NON–TRAUMATIC COMA IN CHILDREN: CLINICAL PROFILE AND OUTCOME

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ABSTRACT

OBJECTIVE

To study the aetiology and clinical profile of non-traumatic coma in children and to determine the clinical signs predictive of outcome.

METHODS

50 cases of non-traumatic coma between 2 months and 12 years were studied. Data collected include temperature, pulse rate, blood pressure, respiratory rate, Glasgow coma scale, papillary reflex, tone and fundus. Data was analysed by Chi-square test and Fisher exact test for differences in proportion of categorical variables between 2 or more groups.

RESULTS

Aetiology of coma in 58% of cases was infectious, other causes were status epilepticus (20%), metabolic (10%), intoxication (4%), intracranial bleed (2%) and miscellaneous (6%). Among the predictions of mortality – GCS <8, abnormal muscle tone and papillary reflex were found to be statistically significant ($p<0.01$).

CONCLUSION

CNS infections were the most common cause of non-traumatic coma in children. Simple clinical signs were good predictors of outcome.

KEYWORDS

Coma, Children, Non-Traumatic.

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INTRODUCTION

Non-traumatic coma is an important source of morbidity and mortality in the paediatric age group.[1] Accurate diagnosis of aetiology of childhood coma in resource poor countries is complicated by overlap in clinical presentation, limited diagnostic resources, disease endemicity and co-morbidity.[2] Considerable skill is required to distinguish the group at high risk for further deterioration, potentially leading either to death or severe handicap. This study attempts to identify the common aetiological factors of paediatric comatose patients as well as the predictors of poor outcomes in these patients.

METHODS

The present observational, retrospective study was conducted at a tertiary care hospital in South India from January 2014 to January 2015. Children of either sex, aged between 2 months and 12 years, admitted with acute alteration of consciousness for more than six hours were the subjects of study. Significant depression of conscious level was defined as a Glasgow coma scale of 12 and below. For children below 5 years of age, the James modification of the Glasgow coma scale was used. Exclusion criteria were coma due to trauma or as part of an anticipated terminal illness. Ethics approved for this study was obtained from the Institutional Ethics Committee.

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Data collected from records include age, sex, other presenting symptoms, diagnosis, clinical features and outcome. Clinical features noted at the time of admission were temperature, heart rate, respiratory rate, systolic blood pressure, Glasgow coma scale, pupillary reflex, tone and fundus.

Data was analysed by Chi Square test and Fisher's exact test to test for differences in proportions of categorical variables between two or more groups. The tests were performed at 5% significance level.

RESULTS

A total of 50 children had been admitted with non-traumatic coma. Out of these 50 cases, 10% belonged to the age group 2-12 months; 30% 1 to 5 years and 60% 5 to 12 years. There was a variation in age dependent incidence with an increased incidence in 5 to 12 year age group. Proportion of male and females were 40.34% and 59.66%, respectively (Table 1). The data related to presentation from 50 records were analysed. The presenting symptoms were convulsions (35 cases), fever (34), headache (26), vomiting (23), cough (5), fast breathing (4), abdominal pain (4), diarrhea (2), visual symptom-blindness (1 case). Most of the children presented with more than one symptom. Convulsions (35, 90%) and fever (34, 68%) were the most common symptoms.

Aetiology

Infections – were the predominant cause of non-traumatic coma, accounting for 58% of cases (Table 2). Acute bacterial meningitis was the most common cause followed by tuberculous meningitis, viral encephalitis, septicemia and cerebral malaria. Among CNS infections, 77% were in the age group of 5 to 12 years.

There were 8 children with status epilepticus, of which 88% were in the 5 to 12 years age group. Febrile status was seen in 2 cases and both belonged to 1 to 5 years age group; 2 children presented with intoxication – one organophosphorus poisoning and another Neem oil poisoning. Both were 12 months – 59 months age group. Coma of accidental cause was identified in 1 case and it was due to near drowning. The child was 1-½ years old.

Metabolic

Diabetic Ketoacidosis (DKA) was the aetiology in 3 cases. The episode of DKA was the first presentation in 2 children. They belonged to 5 o 12 years age group. There was 1 child, 8 weeks old, diagnosed to have medium chain Acyl dehydrogenase deficiency. The aetiology was hepatic encephalopathy in 1 child, who was 8 years old and presented with low-grade fever and acute abdominal pain.

Other Causes

There was 1 child, 4 years who had Intracranial hemorrhage due to acute Idiopathic Thrombocytopenic Purpura. The aetiology was posterior cerebral artery stroke in a 12 years old child, who presented with blindness 3 days prior to the onset of alteration in consciousness. Acute disseminated encephalomyelitis was identified as the cause in a 9 years old child.

Mortality

Among the 50 children experiencing non-traumatic coma, there were 8 deaths (16%). Regarding the outcome according to aetiology (Table 2), out of 7 children admitted with viral encephalitis, 2 expired. These children had diffuse cerebral edema and intracranial hypertension, 2 children were admitted with sepsis of which a 4-month-old infant had failure to thrive, faulty child rearing practices, bronchopneumonia and succumbed. In 2 children the aetiology was intoxication, 1 was organophosphorus poisoning, the other was a 2-year-old child with neem oil poisoning. The child with neem oil poisoning succumbed, had intractable seizures and pulmonary edema. There was a 1-½-year-old child, admitted with drowning, treated in a private hospital for 9 days and referred here in altered sensorium and respiratory failure. This child expired. A 6-week-old child was diagnosed as Medium chain acyl dehydrogenase deficiency by Tandem mass spectrometry at 4 weeks of age for developmental delay and seizers.

This child was admitted at 8 weeks of age with bronchopneumonia, sepsis and seizures and expired after 10 days of hospital stay. An 8-year-old child was admitted for low-grade fever, abdominal pain 2 days, shortly after admission developed increased severity of abdominal pain, altered sensorium and posturing. His serum bilirubin and liver enzymes were grossly elevated and he succumbed in few hours. A 12-year-old girl was admitted with a history of decreased visual acuity for 3 days and was followed by altered sensorium. She had clinical features of increased intracranial pressure. Her CT angiography showed a posterior cerebral artery stroke with diffuse cerebral edema and she succumbed.

The predictors of mortality were analysed (Table 3). Out of 24 children with GCS <8, 8 had died. Among 11 children with nonreactive pupils, 5 expired and out of 9 children with tone abnormalities, 7 had died. The variables GCS <8, pupillary reflex and tone were found to be statistically significant ($p < 0.001$). Other variables like temperature on admission, heart rate on admission, respiratory rate on admission, systolic blood pressure and papilledema were not statically significant.

Age	Sex		Total
	M	F	
2 – 12 months	2	3	5
13 – 59 months	7	8	15
60 months – 12 years	13	17	30
Total	**22**	**28**	**50**

Table 1: Age and Sex Distribution

Aetiology	Total No. of Cases	No. of Cases Survived	No. of Cases Death
Acute bacterial meningitis	11	11	-
Tuberculous Meningitis	8	8	-
Viral encephalitis	7	5	2
Sepsis	2	1	1
Cerebral malaria	1	1	-
Status epilepticus	8	8	-
Febrile status	2	2	-
Intoxication	2	1	1
Drowning	1	-	1
Diabetic ketoacidosis	3	3	-
MCAD deficiency	1	-	1
Hepatic encephalopathy	1	-	1
Intracranial hemorrhage	1	1	-
PCA stroke	1	-	1
ADEM	1	1	-
Total	**50**	**42**	**8**

Table 2: Aetiology of NTC and Distribution of Outcome as per Aetiology

Variables		No. of Patients	Survived	Expired	Chi-sq value	P Value	Result
Temperature on admission	Afebrile Hypothermic >100° F >104° F	11 1 26 12	8 0 24 10	3 1 2 2	7.629	0.054	NS
Heart Rate on admission	Normal Bradycardia Tachycardia	15 5 30	14 3 25	1 2 5	3.617	0.306	NS
Respiratory rate on admission	Normal Bradypnea Tachypnea	37 4 9	33 2 7	4 2 2	4.441	0.349	NS
Systolic BP	Normal Low High	44 3 3	38 1 3	6 2 -	0.571	0.752	NS
GCS	3 3-8 >8	7 17 26	1 15 26	6 2 -	24.246	0.00	**
Pupillary reflex	Reacting Nonreacting	39 11	36 6	3 5	Fisher exact test	0.00	**
Tone	Normal Hypertonia Hypotonia	41 4 5	39 1 2	2 3 3	21.336	0.0002	**
Fundus	Normal Papilledema	47 3	40 2	7 1	Fisher exact test	0.529	NS

Table 3: Factors Predicting Mortality

** - Significant at 1% level NS – Not significant

DISCUSSION

In our study, out of 50 children maximum cases (60%) were in the age group of 5 to 12 years. A recent study from India.[3] showed the preponderance in 3-6 years age group. The causes of non-traumatic coma vary by country, season and period of data collection. Seshia SS, et al.[4] Bansal A, et al.[5] in their study found CNS infections contributed to 60% of aetiology of non-traumatic coma in 100 children. The present study is consistent with this finding. In 58% of the cases, CNS infections were the cause, most notably Acute Pyogenic Meningitis (n=11), Tuberculous Meningitis (n=8), Viral Encephalitis (n=7) and cerebral malaria (n=1). In a study by Ahmed S, et al.[6] out of 100 children with NTC, 66% had infectious aetiologies. Jindal A, et al.[7] also reported that CNS infections were the most common cause of NTC in children. In a study by Fouad H, et al.[8] in 100 consecutive paediatric NTC cases, the most common aetiology was metabolic (33%) followed by CNS (28%) infections. In our study the mortality was 16%, whereas the mortality was around 25% in studies from other countries.[9,10]

In this study, the mortality was found in viral encephalitis (2 out of 7 cases), one each in sepsis, drowning, poisoning, IEM, hepatic encephalopathy and stroke. In a study by Sinelair JR, et al.[11] the aetiology was also important in determining outcome. Hospital morality in their study was lowest in patients with cerebral malaria, organophosphorus poisoning. Matuja WB, et al.[10] also reported that the cause of coma was an important indicator of prognosis. In their study, good recovery was achieved with cerebral malaria, diabetic ketoacidosis, acute bacterial meningitis and drug overdose.

Among the patient's vital signs recorded at the time of admission, temperature, heart rate, respirators rate, systolic blood pressure did not correlate with poor outcome. Other investigators.[11,12] have found hypotension, abnormal respiratory pattern at admission correlated significantly with mortality. Clinical features which showed increased association with mortality include GCS<8, non-reactive pupils and abnormalities in tone (P <.01). The GCS is a standardized system developed initially in traumatic coma to assess the degree of coma and to identify the seriousness of brain injury in relation to outcome. 100 studies in NTC have indicated that the mortality is significantly high when the GCS is less than 8.[13] P. C. Nayanaprabha, et al.[14] have found that the mortality was higher when the ocular and verbal response score is

<2, motor response 1 and brainstem score <1. The use of brainstem reflexes has increased the prediction of mortality in NTC.[15] Periodical evaluation of brainstem reflexes is important to recognize the intracranial hypertension and herniation syndrome.[16]

The examination of brain stem comprises of respiratory pattern, pupil size, response to light, oculocephalic reflexes, tone and posture. The stage of diencephalic herniation may be mimicked by drugs, toxins, metabolic abnormalities as well as intra- and post-ictal states. If children are seen with even some of the signs of diencephalic or uncal or midbrain/upper pontine phases of central herniation, emergency management of presumed raised ICP is mandatory. Recovery is extremely unlikely if the patient had reached the lower pontine or medullary stage. In our study, out of 24 patients with increased intracranial pressure 4 succumbed.

Raised intracranial pressure exists in varying degrees of severity in all encephalopathies of non-traumatic aetiology (Infectious and non-infectious causes).[17] Signs that are almost always present with increased intracranial pressure and herniation are depressed level of consciousness, hypertension with or without heart rate changes- usually bradycardia, sometimes tachycardia.[18]

In our study, we have found that GCS <8, non-reactive pupillary reflex and tone abnormalities were significant predictors of mortality. Presence of papilledema did not correlate with outcome. The limitation of this study is that it is an observational study with a small sample size done in a retrospective manner. But it has provided an understanding of

the aetiology and predictors of outcome of non-traumatic coma, which can influence the development of better management strategies.

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