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STUDY OF NON TRAUMATIC COMA IN CHILDREN AT TERTIARY HOSPITAL.

Pediatrics

Dr Sneha Patil* Junior Resident, Department of Pediatrics, Bharati Vidyapeeth (Deemed To Be University) Medical College & Hospital, Sangli, Maharashtra. *Corresponding Author

Dr. S. G. Kumbhar Professor and Head of Unit, Department of Pediatrics, Bharati Vidyapeeth (Deemed To Be University) Medical College & Hospital, Sangli, Maharashtra.

Dr S. Mirajkar Assistant Professor,Department of Pediatrics, Bharati Vidyapeeth (Deemed To Be University) Medical College & Hospital, Sangli, Maharashtra.

ABSTRACT

Introduction: Non-traumatic coma can have varied etiology and clinical characteristics. These may determine the management and outcome of the patients. We aimed to study the etiology and outcomes of children diagnosed and treated for non-traumatic coma in our hospital.

Methodology: Medical records of children aged 2 months to 18 years, diagnosed with non-traumatic coma from January 2020 till December 2020 were reviewed retrospectively. The final outcome was determined by patient's death or neurological condition at the time of discharge.

Results: In the present study, out of 45 patients of NTC, 44% of the patients were in the age group 2 months to 5 years, 31% in 6 years to 12 years and 24% in 13 years to 18 years and 42% were Males & 57% were Females. In etiology, Infectious and Non-infectious causes contributed equally. Among the infectious causes, the most common ones were acute encephalitis (22%), acute pyogenic meningitis (9%), TBM (9%) and remaining infectious causes contributed to 9%. Among the non-infectious causes, diabetic ketoacidotic coma (22%), epileptic encephalopathy and metabolic encephalopathy contributed to 7% each. In outcome of the sample of 45 patients, 39 (87%) were discharged home and mortality was observed in 6 (13%). Among the total patients discharged (87%), 53% were neurologically normal, 7% had mild disability, 9% had moderate disability and 18% had severe disability. In our study, mortality was 3% in children with moderate brain injury, 33% mortality in children with severe brain injury (p value < 0.01).

Conclusions: In our study, mortality rate was high with severe brain injury as compared to mild and moderate brain injury as assessed by GCS at admission.

KEYWORDS

Non traumatic coma; Meningitis; Encephalitis; Glasgow Coma Scale.

INTRODUCTION

Non-traumatic coma is the diagnosis of approximately 10 to 15% of pediatric intensive care unit admissions.[1] The clinical significance of these admission is due to the fact that these admissions have high mortality.[2] Those who survive may have unfavourable short term outcomes in terms of neurological disabilities. Much of these clinical outcomes depend on the etiological and initial presentation of the patients. Despite much research, the prediction of clinical outcomes in these patients is difficult.[3] The underlying etiology of patients with non-traumatic coma could be neuro-infections, metabolic conditions, epilepsy, and toxic ingestions.[4] Not only can the underlying etiology inform the treating pediatrician about the prognosis, but the can also help in planning the management. Thus to improve the clinical outcomes in these patients, it is important to evaluate the distribution of patients for their etiological factors and their clinical characteristics.[5] In the present study, we aimed to study the etiology and outcomes of children diagnosed and treated for non-traumatic coma in our hospital.

METHODOLOGY

Study Design and Sample population

We conducted a retrospective review of medical records of children aged 2 months to 18 years who were admitted in the Pediatric Intensive Care Unit (PICU) with non-traumatic coma with Glasgow Coma Scale (GCS) score of less than 12. Coma is defined here as a state of unresponsiveness without evidence of self or environment, a state from which the patient cannot be aroused by vocal or sensory stimuli. We excluded children who had a suspected or confirmed history of trauma, pre-existing neurological illness, left against medical advice while receiving treatment and those with incomplete medical records. Records were searched for admissions of all children from January 2020 till December 2020, of which 45 patients fulfilled the study criteria. Prior permission from Medical Superintendent was taken to review the medical records. **The study was approved by the Institutional Ethics Committee.**

Data Collection and Data Analysis

We used a pre-designed semi-structured questionnaire to collect patient related information. The principle investigator extracted patient information from the medical records. The demographic information of the patients, their presenting complaints, findings of general physical and central nervous system (CNS) examination were noted. The severity of brain injury was assessed using pediatric modified GCS.[6] We also noted the findings of various laboratory investigations like complete blood count, arterial blood gas (ABG) analysis, blood sugar, serum electrolytes, blood culture and cerebrospinal fluid (CSF) examination. Electroencephalogram (EEG) was ordered for 13 patients, while all patients underwent neuroimaging with Magnetic Resonance Imaging (MRI). Underlying etiology for all patients was noted from the medical records as well. Outcome was determined by patient's death or neurological condition at the time of discharge. The clinical neurological status was assessed based on cranial and peripheral motor and sensory neurological examination, including cerebellar function. Immediate neurological outcome was divided into normal (normal or no change from premorbid neurological examination), death and disability.[7] We further classified disability into: mild disability: mild (grade 4) weakness or ataxia, isolated cranial nerve palsy, mild alteration of tone, power or deep tendon reflexes, moderate disability: moderate weakness (grade 3) or ataxia, multiple cranial nerve involvement and severe disability: sever weakness (<grade 3) or ataxia, tetraplegia, vegetative state.

The data were compiled and entered in SPSS version 23 for statistical analysis. Categorical data were described as frequency distribution. To assess the association of pediatric GCS on admission with clinical outcomes at discharge, patients were classified in to mild, moderate and severe brain injury based on GCS score. Then analysis was performed using chi-square/Fisher's exact test, considering p value < 0.05 as statistically significant.

RESULTS

In the present study, out of 45 patients of NTC, 44% of the patients were in the age group 2 months to 5 years, 31% in 6 years to 12 years and 24% in 13 years to 18 years age group and 42% were Males & 57% were Females. The most common presenting complaint other than altered sensorium (100%) was fever (67%) and vomiting (58%) (Table 1). After complete work up of the patients, we found that 48% of the patients had an infectious cause of non-traumatic coma, while the rest 52% had non-infectious causes. Among the infectious causes, the most common ones were acute encephalitis (22%) and acute pyogenic meningitis (9%), TBM (9%). The less common infectious causes were VP Shunt Infection (4%) and severe septic shock (4%). Among the non-infectious causes, diabetic ketoacidotic coma (22%), epileptic encephalopathy (7%) and metabolic encephalopathy (7%) were the most common causes. Less common non-infectious causes were

hepatic encephalopathy (4%), ischemic stroke (4%), dyselectrolytemia (hypernetremia) (2%), near drowning-fresh water (2%) and turpentine poisoning (2%). On general physical examination, relative bradycardia was observed in 20%, while tachycardia was present in 13%. Blood pressure above 95th centile was observed in 9%, white it was lower than 3rd centile in 25%. On pediatric modified GCS, 67% had moderate brain injury (score 9 to 13), while rest of the patients had severe brain injury (score 3 to 8). Laboratory investigations revealed total cell count more than 10,000 per cumm in 64% of the patients, while platelet count more than 4,50,000 per ml were found in 22% (Table 2). Metabolic acidosis was found in 22% and hyperglycemia was observed in 31% of the patients. Blood culture was found to be positive in 9% of the patients, while biochemical abnormalities of the CSF were found in 33%. EEG could be performed in 13 (29%) of the patients, in which generalized slowing was the most common finding (n=7). On MRI brain, diffuse cerebral edema (22%) and meningeal enhancement (18%) were the most common findings. Of the sample of 45 patients, 39 (87%) were discharged home and 6 (13%) died. At the time of discharge, 53% were neurological normal, 7% had mild disability, 9% had moderate disability and 18% had severe disability. Table 3 describes the distribution of patients according to pediatric GCS score and final outcomes of the patients. Among those with moderate brain injury (GCS 9 to 13), 70% had full recovery and 3% died, while those with severe brain injury (GCS 3 to 8), 20% had full recovery and 33% died (p value < 0.01).

Table 1. Baseline characteristics of the patients included in the study

Variables	N	%
Age group (years		
2 months to 5 years	20	44%
6 to 12 years	14	31%
13 to 19 years	11	24%
Gender		
Male	19	42%
Female	26	58%
Presenting complaints		
Altered sensorium	45	100%
Fever	30	67%
Vomiting	26	58%
Headache	15	33%
Seizures	13	29%
Etiology		
Infectious causes		
Acute encephalitis	10	22%
Tuberculous Meningitis	4	9%
Acute Pyogenic Meningitis	4	9%
VP Shunt Infection	2	4%
Septic Shock	2	4%
Non-infectious		
Diabetic ketoacidotic coma	10	22%
Epileptic Encephalopathy	3	7%
Metabolic Encephalopathy	3	7%
Hepatic Encephalopathy	2	4%
Ishemic Stroke	2	4%
Dyselectrolytemia (Hypernetremia)	1	2%
Near Drowning-fresh water	1	2%
Turpentine Poisoning	1	2%

Table 2. Findings of various investigations in the patients

Laboratory investigations	N	%
Leucocytosis (>10,000/cumm)	29	64%
Leucopenia (<5000/cumm)	5	11%
Thrombocytopenia (<1,50,000/ml)	8	18%
Thrombocytosis (>4,50,000/ml)	10	22%
Rapid Dengue Test Positive	1	2%
Weil Felix Test Positive	1	2%
Serum ammonia (>90µg/dl)	2	4%
Serum acetone (Positive)	10	22%
Metabolic Acidosis (Blood pH<7.30)	10	22%
Hypergycemia (>180mg/dl)	14	31%
Hypoglycemia (<60mg/dl)	1	2%
Positive Blood Culture	4	9%
Abnormal CSF	15	33%
Dyselectrolytemia	4	9%
Deranged Coagulation profile	2	4%
EEG (n=13)		
Generalized slowing	7	54%
Bicentrotemporal epileptiform discharges	3	23%
Occasional generalized epileptiform discharges	2	15%
Right Hemispheric epileptiform discharges with left hemispheric slowing	1	8%
MRI brain		
Diffuse Cerebral edema	10	22%
Meningeal Enhancement	8	18%
Cerebral Atrophy	4	9%
Cerebral Infarction	2	4%
Hydrocephalous	2	4%
Intracranial Heamorrhage	1	2%
Others	2	4%

Table 3. Association of pediatric modified Glasgow Coma Scale (GCS) with the final outcome of the patients

		Final outcome					
Pedaitric modified GCS		**Full Recovery**	**Mild Disability**	**Moderate Disability**	**Severe Disability**	**Deaths**	
Mild GCS (14-15)	N	0	0	0	0	0	0
	%	0%	0%	0%	0%	0%	0%
Moderate GCS (9-13)	N	21	2	2	4	1	30
	%	70%	7%	7%	13%	3%	100%
Severe GCS (3-8)	N	3	1	2	4	5	15
	%	20%	7%	13%	27%	33%	100%
		24	3	4	8	6	
		p value < 0.01					

DISCUSSION

The present study was done to assess the etiology, clinical characteristics and final outcomes of children who were diagnosed with non-traumatic coma. We observed that 44% of the children were aged less than 5 years and 58% were females. In a recent study by Kermani and colleagues, 101 children aged less than 15 years were evaluated for non-traumatic coma.[8] The authors reported that 53.5% were females and mean age was 3.4 ± 3.3 years. In another similar study from Turkey, Muhterem et al determined the etiology and clinical characteristics of patients and the factors associated with mortality and degree of disability in pediatric patients with non-traumatic coma.[9] Of the 159 patients included in the study, 51% were males and the median age was 55 months. In another study, Suganthi et al studied the etiology and clinical profile of non-traumatic coma in children admitted to a tertiary care hospital in South India.[10] In their study, 40% of the children were aged less than 5 years and 60% were females. Ahmad et al assessed the etiology and clinical characteristics of children aged 6 months to 12 years treated for non-traumatic coma at a tertiary care centre in Srinagar.[11] In their study, 44.5% were females and 54% were aged less than 3 years.

We observed 48% of the patients to have an infectious cause of non-traumatic coma. In the present study, the most common infectious cause of non-traumatic coma were acute encephalitis (22%), acute pyogenic meningitis (9%) and Tuberculos Meningitis (9%) while diabetic ketoacidotic coma (22%), epileptic encephalopathy (7%) and metabolic encephalopathy (7%) were the most common non-infectious causes. It should be noted that the underlying etiology can vary according to the geographical location of the patient. For instance, infectious causes of non-traumatic coma are more common in developing regions. South east Asia has higher incidence of cerebral malaria[12] and in Japan influence and rota virus induced acute encephalitis were the common causes.[13] On the other hand, hypoxic ischemic encephalopathy and toxic metabolic causes are more common in developed countries.[14] Kermani et al found infectious causes in 42.6%, intoxication in 19.8%, epilepsy in 16.8% and metabolic causes in 14.9% of the patients. Among infectious causes, viral encephalitis (45.2%), sepsis (40.4%) and bacterial meningitis (11.9%) were the most common ones. In the study by Suganthi et al, infectious causes were responsible for 58% cases. Among the infectious causes, acute bacterial meningitis, tubercular meningitis ad viral meningitis were the most common causes. Among non-infectious causes, status epilepticus, febrile fever and diabetic ketoacidosis were the most common causes. Ahmad et al reported CNS infections to be the predominant cause of non-traumatic coma. Encephalitis (16.4%),

pyogenic meningitis (11.5%) and tubercular meningitis (6.6%) were the common causes. Toxic-metabolic causes were the underlying etiology in 26.2% of the patients, among which poisoning (11.5%) and hepatic encephalopathy (6.6%) were the common causes. Structural CNS lesions were found in 8.2% of the patients.Mehterem and colleagues found neuroinfectious to be the most common cause (31.4%). Among infections, viral encephalitis and acute bacterial meningitis were the most common ones. Toxic-metabolic causes were observed in 25.8% of the patients, among which neurometabolic disease (8.8%) and drug poisoning (5.7%) were the most common. Other causes included epilepsy (15.1%), stroke (6.3%), hypoxic ischemic encephalopathy (1.9%) and hypertensive encephalopathy (1.3%).

We found generalized slowing on EEG in 54% of the patients. On MRI brain, diffuse cerebral edema (22%) and meningeal enhancement (18%) were the most common findings. In the study by Kermani et al, EEG abnormalities were observed in 69%, while brain CT was abnormal in 58.1%.

In our study sample, 87% were discharged home and 13% died. At the time of discharge, 53% were neurological normal, 7% had mild disability, 9% had moderate disability and 18% had severe disability. Among those with moderate brain injury (GCS 9 to 13), 70% had full recovery and 3% died, while those with severe brain injury (GCS 3 to 8), 20% had full recovery and 33% died (p value < 0.01). Studies in the past have reported mortality rates as low as 12% to as high as 36%.[15,16] The overall mortality in the study by Suganthi et al was 16%. Similar to our findings, GCS score at admission was found to be significantly associated with mortality. In their study, 6 out of 7 children with GCS of 3 died (p value < 0.01). In the study by Ahmad et al reported a mortality of 34%. Of the 39 who survived 19 (48.7%) were normal at discharge and 20 (51.3%) had varying degrees of disability. The authors found that GCS was significantly associated with the mortality in the patients (p value < 0.01). In another study by Kermani et al, 76% recovered completely, 5% were discharged with neurological disability and 19% expired. Among those with neurological disability, the severity was mild in 40%, moderate in 40%, and severe in 20%. Muhterem et al reported 8.2% mortality. Among the 146 survivors, 61.6% were discharged from PICU without any neurological deficit, 9.6% had mild disability, 22.6% had moderate disability, 2.8% had severe disability, and 3.4% were in a vegetative state. Similar to our findings, GCS score at admission was found to be significantly associated with the mortality (p value < 0.001).

There are a few limitations of the study. The retrospective nature of study did not allow us to assess other variables which could predict the outcome of the patients. Also long term follow up of the patients could not be conducted. Second, neurological examination of young children is difficult and assessments can be subjective. Third, our modest sample size would not allow us to generalize the results of the present study to other geographical locations.

CONCLUSION

In the present study, acute encephalitis and diabetic ketoacidosis were the most common causes of non-traumatic coma. The mortality rate in the present study was 13% and it was significantly associated with severe brain injury as assessed by GCS at admission. The study thus reaffirms that GCS at admission is an important assessment for children with non-traumatic coma.

Abbreviations:

NTC: Non Traumatic Coma
TBM: Tubercular meningitis
MRI: Magnetic Resonance Imaging
EEG: Electroencephalogram
CNS: Central Nervous System
ABG: Arterial Blood Gases
CSF: Cerebrospinal fluid
PICU: Pediatric Intensive Care Unit
GCS: Glasgow Coma Scale
VP: Ventriculoperitoneal

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