



Clinical Study of Coma in Children

Authors

Luke Ravi Chelliah^{1*}, Karthick AR², Theranirajan E^{3*}, Dr Gangadharan S²

¹Associate Professor, Department of Paediatrics, Govt. Vellore Medical College, Vellore

²Assistant Professor, Department of Paediatrics, Govt. Vellore Medical College, Vellore

³Professor, Department of Paediatrics, Govt. Vellore Medical College, Vellore

*Corresponding Author

Dr Theranirajan E

Professor of Paediatrics, Department of Paediatrics, Govt. Vellore Medical College, Vellore

Ph: 9003517417, Email: theranirajan1966@gmail.com

ABSTRACT

Background: Coma is a state of unarousable psychogenic unresponsiveness, in which the subjects lie with eyes closed. It has been an enigma for clinicians for many years and searching for the cause and prognosis is always a huge task.

Objective: To study the etiology of coma, find out morbidity and mortality pattern and to determine the outcome of coma in children.

Method: 60 children in the age group of 2 months to 12 years for a period of one year from January 2016 to December 2016, admitted with non-traumatic coma in the Department of Paediatrics, Govt. Vellore Medical College, Vellore were included in the study. A detailed history was taken and thorough examination was done according to the proforma. Patients were evaluated daily till discharge as advised or death. Outcome was classified into good recovery, moderate disability, severe disability, persistent vegetative state and death.

Results: The incidence of non-traumatic coma was 1.18% among all paediatric admissions. The common cause of non-traumatic coma in the present study was due to Metabolic-Toxic lesions (81.66%) as compared to structural lesions (18.34%). Mortality due to structural causes was high (18.18%) when compared to metabolic-toxic lesions. Low GCS, abnormal pupillary reflex, absent corneal reflex and Doll's eye movement had significant impact on prognosis. Children with coma duration more than 48 hours had 22.22% mortality (p value 0.02).

Conclusion: CNS infections are the commonest cause of non-traumatic coma and it is possible to predict mortality and morbidity due to non-traumatic coma with simple clinical signs.

Keywords: Non-traumatic coma, metabolic-toxic lesions, structural lesions.

Introduction

Coma is a state of unarousable psychogenic unresponsiveness in which subject lie with eyes closed⁽¹⁾. Non Traumatic Coma constitutes an important pediatric emergency. It is a non-specific sign with wide differential diagnosis. CNS

infections are the most common cause⁽²⁾. Toxic-metabolic, status-epilepticus, hypoxic-ischemic, intracranial bleed etc. are other important causes. The primary concern for both physicians and parents is the neurological outcome⁽³⁾. Etiology and condition of the child at the time of

presentation along with simple clinical signs have huge predictive value on the outcome ⁽⁴⁾.

Methods

This prospective observational study was done in the Department of Paediatrics, Govt. Vellore Medical College and Hospital, Vellore from January 2016 to December 2016. Patients aged 2 months to 12 years admitted with loss of consciousness for a minimum of 6 hours were included in the study. The study was carried out after obtaining Institutional Ethics Committee clearance and informed consent from the parents. Patients who were less than 60 days of age and with history of trauma were excluded from the study. A detailed history was taken and thorough examination was done according to the proforma. Immediate resuscitative measures were taken and specific treatment modality was started and later changed according to the patient's disease course. The level of consciousness was assessed on admission according to Glasgow coma scale and children with score less than 7 were included in the study. Patients were evaluated daily till discharge as advised or death.

Definitions of study variables were as follows:

Coma: A state of unresponsiveness without evidence of awareness of self or environment, a state from which, the patient cannot be aroused by vocal or sensory stimuli.

Coma Severity: based on score obtained from modified Glasgow coma scale ⁽⁵⁾.

Pupils: i) normal – both pupils equal in size, 2-3 mm and reacting equally to light. ii) abnormal – small (<1 mm) or dilated (> 4mm), unequal and non-reacting to light ⁽³⁾.

Corneal Reflex: Absent or present

Doll's Eye Response: Conjugate deviation of eye in the direction opposite to head's movement – absent or present ⁽⁶⁾.

Recovery: No motor deficit, ataxia, cranial nerve palsy, and functional level back to pre-illness state ⁽³⁾.

Mild Disability: Minimal alteration of tone / deep tendon reflexes, isolated cranial nerve palsy, weakness of grade 4 or ataxia ⁽³⁾.

Moderate Disability: Moderate weakness of grade 3 or ataxia, behavior disturbance and multiple cranial nerve involvement ⁽³⁾.

Severe Disability: Severe weakness (< grade 3) or ataxia and quadriplegia ⁽³⁾.

Statistical analysis was carried out with SPSS software Chi-square test, Fisher's exact test and Pearson Correlation Coefficient were used with confidence interval of 95%.

Results

60 children were included in the study. The incidence of non-traumatic coma was 1.18%. 32 children (53.33%) were male and 28 (46.67%) were female. 20 (33.33%) children were in the age group of 2 months to 2 years, 18 (30%) were between 2 - 5 years and 22 (36.67%) were between 5 - 12 years. Etiologically, 11 (18.34%) had structural lesions and 49 (81.66%) children had metabolic-toxic lesions. Among the structural causes, 1 (1.67%) had neoplasm, 1(1.67%) had vascular lesion, 6 (10%) had focal infection and 3 (5%) had hydrocephalus. 31 (51.67%) children had CNS infection. 13 (21.66%) had seizure disorder. 5 children had metabolic causes which includes hepatic coma (2), hypoglycemia (2) and hypocalcemia (1).

Table 1. Age and Sex distribution

Age Distribution		
Age Group	No. of Cases	Percentage (%)
2mo – 2 years	20	33.33 %
2 – 5 years	18	30 %
5 – 12 years	22	36.67 %
Sex Distribution		
Sex	No. of Cases	Percentage
Male	32	53.33%
Female	28	46.67%

In our study, 5 children (8.33%) expired, 3 (5%) went into persistent vegetative state and 2 (3.33%) cases had moderate disability. The overall mortality due to structural lesion was 18.18% compared to 6.12% due to metabolic-toxic lesions. Individual mortality figures were 2 (33.33%) in focal infection, 2 (6.45%) in CNS infection and 1 (7.69%) in status epilepticus.

Table 2. Etiology and outcome

S. No.	Etiology	No. of cases	Expired	Persistent Vegetative State	Disability		Good Recovery
					Severe	Moderate	
STRUCTURAL LESIONS							
1.	Neoplasm	1	-	-	-	-	1 (100%)
2.	Vascular	1	-	-	-	-	1 (100%)
3.	Focal Infection	6	2 (33.33%)	-	-	-	4 (66.67%)
4.	Hydrocephalus	3	-	1 (33.33 %)	-	1 (33.33 %)	1 (33.33 %)
METABOLIC – TOXIC LESIONS							
1.	Hypoxic Ischemia	-	-	-	-	-	-
2.	Intrinsic Metabolic Disorders	5	-	-	-	-	5 (100%)
3.	Exogenous toxins and poisons	-	-	-	-	-	-
4.	Infections	31	2 (6.45%)	2 (6.45%)	-	1 (3.23%)	26 (83.87%)
5.	Seizures, status epilepticus and post ictal state	13	1 (7.69%)	-	-	-	12 (92.31%)

Table 3. Clinical signs and their association with outcome

Variable	Total	Survived	Died	P Value
GCS				
3	3	0	3	Correlation coefficient of -0.9 p value of 0.03 (significant)
4	6	5	1	
5	12	11	1	
6	18	18	0	
7	21	21	0	
Seizures				
Present	44	41	3	p value of 0.481 (not significant)
Absent	16	14	2	
Pupillary Reflex				
Present	46	45	1	p value of 0.001 (significant)
Absent	14	10	4	
Doll’s Eye Response				
Present	55	53	2	p value of 0.0004 (significant)
Absent	5	2	3	
Corneal Reflex				
Present	52	51	1	p value of 0.001 (significant)
Absent	8	4	4	
Duration of Coma				
6 – 12 hours	8	8	0	Pearson Correlation Factor R is 0.93 p value of 0.02 (significant)
12 – 24 hours	14	14	0	
24 – 48 hours	19	19	1	
48 – 72 hours	12	9	2	
> 72 hours	6	4	2	

Discussion

Non Traumatic coma is a life threatening emergency which requires aggressive intervention in order to preserve intact brain function. Etiology of coma along with clinical status at presentation are important predictors of outcome. Non-

traumatic coma represents a non-specific sign with plethora of differential diagnosis. Good clinical knowledge and advanced lab support are imperative for narrowing the diagnosis. Simple clinical signs are often good predictors of outcome^(3,4)

CNS infection was the commonest cause for non-traumatic coma accounting for 31 cases (51.67%). Other studies done previously also reiterate the same⁽⁷⁻¹¹⁾. In case of adults, degenerative and cerebrovascular diseases predominate⁽¹²⁾. In the present study, among the CNS infection, encephalitis contributed to 16 cases (20%), pyogenic meningitis to 5 cases (8.33%) and tuberculous meningitis to 5 cases (8.33%). The next common cause in the present study was seizure disorder accounting to 13 cases (26.66%). 5 metabolic disorders were encountered leading to coma. Among that, 2 were due to hepatic coma, 2 were due to hypoglycemia and 1 due to hypocalcemia, though hypoglycemia leading to coma is very rare⁽¹³⁾. No cases of poisoning and hypoxic ischemia were encountered in this study. Out of 60 cases, 5 died with overall mortality of 8.34%, 3 went into persistent vegetative state and 2 had moderate disability. Studies done by Bansal et al⁽³⁾, A. M. Ali et al⁽¹⁴⁾ and Fariba⁽⁹⁾ et al reported 35%, 60.5% and 16.6% mortality rates respectively.

Modified GCS scores had significant correlation with outcome with lesser scores having poor outcomes (p value of 0.03). Bansal et al⁽³⁾, Nayana et al⁽⁷⁾ and Ali et al⁽¹⁴⁾ also found similar relation. In studies done previously in both traumatic and non-traumatic comas, mortality was high when Glasgow coma scale was less than 8^(15,16).

There was no significant association between mortality and seizures on admission. Pupillary reaction had good prognostic value (p value 0.001). Abnormal pupil on admission was a strong predictor of poor outcome. Seshia et al⁽¹⁰⁾ and Ogunmekan et al⁽⁸⁾ also had similar findings in their study. Intact corneal reflex indicated intact brainstem response, absence of which prognosticated poor outcomes. Out of 8 children with absent corneal reflex, 4 (50%) expired. Bansal et al⁽³⁾ also reported similar findings. Presence of Doll's eye response indicates intact III, IV and VI cranial nerves. Abnormal response suggests involvement of brainstem. In the present study, 3 children out of 5 with absent doll's eye movement expired (p value 0.0004). Only 2

children out of 55 with intact doll's eye movement expired, which indicated that preserved doll's eye movement was associated with favorable prognosis. Seshia et al⁽¹⁰⁾ also observed similar findings in their study. Duration of coma had significant impact on prognosis and coma lasting for more than 48 hours had poor prognosis (p value 0.02).

Conclusion

This study reiterated the importance of clinical signs and simple clinical signs like low GCS, abnormal pupillary response, absent doll's eye movement and corneal reflex were strong predictors of death following non-traumatic coma.

Acknowledgements

Authors want to thank the caretaker of children for giving consent to participate in the study.

Declarations:

Funding: No funding sources

Conflict of Interest: None

Ethical Approval: The study was approved by Institutional Ethics Committee

References

1. Posner JB, Plum F. Plum and Posner's Diagnosis of Stupor and Coma. Oxford University Press, USA; 2007. 416 p.
2. Wong CP, Forsyth RJ, Kelly TP, Eyre JA. Incidence, aetiology, and outcome of non-traumatic coma: a population based study. Arch Dis Child. 2001 Mar 1;84(3):193-9.
3. Bansal A, Singhi SC, Singhi PD, Khandelwal N, Ramesh S. Non traumatic coma. Indian J Pediatr. 2005 Jun 1;72(6):467-73.
4. Fouad H, Haron M, Halawa EF, Nada M. Nontraumatic Coma in a Tertiary Pediatric Emergency Department in Egypt: Etiology and Outcome. J Child Neurol. 2011 Feb 1;26(2):136-41.
5. Kirkham FJ, Newton CRJC, Whitehouse W. Paediatric coma scales. Dev Med Child Neurol. 2008 Apr;50(4):267-74.

6. Doll's Eye Sign [Internet]. The Free Dictionary.com. [cited 2017 Jul 25]. Available from: <http://medical-dictionary.thefreedictionary.com/Doll%e2%80%99s+Eye+Sign>
7. Nayana Prabha PC, Nalini P, Tiroumourougane Serane V. Role of Glasgow Coma Scale in pediatric nontraumatic coma. *Indian Pediatr.* 2003 Jul;40(7):620–5.
8. Ogunmekan AO. Non-traumatic Coma in Childhood: Etiology, Clinical Findings, Morbidity, Prognosis and Mortality. *J Trop Pediatr.* 1983 Aug 1;29(4):230–2.
9. Khodapanahandeh F, Najarkalayee N. Etiology and Outcome of Non-traumatic Coma in Children Admitted to Pediatric Intensive Care Unit. *Iran J Pediatr.* 2009;19(4):393–8.
10. Johnston B, Seshia SS. Prediction of outcome in non-traumatic coma in childhood. *Acta Neurol Scand.* 1984 Jun 1;69(6):417–27.
11. Tasker RC, Matthew DJ, Helms P, Dinwiddie R, Boyd S. Monitoring in non-traumatic coma. Part I: Invasive intracranial measurements. *Arch Dis Child.* 1988 Aug 1;63(8):888–94.
12. Bates D, Caronna JJ, Cartlidge NE, Knill-Jones RP, Levy DE, Shaw DA, et al. A prospective study of nontraumatic coma: methods and results in 310 patients. *Ann Neurol.* 1977 Sep;2(3):211–20.
13. Kim SY. Endocrine and metabolic emergencies in children: hypocalcemia, hypoglycemia, adrenal insufficiency, and metabolic acidosis including diabetic ketoacidosis. *Ann Pediatr Endocrinol Metab.* 2015 Dec;20(4):179–86.
14. Ali AM, Al-Abdulgader A, Kamal HM, Al-Wehedy A. Traumatic and non-traumatic coma in children in the referral hospital, Al-Hasa, Saudi Arabia. *East Mediterr Health J Rev Sante Mediterr Orient Al-Majallah Al-Sihhiyah Li-Sharq Al-Mutawassit.* 2007 Jun;13(3):608–14.
15. Chaturvedi P, Kishore M. Modified Glasgow Coma Scale to predict mortality in febrile unconscious children. *Indian J Pediatr.* 2001 Apr;68(4):311–4.
16. Palmer S, Bader MK, Qureshi A, Palmer J, Shaver T, Borzatta M, et al. The impact on outcomes in a community hospital setting of using the AANS traumatic brain injury guidelines. *American Association for Neurologic Surgeons. J Trauma.* 2001 Apr;50(4):657–64.