Correlation of risk factors with Glasgow coma scale to predict the severity and outcome of children with non-traumatic coma

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ABSTRACT

Background: Non-traumatic coma is the problem of pediatric group, accounts 10-15% in hospital admissions. Assessment of the severity of coma is useful to speculate the survival. The aim was to assess outcome in pediatric non-traumatic coma with role of Glasgow coma scale and modified Glasgow coma scale.

Methods: Total of 80 cases of non-traumatic coma between 1 month to 12 years, coma severity was assessed by using Glasgow coma scale. A score of less than 8 and more than 8 were used for analysis of outcome.

Results: The maximum number of patients with non-traumatic coma were in the age group of 1 month-5 years, 40 children (50%). On neurological examination 42 (52.5%) children has GCS score of >8, 38 cases (47.5%) has GCS <8, 20 children had meningeal signs, 7 children had cranial nerve deficit (7th nerve), 9 children had deicate posture. Out of 80 cases, 8 cases expired (10%), 4 cases were discharged against medical advice (4%), 68 cases were improved and discharged (85%), among these, 8 cases were discharged with complication (11.7%). Overall mortality was (10%) (8/80), males outnumbered females in frequency with ratio of 1.28:1. CNS infection accounted for almost about 66%.

Conclusions: Children with GCS and MGCS scores of less than 8 have poor prognosis and a very high probability of death. Those with GCS score of more than 8 have good prognosis. Identification of these cases at the outset can help prepare the treating physician to plan critical care referral and to give a preliminary assessment of outcome to the family.

Keywords: Diagnosis, Glasgow coma scale, Non-traumatic coma, Outcome

INTRODUCTION

Acute non-traumatic coma is a common problem in pediatric practice accounting for 10-15% of all hospital admissions and is associated with significant mortality. Assessment of the severity of coma is essential to comment on the likelihood of survival in comatose children. In the last three decades, various scores have been used to assess the severity of coma and to predict its outcome. These includes the Glasgow coma scale, the James adaptation of Glasgow coma scale, the Simpson and Reilly scale, the Children’s coma scale, and the Jacob’s scale.

Among these, the modified Glasgow Coma Scale (MGCS) in spite of its various drawbacks, has been widely used for assessing pediatric coma, though only few studies are available to support its use in pediatric
coma as a whole. This study was conducted to assess the relationship between MGCS, its components and survival in children with acute coma. Coma scales have been devised to determine the depth of unconsciousness in children (Simpson and Reilly) and some have included brainstem signs (Born et al).\(^2\)\

Non-traumatic coma in childhood is an important pediatric emergency. It can result from a wide range of primary etiologies. Neurological outcome in comatose children is concern to parents and physicians. It may range from absence of impairment to severe disability and death. The most important cause of non-traumatic coma in developing countries is CNS infections.\(^5\) Most cases with score of 8 are also comatose. Patient with a GCS score of 8 or less may require aggressive management including mechanical ventilation and intracranial pressure monitoring.\(^2\)

A study of 100 cases of non-traumatic coma in children found CNS infections in 56% cases, with tubercular and pyogenic meningitis encephalitis almost equally common.\(^6\) The other common cause were seizure disorder, Reye’s syndrome, hepatic coma and non-traumatic intracranial bleeds. In diabetic coma, if serum sodium does not increase in parallel with the reduction in plasma glucose, there is a serious risk of cerebral edema and herniation, this is the commonest cause of death in diabetic children.\(^3,\)\(^,\)\(^4\) This is probably preventable if fluid losses are replaced over a 48 hour period with at least 125 mmol of sodium per liter over the first 24 hours.\(^9\)

The prognosis for hypertensive encephalopathy is excellent if an ischemic insult is not superimposed, so if blood pressure is raised in an unconscious patient, it should be reduced extremely slowly.\(^10\)

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. Very little information is available particularly so from developing countries including India. In a prospective study the authors have therefore worked out on the etiology, clinical signs and severity of non-traumatic coma in children with a view to define predictors of outcome.

The current study was to assess the outcome in pediatric non-traumatic coma with role of Glasgow coma scale in children admitted at Narayana Medical College Hospital, Nellore, Andhra Pradesh, India.

### METHODS

Prospective study of 80 paediatric cases between age group of 1 month to 12 years with non-traumatic coma in Narayana Medical College Hospital, Nellore, Andhra Pradesh, India from November 2016 to September 2018.

### Inclusion criteria

All the children admitted with non-traumatic coma in Narayana Medical College Hospital, Nellore, Andhra Pradesh, India from November 2016 to September 2018. Age between 1 month to 12 years.

### Exclusion criteria

Age includes <1 month and >12 years were excluded from the study and the children presenting with traumatic coma.

### Procedure

The detailed neurological and physical examination were carried out, coma severity was assessed by using Glasgow coma scale. In the birth history, detailed antenatal and post-natal history was stressed to know about any predisposing factors responsible for the present illness.

Any neurological or other complications occurring in the course of the study along with morbidity and mortality were noted.

Data was analyzed using a computer aided statistical package SPSS version 18. The statistical methods used were Chi-Square test and student’s t test. All values were expressed as mean percentages.

### RESULTS

Intracranial infection was the most common cause of coma in this study forming the largest group. 17 (21%) children with TB meningitis presented with acute coma. 12 children presented with viral encephalitis (15%), 12 (15%) with pyogenic meningitis, 10 (12%) with seizure disorder, 8 (10%) with atypical febrile seizures, 5 with enteric encephalopathy (6.25%), 5 with measles encephalopathy (6.25%), hepatic coma 4 (5%), hypoglycemic seizures 2 (2.5%) cases, cerebral malaria 2 (2.5%) cases, unknown diagnosis 3 (3.75%). Mortality was highest in the intracranial infection, 8 persons died (10%). About 45 males and 35 females are recorded.

In diagnosis of distribution of cases, 17 TB meningitis, 12 viral encephalitis, 12 pyogenic meningitis, 10 seizure disorder, 8 atypical febrile seizures, 5 enteric encephalopathy, 5 measles encephalopathy, 4 hepatic coma, 2 cerebral malaria, 2 hypoglycemic seizures, and 3 unknown diagnosis cases were recorded.

In modes of presentation, fever 62, convulsions 56, vomiting, headache 20, ear discharge 5, icterus 4, loose stools 4 and hypoglycemic attacks 2 were noticed. The most frequently encountered symptoms in order of frequency in this are fever, convulsions, vomiting, headache, etc.
In level of consciousness, confusion in 12, stuporous in 25, drowsy in 18, coma in 25 cases were observed.

Depending on the level of consciousness, coma and stuporous were the major presentation of cases (31.25%, 31.25% each), drowsiness was seen in 22.5% cases and confusion in 15% cases.

Neurological examination

Confusion in 12, stuporous in 12, drowsy in 18, coma in 25, decerebrate posture in 15, cranial nerve deficits (7th nerve) in 9, exterior plantar response in 30, meningeal signs in 25 cases were observed. 38 cases had GCS of less than 8 the following are the details of the 38 cases (Table 1).

Table 1: Results of cases in Glasgow coma scale of <8.

| GCS <8                  | No. of cases (n=38) | %     |
|------------------------|---------------------|-------|
| Confusion              | 5                   | 13    |
| Stuporous              | 10                  | 26    |
| Drowsy                 | 5                   | 13    |
| Coma                   | 20                  | 52    |
| Cranial nerve deficit  | 7                   | 18    |
| Decerebrate posture    | 9                   | 23    |
| Exterior plantar response | 20               | 52    |
| Meningeal signs        | 20                  | 52    |
| Improved and discharged| 32                  | 84    |
| Expired                | 6                   | 15    |
| Discharged against medical advice | 3          | 7     |

Out of the 38 cases in whom the GCS score was <8, 20 cases had meningeal signs, 20 cases were in coma, 20 cases had exterior plantar response, 10 cases were stuporous, 5 drowsy, 5 confusion, 9 cases decerebrate posture. 9 cases had cranial nerve deficit, the common cranial nerve involved was the 7th nerve (facial nerve) in all the 9 cases. Total 32 (84%) cases were improved and discharged, 3 cases (7%) with unknown diagnosis, 6 (15%) cases expired and 3 (7%) cases were discharged against medical advice.

In 42 cases, the GCS score was >8, 39 patients were improved and discharged without any complications i.e., 92%, 2 cases expired, 1 case was discharged against medical advice.

It is observed in this study that the majority of cases improved without complication and has accounted for about 85%, 4 patients discharged against medical advice and 8 patients expired (Table 2). Among the expired cases, 4 cases were TBM, 2 cases were pyogenic meningitis, 1 case was viral encephalitis, and 1 case was hepatic encephalopathy accounting for 10% of mortality.

Among the 8 patients expired, 6 patients had a GCS score of <8 at the time of admission and 2 patients had GCS >8. So, the likelihood of death in patients with GCS score less than 8 was much higher than when the GCS score was >8. Studies in both traumatic and non-traumatic coma have indicated that mortality is high when the GCS score is less than 8.

Table 2: Results of cases.

| Results                      | No. of cases | %  |
|------------------------------|--------------|----|
| Total no. of case            | 80           |    |
| Improved and discharge       | 68           | 85 |
| DAMA                         | 4            | 5  |
| Expired                      | 8            | 10 |

DISCUSSION

It is a well-known fact that the prognosis in coma depends on its severity. Assessing the severity of coma by subjective, poorly defined terms such as stupor, semicom, deep coma was ineffective in predicting the outcome and there was a great deal of inconsistency when different observers carried out assessment. The Glasgow coma scale 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. It has gained widespread use as it is highly reproducible, can be quickly performed at the bedside and provides useful information on the progress and prognosis of a comatose individual.

In the present study, total number of cases of non-traumatic coma with the role of Glasgow coma scale were 80 cases. There were 40 children were admitted in age group between 1 month to 5 years in total of 80 children. 25 children admitted between age group 5 to 10 years (Table 3).

In the present study, a low total GSC score was found to be associated with adverse short-term outcome. The likelihood of death in patients with GCS less than 8 was much higher than when the GCS was >8 i.e., 6 (15%) cases expired out of 38 cases with GCS <8 and 2 (4%) cases expired out of 42 cases with GCS >8.

In another study conducted at JIPMER, Pondicherry by P. C. Nayana Prabha et al, the ratio was (78.9%, 27.1%). Studies in both traumatic and non-traumatic coma have indicated that mortality is high when the GCS is less than 8.

In the present study of 80 cases, 17 children had TB meningitis, 12 children had viral encephalitis, 12 had pyogenic meningitis, 10 had seizure disorder, 8 had atypical febrile seizures, 5 had enteric encephalopathy, 5 had measles encephalopathy, 4 had hepatic coma, 2 had hypoglycemic seizures, 2 had cerebral malaria, 3 cases with unknown diagnosis.
In the present study, CNS infection accounted for almost 70%, so it was observed that CNS infections were the commonest cause of non-traumatic coma. This is also supported by other studies by Vijay Kumar et al, Ogunmeka et al, Mekan et al, Seshia et al, Matu et al, Sofiah et al, wherein infections of the CNS were found to be the leading cause of non-traumatic coma in children (Table 4). 4,15-18

### Table 3: Age distribution of cases.

| Age group     | Present study | Arun Bansal et al 16 | Prabha et al 11 |
|---------------|---------------|----------------------|-----------------|
| 1 month-5 years | 40            | 2 months-1 year 15   | 3-36 months 100 |
| 6-10 years    | 25            | 1-3 years 25         | 37-72 months 74 |
| 11-12 years   | 15            | 4-5 years 34         | 73 months-108 months 54 |
| >12 yrs       | 26            | 109 months-144 months 42 |

### Table 4: Comparison of causes and mortality of acute non-traumatic coma in childhood reported by various authors.

| Causes             | Present study (n=80) | Vijay Kumar et al 16 (n=328) | Ogunmeka R et al 16 (n=225) | Seshia et al 18 (n=75) | Sofiah et al 18 (n=116) |
|--------------------|----------------------|-------------------------------|-----------------------------|------------------------|-------------------------|
| CNS infections     | 70.00 %              | 50.00                         | 41.80                       | 34.70                  | 69.00                   |
| Atypical febrile   | 1                    | --                           | 4.00                        | 4.00                   | --                      |
| Metabolic          | 2.5                  | 12.00                        | 19.10                       | 14.70                  | 13.00                   |
| Seizure disorder   | 12                   | 27.00                        | 19.00                       | 14.70                  | --                      |
| Hypoxia            | --                   | 4.00                         | 4.00                        | 24.00                  | 5.00                    |
| IC Bleed           | --                   | --                           | --                          | 4.00                   | 3.00                    |
| Unknown diagnosis  | 3.75                 | 7.00                         | --                          | 4.00                   | 9.50                    |
| Overall mortality  | 10                   | 12.00                        | 26.70                       | 26.70                  | 35.790                  |

In the present study, 4 (5%) deaths occurred within the 24 hours of admission using the GCS we could identify children at highest risk of death even on admission. An earlier study showed that 44.2% death occurred within 72 hours of admission and these cases could be identified on admission with the help of GCS scores. 19

When compared to other studies the mortality in metabolic causes was low. In this study, total 6 cases of metabolic causes were admitted, 4 hepatic coma, 2 hypoglycemic seizures 1 death occurred. When compared to a study in Department of Pediatrics and Radio diagnosis, postgraduate Institute of Medical Education and Research Center, Chandigarh, Punjab, India number of cases were 19, 12 died (63.2%). In the present study, one death occurred.

In this study, author concluded that outcome of coma was dependent on the etiology. This is in similar to other authors who have concluded that outcome of coma was dependent on the etiology. 12,20,21 It is important to realize that studies on prognosis of coma are affected by certain issues like belt-fulfilling nature of the underlying disease, host response and treatment strategies and these are likely to have a significant effect on the outcome. Further death in coma often results not from the failure of the primary neurological mechanisms but from other secondary non-neurological causes. Also, the distribution of etiology of coma in this study population was uneven with infection accounting for more than three- fourths of comatose patients. These issues could have affected the assessment of the effect of etiology on outcome in this study.

The present study concluded that children with GCS score of less than 8 on admission have the worst prognosis and a very high probability of death.

Those with GCS scores of more than 8 at the time of admission have a good prognosis and better improvement. Identification of these cases at the outset can help prepare the treating physician to plan critical care referral and to give a preliminary assessment of outcome to the family.

### CONCLUSION

Acute non-traumatic coma is a common problem in pediatric practice accounting for 10-15% of all hospital admissions and is associated with significant mortality. Assessment of the severity of coma is essential to comment on the likelihood of survival in comatose children. Mortality rates progressively increased with decreasing GCS. Patient with GCS score of 8 or less may require aggressive management including ABC,
mechanical ventilation and intracranial pressure monitoring. A good prognosis was observed in patients whose GCS was more than 8 on admission. CNS infections accounted for majority of the cases of pediatric non-traumatic coma. 3 cases had unknown diagnosis even though extensive investigations were done which require further work up.

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REFERENCES

1. Tasker RL, Cole GF. Acute encephalopathy of childhood and intensive care. In: Brett EM, eds. Pediatric Neurology. 3rd ed. Edinburgh, Churchill Livingstone; 1996.
2. Simpson D, Reilly P. Paediatric coma scale. Lancet. 1982;320(8295):450.
3. Born JD, Hans P, Albert A, Bonnal J. Interobserver agreement in assessment of motor response and brain stem reflexes. Neurosurg. 1987;20(4):513-7.
4. Ogunmekan AO. Non-traumatic coma in childhood: etiology, clinical findings, morbidity, prognosis and mortality. J Trop Pediatrics. 1983;29(4):230-2.
5. Swaiman KF. Pediatric Neurology- Principles and Practices. 3rd ed. Elsevier Health Sciences; 2006: 864-865.
6. Bansal A, Singhi SC, Singhi PD, Khandelwal N, Ramesh S. Non traumatic coma. Indian J Pediatrics. 2005;72(6):467-73.
7. Shrier DA, Shibata DK, Wang HZ, Numaguchi Y, Powers JM. Central brain herniation secondary to juvenile diabetic ketoacidosis. Am J Neuroradiol. 1999;20(10):1885-8.
8. Edge JA, Adams ME, Dunger DB. Causes of death in children with insulin dependent diabetes 1990-96. Arch Dis Childhood. 1999;81(4):318-23.
9. Mahoney CP, Vlcek BW, Aguila M. Risk factors for developing brain herniation during diabetic ketoacidosis. Pediatric Neurol. 1999;21:721-7.
10. Wright RR, Mathews KD. hypertensive encephalopathy in childhood. J Child Neurol. 1996;11:193-6.
11. Prabha NPC, Nalini P, Serene VT. Role of Glasgow coma scale in pediatric nontraumatic coma. Indian Pediatr. 2003;40:620-25.
12. Bates D. Defining prognosis in medical coma. J Neurol Psychiatry. 1981;44:552-4.
13. Teasdale G, Jennett B. Assessment of coma and impaired consciousness: a practical scale. Lancet. 1974;2:81-4.
14. Prasad K. The Glasgow coma scale—a critical appraisal of its clinimetric properties. J Clin Epidemiol. 1996;49:755-63.
15. Kumar VK, Knight R, Prabhakar P, Murphy PJ, Sharpes PM. Neurological outcome in children with non-traumatic coma admitted to a regional pediatric intensive care unit. Arch Dis Child. 2003;88:A30-2.
16. Seshia SS, Seshia MMK, Sachadeva RK. Coma in childhood. Dev Med Child Neurol. 1977;19:614-28.
17. Matuwa WB, Matekere NJ. Causes and early prognosis of non-traumatic coma in Tanzania. Muhimbili Medical Centre Experience. Trop Geogr Med. 1987;39:330-5.
18. Sofiah A, Hussain HM. Childhood non-traumatic coma in Kuala Lumpur, Malaysia. Ann Trop Pediatr. 1997;17:327-31.
19. Awasthi S, Moin S, Iyer S, Meenakshi, Rehman H. Modified Glasgow Coma Scale to predict mortality in children with acute infections of the central nervous system. Nat Med J India. 1997;10:214-6.
20. Sacco RL, Gool R, Mohr JP, Hauser WA. Non-traumatic coma. Glasgow coma score and coma etiology as prediction of 2-week outcome. Arch Neurol. 1994;47:1181-4.
21. Levy DE, Bates D, Caronna JJ, Cartilidge NE, Knill Jones RP, Lapinski RH, et al. Prognosis in non-traumatic coma. Ann Intern Med. 1981;94:293-301.

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