whereas 95.7% (n=44) of the subjects

recovered without neurological sequelae, 4.3% (2) recovered with neurological sequelae, and 0 subjects died. GCS was 9–12 in 28.6% (n=8) of the subjects that recovered without and with neurological sequelae. With $p=0.001$, the difference was extremely statistically significant. In 50% (n=24) of the participants who recovered without neurological sequelae, 0% of the subjects who recovered with neurological sequelae, and 50% of the subjects who died, the length of hospital stay ranged from 0 to 7 days, 8-15 days in 50% (n=26), 37.5% (n=6), and 14.3% (n=4) subjects recovered without neurological sequelae, recovered with neurological sequelae, and dead subjects respectively which was statistically significant with $p=0.001$ (Table 2).

According to the study's findings, 53.8% (n=14) of the subjects who recovered without neurological sequelae, 0 subjects who recovered with neurological sequelae, and 46.2% (n=12) of the subjects who died had serum sodium levels of less than 135 meq/L, while 33.3% (n=2), 0 subjects, and 67.7% (n=4) of the subjects who recovered without and with neurological sequelae, and the subjects who died, had serum sodium levels between 135 and 145 meq/L, respectively. These differences were not statistically significant, with $p=0.08$. Subjects who recovered without neurological sequelae, those who had neurological sequelae, and those who died showed non-significant differences in CSF protein levels of ≤ 40 mg/dl and >40 mg/dl ($p=0.276$). TCL cells <4000 , 4000-11000, and >11000 did not differ significantly between those who died with $p=0.766$ and those who recovered without neurological sequelae (Table 2).

In terms of CSF cells, patients who recovered without neurological sequelae, those who had neurological sequelae, and those who died showed statistically non-significant differences in cells as 0–5 and >5 cells/mm³ ($p=0.575$). Similarly, serum potassium levels of 2.5-5.5 mmol/l and >5.5 mmol/l were statistically non-significant in individuals with neurological sequelae, recovering subjects without neurological sequelae, and deceased subjects ($p=0.605$) (Table 2).

DISCUSSION

In this study, 98 participants between the ages of one month and twelve years who were admitted to the Institute during the designated study period and had a confirmed diagnosis of acute encephalopathy syndrome were evaluated. HSV, JE, tubercular meningitis, pyogenic meningitis, and unknown disease etiology were present in 61.2% (n=60), 2.04% (n=2), 4.1% (n=4), and 28.6% (n=28) of the study participants, respectively.

The following clinical characteristics were present in 22.4% (n=22), 26.5% (n=26), 34.7% (n=34), 63.3% (n=62), 100% (n=98), and (n=98) of the participants, respectively: diarrhoea, vomiting, headache, seizures, and fever. In 25% (n=4), 37.5% (n=6), 12.5% (n=2), and 25% (n=4) of the individuals, respectively, behavioural abnormalities, cranial nerve palsy, motor impairment, and aphasia/incomprehensible sound were the neurological sequelae. The results of the study showed that 55.1% (n=54) of the individuals died, 16.3% (n=16) recovered with neurological sequelae, and 28.6% (n=28) recovered with neurological sequelae. These findings were similar to those of earlier research by Khound M et al. (2017) and De S et al. (2015), in which the authors evaluated the individuals' AES and demographics similar to those of the current study.

GCS (Glasgow coma scale) scores were 3-8 in 8.3% (n=2) of subjects who recovered without neurological sequelae, 9-12 in 25% (n=6) of subjects who recovered with neurological sequelae, and 66.7% (n=16) of subjects who died, according to the study's findings on the correlation of different variables affecting AES outcomes in study participants. 42.9% (n=12) of the individuals who died had a GCS of 13–15, whereas 95.7% (n=44) of the subjects recovered without neurological sequelae, 4.3% (2) recovered with neurological sequelae, and 0 subjects died. GCS was 9–12 in 28.6% (n=8) of the subjects that recovered without and with neurological sequelae. With $p=0.001$, the difference was extremely statistically significant.

50% (n=24) of the participants who recovered without neurological sequelae spent 0–7 days in the hospital, while 0 subjects who recovered with neurological sequelae did not, and 50% (n=24) subjects that died, was 8-15 days in 50% (n=26), 37.5% (n=6), and 14.3% (n=4) 50% (n=24) of the subjects died within 8–15 days, while 50% (n=26), 37.5% (n=6), and 14.3% (n=4) of the participants recovered without and with neurological sequelae, respectively. This was statistically significant ($p=0.001$). These findings were in line with those of Kakoti G. et al.⁷ and Barman M. et al.⁸, who found that factors influencing AES outcomes, such as GCS and length of hospital stay, had significant results comparable to the current study in their separate investigations.

57.6% (n=38), 24.2% (n=16), and 18.2% (n=12) of the subjects who recovered without neurological sequelae, recovered with neurological sequelae, and died, respectively, had serum sodium levels of <135 meq/L, whereas 53.8% (n=14) of the subjects who recovered without neurological sequelae, 0 subjects who recovered with neurological sequelae, and 46.2% (n=12) of the subjects who died had serum sodium levels of <135 meq/L and was >145 meq/L in 33.3% (n=2), 0, and 67.7% (n=4) subjects that recovered without neurological sequelae, recovered with neurological sequelae, and died subjects which was non-

significant with $p=0.08$. Subjects who recovered without neurological sequelae, those who had neurological sequelae, and those who died showed non-significant differences in CSF protein levels of ≤ 40 mg/dl and >40 mg/dl ($p=0.276$). Subjects who recovered without neurological sequelae, those who had neurological sequelae, and those who died showed non-significant differences with TLC cells <4000 , $4000-11000$, and >11000 ($p=0.766$). These results were consistent with those of Prakash Sahay B et al. (2020) and Sambasivam E et al. (2017), who found that TLC counts, salt levels, and CSF protein value were all non-significant, just like the current study.

Regarding CSF cells, it was observed that there were statistically non-significant differences between those who recovered without neurological sequelae, those who had neurological sequelae, and those who died ($p=0.575$) for cells as $0-5$ and >5 cells/mm³.

Similar results were seen for serum potassium levels where serum potassium levels of $2.5-5.5$ mmol/l and >5.5 mmol/l were statistically non-significant in subjects that recovered without neurological sequelae, with neurological sequelae, and dead subjects with $p=0.605$. Serum potassium levels showed similar results, with levels of $2.5-5.5$ mmol/l and >5.5 mmol/l being statistically non-significant in individuals who recovered without neurological sequelae, those who had neurological sequelae, and those who died ($p=0.605$). The results were consistent with the findings of Baranwal SK et al. (2011) and Hoshino A et al. (2012), who reported comparable results to the current study regarding CSF cells and serum potassium levels.

CONCLUSIONS

Clinical manifestations of AES typically include fever, altered sensorium, headache, vomiting, seizures, and meningeal irritation symptoms. One of the main causes of AES in Indian children is Japanese encephalitis. Results are significantly correlated with GCS both at the time of admission and throughout the hospital stay. Neurological sequelae depict the need for global attention to help save the lives of children with AES.

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Characteristics	Subgroup	Number (n=98)	Percentage (%)
Etiology	Unknown	60	61.2
	Tubercular meningitis	2	2.04
	Pyogenic meningitis	4	4.1
	HSV	4	4.1
	JE positive	28	28.6
Clinical features	Diarrhea	22	22.4
	Vomiting	26	26.5
	Headache	34	34.7
	Seizures	62	63.3
	Altered sensorium	98	100
	Fever	98	100
Neurological sequelae	Behavioral disorders	4	25
	Cranial nerve palsy	2	12.5
	Motor deficit	6	37.5
	Aphasia/incomprehensible sound	4	25
Outcome	Recovered without neurological sequelae	54	55.1
	Recovered with neurological sequelae	16	16.3
	Death	28	28.6

Table 1: Clinical profile of study subjects with AES

Variables		Recovered without neurological sequelae		Recovered with neurological sequelae		Death		p-value
	Subgroup	n	%	n	%	n	%	
GCS44	3-8	2	8.3	6	25	16	66.7	0.001
	9-12	8	28.6	8	28.6	12	42.9	
	13-15	44	95.7	2	4.3	0	0	
Hospital stays duration (days)	0-7	24	50	0	0	24	50	0.001
	8-15	26	50	6	37.5	4	14.3	
	>15	2	16	10	83.3	0	0	
Serum sodium (meq/l)	<135	14	53.8	0	0	12	46.2	0.08
	135-145	38	57.6	16	24.2	12	18.2	
	>145	2	33.3	0	0	4	67.7	
CSF protein	≤40 mg/dl	2	33.3	0	0	4	66.7	0.276
	>40 mg/dl	52	56.5	16	17.4	24	26.1	
TLC (cells/mm3)	<4000	0	0	0	0	0	0	0.766
	4000-11000	26	60	6	15	10	25	
	>11000	28	50	10	17.9	18	32.1	
CSF (cells/mm3)	0-5	32	61.5	8	15.4	12	23	0.575
	>5	22	47.8	8	17.4	16	34.8	
Serum potassium	2.5-5.5 mmol/l	44	52.4	14	16.7	26	31	0.605
	>5.5 mmol/l	10	71.4	2	14.3	2	14.3	

Table 2: Correlation of various variables affecting AES outcomes in study subjects