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Original Research Article

CLINICO ETIOLOGICAL PROFILE AND OUTCOME OF NON-TRAUMATIC COMA IN CHILDREN

Hema Budigina[1]

[1]*Assistant Professor of Pediatrics, Guntur Medical College, Guntur, A. P., India.*

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Corresponding Author:
Dr. Hema Budigina
Assistant professor of pediatrics,
Guntur medical college, Guntur, A. P
Email: hemachakri23@gmail.com

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ABSTRACT

Background: Aim: The aim of this study is to study the etiology, clinical profile and outcome of non-traumatic coma in children admitted to pediatric ward in King George Hospital, Visakhapatnam.

Materials and Methods: This prospective observational study was conducted on 75 children who were admitted in the pediatric intensive care unit of King George Hospital, Visakhapatnam, over a period of 18 months i.e. from December 2008 to June 2010.

Results: In the present study, CNS infections are the most common causes of non-traumatic coma in children accounting for 56% of cases followed by status epilepticus (20%). Among the CNS infections cerebral malaria is the most common cause (35.7%) followed by TBM and bacterial meningitis. There is no significant sex difference in the incidence of coma. Clinical variables like hypothermia, bradycardia, apnoea, hypotension, non-reacting pupils, absent extra ocular movements, flaccidity associated with high mortality and severe disability. As the Glasgow Coma Scale score is decreasing, mortality rate was progressively increasing. Mortality rate was high in children below the age of 3 years. Mortality was high with CNS infections and infestations followed by toxic metabolic causes. Normal outcome was more with toxic-metabolic causes followed by status epilepticus. Disability was more with CNS infections and infestations followed by post status epilepticus group.

Conclusion: The present study concluded that, early recognition of the danger signs and appropriate measures taken early to combat the problem will improve the outcome. Most of the CNS infections are curable, so early institution of appropriate treatment will decrease mortality and disability.

Keywords: CNS Infections, epileptic, Mortality, Glasgow Coma Scale, Bacterial meningitis.

INTRODUCTION

Non traumatic coma in children is an important pediatric emergency accounting for 10-15% of all hospital admissions. The episodes were defined on the basis of GCS score <12 lasting for >6 hrs. It can result from a wide range of primary etiologies. Neurological outcome is often of foremost concern to parents and physicians. It may range from absence of impairment to severe disability or death. Etiology of coma and clinical status at the time of presentation are likely predictors of outcome. A better understanding of causes and outcome is essential to help improve the approach and to plan rational management of non-traumatic coma.[1-4]

In this prospective study we have examined the etiology, clinical signs, and severity of non-traumatic coma with a view to predict outcome.

Aim of the Study

The aim of this study is to study the etiology, clinical profile and outcome of non-traumatic coma in children admitted to pediatric ward in King George Hospital, Visakhapatnam.

MATERIAL AND METHODS

This prospective observational study was conducted on 75 children who were admitted in the pediatric intensive care unit of King George Hospital,

Visakhapatnam, over a period of 18 months i.e. from December 2008 to June 2010.

Inclusion and Exclusion Criteria

All the children between 2 months to 12 years of age, presenting with coma were eligible for inclusion in the study. Those with history of trauma were excluded from the study.

Clinical profile was recorded at admission and patients were reexamined before discharge to record the outcome. The clinical variable recorded were the following.

1. Pulse rate and pulse volume
2. Temperature
3. Blood pressure
4. Respiratory pattern
5. Coma severity using modified Glasgow coma scale
6. Pupillary size and response to light
7. Extra ocular movements
8. Corneal reflex
9. Neck rigidity
10. Motor response
11. Papilledema
12. Seizures if any

The etiology of coma was determined on the basis of history, clinical examination, and relevant laboratory investigations like,

1. Metabolic work up
2. Lumbar puncture
3. CT scan or MRI scan brain
4. EEG
5. Complete blood picture
6. Smear for MP or QBC
7. ABG analysis
8. Liver function tests
9. Urinalysis including ketone bodies
10. X-ray skull lat view
11. Mantoux and CXR

Etiology was classified into 5 groups. They are

1. CNS infections and infestations
2. Toxic metabolic including HIE following shock (e.g. hepatic coma, diabetic
1. coma, poisoning.)
2. Post status epilepticus
3. Intracranial bleed (other than trauma, i.e. DIC, ITP, DHF etc.)
4. Others (e.g. Hypertensive encephalopathy, congenital A-V Malformations etc.).

RESULTS

A total of 75 children were observed in this study. Among those 40% were of 4-5 years age and 13.3% were of less than 1 year age group. Second largest group was the 6-12 age group which contributes 26% of the total study population. [Table 1]

Among the 75 children of the study group 53.3 (n=40) were boys and 46.6% (n=40) were girls. Sex incidence was almost equal for both sexes. [Table 2]

All right you the study group central nervous system infections accounted for majority (56%) of the cases. Post status epilepticus and toxic metabolic end encephalopathy accounted for 15% and 12% respectively. Intracranial bleed and hypertensive encephalopathy accounted 3% each. [Table 3]

Cerebral malaria accounts for majority of the CNS infections and infestations (35.71%), as most of the cases came from malaria endemic areas situated near Visakhapatnam. TB meningitis and bacterial meningitis had similar incidence (23.8%). Only one case of multiple neurocysticercosis was presented with coma following seizures during the study period. [Table 4]

Poisonings like kerosene, organophosphorus, or drug overdoses are included in the same entity and they accounted for 41.66%. Hypoxic ischemic encephalopathy following septic shock or severe dehydration due to cardiopulmonary compromise accounted for 25%. Hepatic coma and diabetic ketoacidosis accounted for two cases each contributing 16% of each among the total of 12 toxic metabolic causes. [Table 5]

Abnormal temperature includes hypo (5.3%) and hyperthermia (53.3%). Abnormal heart rate includes bradycardia (5.3%) and tachycardia (64%)

Abnormal BP includes hypertension (16%) and hypertension (17.3%). Abnormal respiratory pattern includes apnoea (6.6%), keen Stokes or ataxic breathing (45.3%).

Abnormal pupils include non-reacting (20%) and abnormally reacting (60%). Abnormal extraocular movements include absent movements (26.6%), lateral rectus palsy (16%).

Abnormal motor responses include hypotonia (34.6%) and hypertonia (48.48%). 33.33% children presented without seizures and 66.66% presented with seizures. Among the 75 cases of this study group 60% had papilledema at the time of admission and 73.33% of the children had neck rigidity. [Table 6]

A GCS score of less than 12 was taken as coma. 26.6% children presented with a GCS score of 6 and 10.6% score of 4.16% children presented with a GCS score of 3. [Table 7]

Out of the 75 children 53 were survived (70.6%9 and 22 were died (29.3%). Survival rate was more in post status epilepticus group where 12 out of 15 were survived. Mortality rate was higher in CNS infections and infestations group when compared to toxic metabolic group. Mortality was very high with intracranial bleed but the number of the group was very small to give a conclusive statement. [Table 8]

Disability was more in CNS infections and infestations group (52.3%). Normal outcome was more in toxic metabolic group (41.6%). Outcome was better in post status epilepticus group as compared to CNS infections and infestations group. As a whole 24% of the study population (n=75) had normal outcome and 46.6% children survived with disability. [Table 9]

Severe disability was more with TB meaning it isencephalitis. Normal outcome was more with cerebral malaria probably due to early intervention of effective treatment with artesunate. [Table 10]

100% normal outcome was observed with diabetic ketoacidosis. One out of the 3 cases of hypoxic ischemic encephalopathy had severe disability, and another had moderate disability. Most of the poisoning cases recovered without any disability. As a whole 33.26% of the toxic metabolic cases (n=12) survived with disability. [Table 11]

52.17% of children with abnormal pulse volume, 48% of children with abnormal BP and abnormal respiratory patterns died. All of the children who presented with absent corneal reflex died. 46.8% of the children with abnormal extraocular movements and 75% of the children with GCS score of 3 were died.

Table 1: Age distribution of study population

Age group	No. of patients	Percentage
Less than 1 year	10	13.3%
1 – 3 years	15	20%
4 – 5 years	30	40%
6 – 12 years	20	26.6%
Total	75	100%

Table 2: Sex distribution of study population

Group	No. of patients	Percentage
Boys	40	53.3%
Girls	35	46.6%
Total	75	100%

Table 3: Etiology of coma in the study population

Etiology	Incidence	Percentage
CNS infections and infestations	42	56%
Toxic-metabolic	12	16%
Post status epilepticus	15	20%
Intracranial bleed	3	4%
Others	3	4%
Total	75	100%

Table 4: Incidence of various CNS infections and infestations in the causation of coma in the study population

Etiology	No of Patients	Percentage
Cerebral malaria	15	35.71%
TB meningitis	10	23.80%
Bacterial meningitis	10	23.80%
Encephalitis	6	12.28%
Multiple neurocysticercosis	1	2.38%
Total	42	100%

Table 5: Toxic – metabolic causes of coma in the study population

Cause	No. of patients	Percentage
Poisoning	5	41.66%
Hypoxic ischemic encephalopathy	3	25%
Hepatic coma	2	16%
Diabetic ketoacidosis	2	16%
Total	12	

Table 6: Clinical variables of the study population

Clinical variable	Normal	%	Abnormal	%
Temperature	31	41.33	44	58.66
Heart rate	23	30.66	48	64
Pulse Volume	52	69.33	23	30.66
Blood Pressure	50	66.66	25	33.33
Respiratory pattern	36	48	39	52
Pupils	15	20	60	80
Corneal reflex	68	90.66	7	9.33
Extra ocular movements	43	57.33	32	42.66
Motor response	13	17.33	61	81.33
Neck rigidity	Absent in 20	26.66	Present in 55	73.33
Papilledema	Absent in 30	40	Present in 45	60
Seizures	Absent in 25	33.33	Present in 50	66.66

Table 6: Coma severity in the study population

GCS score	No. of patients	Percentage
8	10	13.3%
7	15	20%
6	20	26.6%

5	10	13.3%
4	8	10.6%
3	12	16%
Total	75	100%

Table 7: Outcome of the study population

Etiology (n=75)	No. of patients	Survived	%	Deaths	%
CNS infections and infestations	42	29	69.04	13	30.95
Toxic – metabolic	12	9	75	3	25
Post status epilepticus	15	12	80	3	20
Intracranial bleed	3	1	33.33	2	66.66
Others	3	2	66.66	1	33.33
Total	75	53	70.6%	22	29.3%

Table 8: Normal outcome & Disability in survived patients

Etiology	Normal	Mild Disability	Moderate Disability	Severe Disability
CNS infections & infestations(n=42)	7(16.6%)	12(28.5%)	5(11.9%)	5(11.9%)
Toxic-metabolic (n=12)	5(41.6%)	2(16.6%)	1(8.3%)	1(8.3%)
Post status epilepticus(n=15)	5(33.3%)	1(6.6%)	4(26.6%)	2(13.3%)
Intracranial bleed(n=3)	-	-	-	1(33.3%)
Others(n=3)	1(33.3%)	-	1(33.3%)	-
Total	18(24%)	15(20%)	11(14.6%)	9(12%)

Table 9: Normal outcome and disability in CNS infections and infestations

Etiology (n=42)	Normal	Mild Disability	Moderate Disability	Severe Disability
Cerebral malaria(n=15)	4(26.6%)	5(33.3%)	1(6.6%)	1(6.6%)
TB meningitis(n=10)	-	4(40%)	2(20%)	2(20%)
Bacterial meningitis(n=10)	2(20%)	3(30%)	2(20%)	-
Encephalitis(n=3)	-	-	-	2(33.3%)
Multiple neurocysticercosis(n=3)	1(100%)	-	-	-
Total	7(16.6%)	12(28.57%)	5(11.9%)	5(11.9%)

Table 10: Normal outcome and disability in toxic-metabolic encephalopathy

Etiology	Normal	Mild Disability	Moderate Disability	Severe Disability
Poisoning(n=5)	3(60%)	1(20%)	-	-
Hypoxic-ischemic encephalopathy(n=3)	-	-	1(33.3%)	1(33.3%)
Hepatic encephalopathy(n=15)	-	1(50%)	-	-
Diabetic ketoacidosis(n=2)	2(100%)	-	-	-
Total	5(41.66%)	2(16.66%)	1(8.3%)	1(8.3%)

Table 11: Clinical variables associated with fatal outcomes

Clinical variable	Normal	Death	Abnormal	Death
Temperature	31(41.33%)	4(12.9%)	44(58.66%)	18(41%)
Heart rate	23(30.66%)	3(13.04%)	48(64%)	19(39.5%)
Pulse Volume	52(69.33%)	10(19.2%)	23(30.66%)	12(52.17%)
Blood Pressure	50(66.66%)	10(20%)	25(33.33%)	12(48%)
Respiratory pattern	36(48%)	3(8.3%)	39(52%)	19(48.7%)
Pupils	15(20%)	1(6.6%)	60(80%)	21(35%)
Corneal reflex	68(90.66%)	15(22.5%)	7(9.33%)	7(100%)
Extra ocular movements	43(57.33%)	7(16.27%)	32(42.66%)	15(46.8%)
Motor response	13(17.33%)	2(15.38%)	61(81.33%)	20(32.78%)
Neck rigidity	Absent in 20(26.66%)	4(20%)	Present in 55(73.33%)	18(32.72%
Papilledema	Absent in 30(40%)	14(46.6%)	Present in 45(60%)	8(17.7%)
Seizures	Absent in 25(33.33%)	3(12%)	Present in 50(66.6%)	15(30%)

GCS score	No. of patients	Deaths
8	10(13.33%)	1(10%)
7	15(20%)	3(20%)
6	20(26.66%)	4(20%)
5	10(13.33%)	2(20%)
4	8(10.66%)	3(37.5%)
3	12(16%)	9(75%)

DISCUSSION

In comatose patients, evidence of widespread damage of brain stem or cerebral hemispheres at onset usually predicts death and severe disability. Therefore, clinical signs that reflect extent and severity of dysfunction of cerebral hemispheres and brain stem were studied.

Glasgow Coma Scale score reflects the integrity of cerebral functions. Eye movements, pupillary response, corneal responses primarily express functions regulated by the brain stem.

Breathing pattern and signs of abnormal tone of limbs and posturing indicate the extent and severity of cerebral hemisphere as well as brain stem damage.

It was observed that CNS infections were the commonest cause of non- traumatic coma. This is also supported by other studies, wherein infections of the CNS were found to be the leading causes of non-traumatic coma in children. However, the type of infection seems to vary in different regions. TB meningitis was an important cause of coma in a study done at Chandigarh where as cerebral malaria was the common cause of coma in the present study. [Table 12]

The importance of infective etiologies in children is in sharp contrast to adult hospital-based series where degenerative and cerebrovascular pathologies predominate.

Among the non-infectious causes, status epilepticus was the commonest and it is also comparable with other studies.

In series from Canada toxic-metabolic causes were equally in incidents with CNS infection. It was the second most common cause in a study from Chandigarh; In contrast it was seen in 16% of the cases in the present study.

In the present study the modified Glasgow coma scale recorded at admission had significant association with outcome; Mortality rates progressively increased with the decreasing GCS score.

All the signs of circulatory instability predicted higher mortality and poor neurological outcome. Bradycardia had a very high association with mortality; three out of the four children with bradycardia at admission were died. Tachycardia was also associated with increased risk of death when compared with a normal heart rate.

Hypotension was a poor prognostic sign; 9 out of 12 children with hypotension at admission died. This is similar to the study by Johnston and Seshia, wherein 14 of 15 hypotensive children died. The risk of mortality was almost similar in hypertensive and normotensive patients. This data supports the view that acute hypertension in comatose patients may be neuro protective and should not be brought down unless it is very severe.

The prognosis was best with normal respiratory pattern while apnea at admission had highest risk of mortality,[5] out of 5 patients with apnea died. This is similar to a study from Canada in which 60%of apneic children died. Abnormal breathing pattern including Apneustic, ataxic, Cheyne - Stokes respiration also had significant association with increased mortality and moderate to severe neurological disability.

Non-reactive pupils at admission had high mortality. 12 out of 15 children with non-reactive pupils at admission died. In the study by Seshia et al,[6] 8% ofchildren with fixed dilated pupils for more than 2 hours died. Ogunmekan made similar observations in a large retrospective study from Nigeria.

Presence of corneal reflex indicates functional interconnections in the pons. Absent corneal reflexes in children with deep coma associated with high mortality.

Presence of Doll's eye movements suggests interconnections between cranial nerve nuclei III, IV and VI via the medial longitudinal fasciculus and intact vestibular input to this system. Asymmetrical or partial absence of eye movements generally indicates asymmetric brainstem lesions in midbrain or pons while complete absence of eye movements indicates bilateral brain stem lesions or toxic metabolic encephalopathy.

In the present study 84% of children with preserved extra ocular movements (EOM) at presentation survived and 16% were died, where as 65% of children with absent EOM died and.

None of the children with impaired eye movements had normal neurological outcome. This goes with the earlier studies. Seshia et al,[7] observed that 67% of children who had normal EOM recovered and 16% died; in contrast, all those with absent EOM died and needed assisted ventilation.

In the present study abnormal motor response associated with higher mortality. Interestingly it was flaccidity that was associated with higher as compare it to hypertonia. 46% of those with flaccidity died whereas 22% of those with hypertonia died. This is similar to findings of Seshia et al where in 82% children with flaccidity died. [Table 13]

Overall mortality in the present study is 29.3%. This is similar to other paediatric hospital based series from Nigeria (26.7%) and Canada (26.7%). Mortality was considerably lower than that reported in adults. Mortality rate among children under 3 years was significantly higher in the present study (80%).

It was related to higher frequency of toxic-metabolic causes, intracranial bleed and higher mortality with CNS infections.

The incidence and outcome of coma were not associated with gender. Seshia et al also did not observe any significant difference in the incidence of coma between the two sexes. Mortality was very high with IC bleed but the number was too small for any meaningful conclusion. Toxic-metabolic group and Post status epilepticus group fared significantly better than the CNS infections group.

In the study by Seshia et al,[7] both toxic-metabolic group and CNS infections group had similar outcome. In the study by Arun Bansal et al 8 outcome of CNS infections group was better than toxic-metabolic group. In the present study only 16.6% of the total

CNS infection cases had normal outcome and 52.38% became disabled, among these 11.9% had severe disability. [Table 14]

Table 12: Comparison of causes of non-traumatic coma in childhood reported by various authors

Etiology	Present study(n=75)	Vijaykumar et al[5] (UK)(n=328)	Ogunmekan et al[6](Nigeria)(n=225)	Seshia et al[7](canada) (n=75)	Arun Bansal et al[8] (Chandigarh)(n=100)
CNS infections and infestations	42(56%)	164(50%)	103(45.8%)	29(48.7%)	60(60%)
Toxic-metabolic	12(16%)	52(12%)	52(23.1%)	29(48.7%)	19(19%)
Status epilepticus	15(20%)	88(27%)	64(28.5%)	11(14.7%)	10(10%)
IC bleed	3(4%)	-	-	3(4%)	7(7%)
Others	3(4%)	24(7%)	-	4(4%)	4(4%)

Table 13: Comparison of clinical variables associated with fatal outcome reported by various authors

Clinical Variable	Present study	Seshia et al[7]	Arun Bansal et al[8]
Hypotension	9/12(75% died)	93.3%	67%
Apnoea	5/5(100% died)	60%	90%
NR pupils	12/15(80% died)	68%	36%
Absent EOM	13/20 (65% died)	100%	76%
Flaccidity	12/26 (46%)	82%	53%

Table 14: Comparison of outcomes of non-traumatic coma in childhood reported by various authors

Sl. No	Study	Normal	Disabled	Death
1.	Present study (n=75)	17(22.6%)	35(46.6%)	22(29.3%)
2.	Vijaykumar et al 5 (n=328)(UK)	178(54%)	57(18%)	39(12%)
3.	Ogunmekan et al 6 (n=225) (Nigeria)	102 (45.33%)	63(28%)	60(26.66%)
4.	Seshia et al 7 (n=75)(Canada)	34(45.33%)	21(28%)	20(26.6%)
5.	Arun Bansal et al 8 (n=100)(Chandigarh)	10(10%)	55(55%)	35(35%)

CONCLUSION

CNS infections are the most common causes of non-traumatic coma and disability in children. Clinical signs at the time of admission are good predictors of outcome, so early recognition of the danger signs and appropriate measures taken early to combat the problem will improve the outcome. Most of the CNS infections are curable, so early institution of appropriate treatment will decrease mortality and disability.

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