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Research Article

Non traumatic coma in children: a prospective observational study

Ikhlas Ahmad, Kaisar Ahmed, Imran Ahmad Gattoo*, Mohmmad Younus Mir, Muddasir Maqbool, Akhter Rasool Baba

Department of Pediatrics, Government Medical College, Srinagar, J&K, India

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***Correspondence:**
Dr. Imran Ahmad Gattoo,
E-mail: immz24@gmail.com

ABSTRACT

Background: Non Traumatic Coma (NTC) in children is an important pediatric emergency. It has been an enigma for clinicians for many years. To find the cause of coma and to determine the prognosis is a taxing question for the attending clinicians and the primary concern for relatives in every case. Objective: To study the etiology, clinical profile, and to determine the clinical signs predictive of outcome in non-traumatic coma in children aged between 6 months and 12 years.
Methods: Sixty-one consecutive cases with details of demographic data, history and clinical examination at admission and after 48 hours were recorded in a predesigned proforma. The clinical variables recorded were heart rate, respiratory rate and pattern, blood pressure, temperature, coma severity (using modified Glasgow Coma Scale), pupillary size and response to light, extra ocular movements and fundus picture. Etiology was classified into infections, toxic-metabolic, hypoxic-ischemic, post-status epilepticus, intracranial bleeding and miscellaneous. The outcome was recorded as survived or died, and among those who survived as normal, mild, moderate, or severe disability.
Results: Infection was the commonest cause of non-traumatic coma accounting for 25 (41%) cases. Toxic-metabolic 16 (26.2%), status-epilepticus in 10 (16.4%), 5 (8.2%) were caused by structural CNS lesions and in 5 (8.2%) cause could not be determined. In infectious causes, encephalitis was the largest subgroup contributing to 10 (16.4%) cases. Overall mortality was 33.9 % in our study. 40.9 % were normal or had mild disability and 23 % had moderate to severe disability when examined at discharge. The mortality was higher in cases of toxic-metabolic coma (43.8%) than in CNS infections (29.2%) or status-epileptics (10%). Factors that correlated significantly with mortality both at admission and after 48 hours were age ≤3, poor pulse volume, hypotension, abnormal respiratory pattern, abnormal pupils, absent corneal reflex, abnormal extra-ocular movements, lower modified GCS and papilledema.
Conclusions: CNS infections are the commonest cause of non-traumatic coma; simple clinical signs are strong predictors of both morbidity and mortality.

Keywords: Non traumatic coma (NTC), Meningitis, Encephalitis

INTRODUCTION

Coma has been an enigma for clinicians for many years. To find the cause of coma and to determine the prognosis is a taxing question for the attending clinicians and the primary concern for relatives in every case.

Non Traumatic Coma (NTC) in children is an important pediatric emergency. Etiologically it can be divided into three broad categories: those without focal neurologic signs (e.g., metabolic encephalopathies); meningitis syndromes, characterized by fever or stiff neck and an excess of cells in the spinal fluid (e.g., bacterial meningitis, subarachnoid hemorrhage); and conditions associated with prominent focal signs (e.g., stroke,

cerebral hemorrhage). In most instances coma is part of an obvious medical problem such as drug ingestion, hypoxia, stroke, trauma, or liver or kidney failure. Conditions that cause sudden coma include drug ingestion, cerebral hemorrhage, trauma, cardiac arrest, epilepsy, or basilar artery embolism. Coma that appears subacutely is usually related to a preceding medical or neurologic problem, including the secondary brain swelling of a mass lesion such as tumor or cerebral infarction.[1] NTC in children is a non-specific sign with a wide potential of differential diagnosis[2]. CNS infections are the most common cause.[2-5] Toxic-metabolic, status-epilepticus, hypoxic-ischemic, intracranial bleed etc. are other main causes. Neurological outcome is often the foremost concern of parents and physicians.[3] Etiology of coma and clinical status at the time of presentation are the most likely predictors of outcome.[6] Simple clinical signs have been found as good predictors of outcome.[3,6]

METHODS

This prospective observational study was conducted at a tertiary care pediatric unit of the affiliated hospital of GMC Srinagar. The objective was to study the etiology of non-traumatic coma in children (aged between 6 months and 12 years) and to determine the clinical signs predictive of outcome in NTC in children. After approval from institutional ethics committee informed consent was obtained from the patients/parents/legal guardians.

Patients aged between 6 months and 12 years, admitted between August 2009 and August 2010 with coma were eligible for inclusion in the study. Exclusion criteria were:

1. Children aged <6 months or >12 years.
2. Coma of traumatic etiology.
3. Coma as part of an anticipated terminal illness.

Details of demographic data, history and examination findings were recorded in a predesigned proforma. Patients were re-examined at 48 hours to record clinical data and at discharge to record the outcome.

The clinical variables recorded were heart rate, respiratory rate and pattern, blood pressure (average of three recordings using mercury sphygmomanometer, by auscultatory method), temperature, coma severity (using modified Glasgow coma scale),[7-9] pupillary size and response to light, extra ocular movements and fundus picture. Etiology of coma was determined on the basis of history, clinical examination and relevant laboratory investigations. The investigations such as lumbar puncture, CT scan and metabolic work up were determined by clinical presentation and decided by the consultant in-charge. Etiology was classified into infections, toxic-metabolic, hypoxic-ischemic, post-status epilepticus, intracranial bleeding and miscellaneous. Bacterial meningitis was defined as acute febrile encephalopathy with identification of microorganisms from the CSF culture or presence of 3 or more of the following abnormalities in CSF:

1. Polymorphonuclear leucocytosis ≥100 cells/mm^3.
2. Glucose ~40 mg/dl or 50% of blood sugar.
3. Elevated proteins >40 mg/dl.
4. Micro-organisms seen by Gram staining.[3,10]

Diagnosis of tuberculous meningitis was made when there was family history of tuberculosis, positive tuberculin skin test, CSF analysis showing pleocytosis with leukocyte count 10-250/mm^3 (rarely >500/mm^3) with lymphocyte predominance and protein concentration 100-3000 mg/dl with decreased glucose usually <50 mg/dl and/or CT/MRI showing basilar enhancement or communicating hydrocephalus with signs of cerebral edema or early focal ischemia[10]. Encephalitis was defined as acute febrile encephalopathy with CSF pleocytosis with lymphocyte predominance (>5 cells/mm^3), slightly elevated protein (usually 50-200 mg/dl) and absence of bacteria on direct microscopy or culture and where no other alternative diagnosis was identifiable.[3,4] Focal seizures or focal findings on CT/MRI scans suggested HSV encephalitis.[10] Hypertensive encephalopathy was diagnosed when coma of acute onset was associated with blood pressure more than 95th percentile for age and sex, with or without retinal changes.[3,4] When there was a metabolic derangement commensurate with the clinical picture or toxic ingestion was confirmed, a label of toxic-metabolic coma was used[3]. Coma following hypoxic cerebral injury such as after cardio-respiratory compromise, shock, near-drowning or accidental or homicidal hangings was considered to be hypoxic-ischemic.[3,4] Children with coma with evidence of bleed on radio imaging of head were labeled as having intracranial bleed[3].

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.

Tachycardia: Heart rate above upper limit for that age.

Bradycardia: Heart rate less than sixty per minute.

Hypertension: Blood pressure more than 95th centile for age and sex.

Hypotension: Blood pressure below 5th centile for the age and sex.

Hyperthermia: Axillary temperature above 38°C.[3]

Hypothermia: Temperature below 35°C.[3]

Coma severity: Based on score obtained on the modified Glasgow coma scale.[7]

Pupils: (i) normal - both pupils equal in size, 2-3 mm in diameter and reactive to light, (ii) abnormal-pupils small (= 1 mm), or dilated (= 4 mm), unequal or non-reactive to light.[3]

Extra Ocular Movements (EOM): (i) normal - no impairment of movement in any direction, (ii) abnormal - if lateral, medial, upward, downward or all movements of eyeballs were absent.[3]

Corneal reflex: Absent or present.

Motor patterns were recorded as normal pattern or abnormal if there was diffuse decrease in tone (flaccidity), increase in tone (hypertonia) or decerbrate or decorticate posture.[3] Respiratory pattern was said to be abnormal if the breathing was central neurogenic hyperventilation, apneustic, ataxic or apnoeic.[3,11]

Outcome variables: Outcome was recorded as survived and died. Among those who survived it was further graded as normal, or those having mild, moderate or severe disability, defined as:

Normal: no motor deficit, ataxia, cranial nerve palsy, and functional level back to pre-illness state.[3]

Mild disability: minimal alterations of tone/deep tendon reflexes, isolated cranial nerve palsy and weakness of grade 4 or ataxia.[3]

Moderate disability: moderate weakness (grade 3) or ataxia, behaviour disturbance and multiple cranial nerve involvement.[3]

Severe disability: severe weakness (<grade 3) or ataxia and quadriplegia.[3,12]

Statistical analysis: Descriptive statistics (frequency, percentages) were calculated. The study variables were analyzed for their association with the outcome by applying the Chi-square test, Fisher's exact test and calculation of Odds ratio and Relative Risk (RR) with 95% Confidence Interval (95% CI). SPSS 10.0 and GraphPad Instat statistical packages were used.

RESULTS

Sixty one patients were included in the study, 34 (55.7%) were male and 27 (44.3%) were female. 33 (54.1%) were of age ≤3 years and 28 (45.9%) were >3 years of age. Etiologically out of 61 patients, 25 (41.0%) were caused by infection. Toxic-metabolic causes were found in 16 (26.2%), status-epilepticus was causative in 10 (16.4%) and structural CNS lesions in 5 (8.2%). No cause could be found in 5 (8.2%) patients. In infections, encephalitis was the largest sub-group responsible for 10 (16.4%) cases. Rest of the cases were caused by pyogenic meningitis 7 (11.5%), tubercular meningitis 4 (6.6%), herpes encephalitis 2 (3.3%) and septic shock and HIV encephalopathy 1 (1.6%) each.

Table 1: Etiology of coma, mortality and outcome of survivors.

Diagnosis	Total	Survived	Died	Disability				No follow up
				Normal	Mild	Moderate	Severe	
Total	61	39 (66.1%)	20 (33.9%)	19 (31.1%)	6 (9.8%)	7 (11.5%)	7 (11.5%)	2
CNS infections	25 (41.0%)	17 (70.8%)	7 (29.2%)	3	3	5	6	1
Encephalitis	10 (16.4%)	8	2 (20.0%)	3	3	1	1	
Pyogenic meningitis	7 (11.5%)	4	3 (42.9%)			2	2	
Tubercular meningitis	4 (6.6%)	3	1 (25.0%)			1	2	
Herpes encephalitis	2	2				1	1	
Septic shock	1		1 (100%)					
HIV encephalopathy	1							1
Toxic-metabolic	16 (26.2%)	9 (56.2%)	7 (43.8%)	**7**		**2**		
Poisoning	7 (11.5%)	5	2 (28.8%)	5				
Hepatic encephalopathy	4 (6.6%)		4 (100%)					
Hypoxic encephalopathy	3	2	1 (33.3%)	2				
Diabetic ketoacidosis	1	1				1		
Leigh's encephalitis	1	1				1		
Status epilepticus	10 (16.4%)	9 (90.0%)	1 (10.0%)	**7**	**2**			
Structural CNS lesions	5 (8.2%)	2 (50.0%)	2 (50.0%)	1			1	1
Intra-cranial hemorrhage	2 (3.3%)		2 (100%)					
CNS infarct	2	1					1	1
Hypertensive encephalopathy	1	1		1				
Unknown	5 (8.2%)	2 (40.0%)	3 (60.0%)	1	1			

Table 2: Clinical signs at admission and at 48 hours and their association with survival.

Variable	At admission					At 48 hours				
	Total	Survived	Died	Odds ratio (95 % CI)	P value	Total	Survived	Dead	Odds ratio (95% CI)	P value
Age										
≤3	32	17	15	3.88 (1.2-13)	0.029					
>3	27	22	5							
Sex										
Male	32	20	12	1.43 (0.48-4.25)	0.589					
Female	27	19	8							
Temperature										
Normal	29	21	8	1.75 (0.59-5.23)	0.412	36	33	3	4.40 (0.58-33.23)	0.180
Abnormal	30	18	12			7	5	2		
Hyperthermia	24	15	9	1.67 (0.28-10.10)	0.660	5	4	1	4.00 (0.12-137.08)	1.000
Hypothermia	6	3	3			2	1	1		
Heart rate										
Normal	28	20	8	1.58 (0.53-4.71)	0.582	38	36	3	8.00 (0.94-68.12)	0.091
Abnormal	31	19	12			5	3	2		
Tachycardia	27	18	9	6.00 (0.54-66.21)	0.272	4	3	1	7.00 (0.17-291.61)	0.400
Bradycardia	4	1	3			1	0	1		
Pulse volume										
Good	42	33	9	6.72 (1.95-23.19)	0.002	39	37	2	55.50 (3.83-804.59)	0.003
Poor	17	6	11			4	1	3		
Blood pressure										
Normal	39	29	10	2.90 (0.93-9.01)	0.084	28	34	4	2.13 (0.18-24.01)	0.479
Abnormal	20	10	10			5	4	1		
Hypertension	10	8	2	16.00 (1.79-143.2)	0.023	3	3	0	7.00 (0.17-291.61)	0.400
Hypotension	10	2	8			2	1	1		
Respiratory pattern										
Normal	46	34	12	4.53 (1.24-16.59)	0.024	40	37	3	24.67 (1.70-357.60)	0.032
Abnormal	13	5	8			3	1	2		
Pupils										
Normal	39	30	9	4.07 (1.29-12.92	0.020	36	33	3	8.25 (1.22-55.59)	0.045
Abnormal	20	9	11			7	4	3		
Corneal reflex										
Present	54	38	16	9.50 (0.98-91.81)	0.041	41	38	3	53.00 (2.18-1388.0)	0.010
Absent	5	1	4			2	0	2		
Extraocular movements										
Normal	52	37	15	6.17 (1.08-35.37)	0.038	38	36	2	27.00 (2.74-265.88)	0.008
Abnormal	7	2	5			5	2	3		
Papilledema										
Present	7	2	5	6.17 (1.08-35.37)	0.038	6	3	3	17.50 (2.05-149.23)	0.014
Absent	52	37	15			37	35	2		
Seizures										
Present	39	27	12	0.67 (0.22-2.05)	0.566	9	7	2	2.95 (0.41-21.14)	0.277
Absent	20	12	8			34	31	3		
Motor patter										
Normal	38	28	10	2.33 (0.77-7.06)	0.161	27	26	1	8.67 (0.87-86.11)	0.056
Abnormal	21	12	10			16	12	4		
Hypertonia	9	4	5	1.75 (0.31-10.03)	0.670	5	4	1	0.67 (0.05-8.64)	1.000
Hypotonia	12	7	5			11	8	3		
GCS										
≥11	0	0	0			26	25	1		
9-10	0	0	0			6	6	0		
7-8	35	28	7		0.016	4	4	0		
5-6	13	7	6			3	3	0		
3-4	11	4	7			4	0	4		

In our study, 39 (66.1%) survived and 20 (33.9%) died. Of the 39 who survived 19 (48.7%) were normal at discharge and 20 (51.3%) had varying degrees of disability. The mortality was 7 (29.2%) in CNS infections, 7 (43.8%) in toxic-metabolic coma, 1 (10%) in status-epilepticus, 2 (50%) in coma due to structural CNS lesions and 3 (60%) in patients in whom etiology of coma could not be determined. Individual mortality figures were 2 (20%) in encephalitis, 3 (42.9%) in pyogenic meningitis, 1 (25%) in TBM, 4 (100%) in hepatic encephalopathy, 2 (100%) in ICH and 2 (28.8%) in coma due to poisoning.

DISCUSSION

NTC in children represents an acute, life threatening emergency, requiring prompt intervention for preservation of life and brain function. This prospective observational study was undertaken to study the etiology and outcome of NTC in Kashmir and to fill in the voids left in earlier studies. Etiology and clinical status at presentation are the most likely predictors of outcome[3]. However, NTC is a non-specific sign with a wide potential for differential diagnosis[2] and considerable expertise, good clinical acumen and advanced laboratory support is needed to reach to a correct diagnosis and manage every case properly. Simple clinical signs are often good predictors of outcome.[3,6]

In this study, infection was the commonest cause of NTC accounting for 25 (41%) cases. The importance of infection as an etiology of non-traumatic coma is also supported by other studies.[3,5,12,13] The importance of infective etiologies in children is in sharp contrast to adult-hospital based series where degenerative and cerebrovascular pathologies predominate.[14] However the type of infection in children with NTC varies in different regions. Encephalitis contributed to 10 (16.4%), pyogenic meningitis to 7 (11.5%) and TBM to 4 (6.6%) cases. Toxic-metabolic causes formed the second major cause of non-traumatic coma responsible in 16 (26.2%) cases. Similar findings were reported by S Singhi et al.[3] and B Balaka et al[15], attributing 19 (19%) cases and 19 (15.2%) cases to toxic-metabolic causes respectively. However, in the Egyptian study[6], toxic-metabolic causes outnumbered infections and were causative in 33 (33%) cases. Status-epilepticus was another main etiologic group responsible in 10 (16.4%) cases in our study. The number of such patients was 10 (10%) in study of S Singhi[3], 44 (29.4%) in the Iranian study[12] and 88 (27%) in the study of Vijay Kumar et al.[13] The cause remained unknown in 5 (8.2%) patients in our study, in 9 (6%) in Iranian study[12] and in 14 % in the study of Wong CP et al.[2]

Mortality associated with NTC was 20 (33.9%) of our patients. The mortality was reported as 35 % in the study of S. Singhi et al.,[3] 60.5% in that of A. M. Ali et al.,[16] 50% in the Egyptian study[6] and 16.6% in the Iranian study.[12] However, mortality was considerably lower than that reported in adults; their mortality rates being 60% and neurological intact survival rates 10%.[17] The mortality was 7 (29.2%) in CNS infections, 7 (43.8%) in toxic-metabolic coma, 1 (10%) in status-epilepticus, 2 (50%) in coma due to structural CNS lesions and 3 (60%) in patients in whom etiology of coma could not be determined. Individual mortality figures were 2 (20%) in encephalitis, 3 (42.9%) in pyogenic meningitis, 1 (25%) in TBM, 4 (100%) in hepatic encephalopathy, 2 (100%) in ICH and 2 (28.8%) in coma due to poisoning. Though mortality was not significantly different when infections were compared with toxic-metabolic causes (p value 0.5), the odds of dying were twice in toxic-metabolic causes (Odds ratio 1.9, RR 1.5). The mortality was not significantly different among various infectious causes, however pyogenic meningitis patients presenting in coma had almost twice the risk of dying when compared with other infections (P value 0.37, RR 1.8). This was probably because all the patients who had pyogenic meningitis and died, were infants. The mortality was significantly higher in hepatic encephalopathy and ICH. These results were comparable with other studies[3]. Survival was significantly better in patients with CNS infection (63%) as compared to those with toxic-metabolic causes (27%) and intracranial bleed (43%, P <0.05) in the study of S. Singhi et al.[3] However, B. Balaka et al.[15] and F. Khodapanahandeh et al.[12] reported a worse outcome with infections.

Two patients were lost to follow-up and of the remaining 39 (66.1%) that survived, 19 (31.1%) were normal at discharge, 6 (9.8%) had mild, 7 (11.5%) moderate and 7 (11.5%) had severe disability. In the study of S. Singhi et al.,[6] of the 65 (65%) children that survived, 11 (11%) were normal, 14 (14%) had mild disability, 21 (21%) had moderate disability and 14 (14%) were severely disabled and dependent. In the Iranian study[12], 25 (16.6%) patients died and 125 (83.3%) survived. Of the 125 survivors, 82 (54.7%) were discharged with normal neurological examination, 9 (6%) had mild disability, 18 (12%) were moderately disabled and 16 (10.7%) were severely disabled. The incidence of disability among survivors was higher in CNS infections compared to toxic-metabolic group and this matched the results of S Singhi et al.[3]

On bivariate analysis, age ≤3, poor pulse volume , hypotension, abnormal respiratory pattern, abnormal pupils, absent corneal reflex, abnormal extra-ocular movements, lower modified GCS and papilledema correlated significantly with mortality at admission. 5 deaths occurred after 48 hours. At 48 hours, poor pulse volume, abnormal respiratory pattern, abnormal pupils, absent corneal reflex, abnormal extra-ocular movements and papilledema had a significant correlation with mortality.

Mortality rate among children under 3 years was significantly higher. 46.9% children ≤3 died against 18.5% of children >3 (Odds ratio 3.88, P value 0.03). Similar observations were made by S. Singhi et al.[3] The

outcome of coma was not associated with gender (Odds ratio 1.43, P value 0.59). Seshia and Seshia[18] and S Singhi et al.[3] also did not observe any significant difference in the incidence of coma between the two sexes. Earlier studies had shown a greater mortality in male (42%) compared to female children (20%).[19]

Modified GCS recorded at admission had significant association with outcome; mortality rates progressively increased with decreasing GCS (P value 0.016). S. Singhi et al[3] and P. C. Nayana Prabha et al.[4] also found same relation. Studies in both traumatic and non-traumatic coma have indicated that mortality is high when the GCS is less than 8.[20,21] A. M. Ali et al.[16] found a mildly significant relation between GCS and mortality in non-traumatic coma, however morbidity was higher in patients with lower GCS.

There was no significant correlation between mortality and abnormal temperature at admission (Odds ratio 1.75, P value 0.41) or at 48 hours (Odds ratio 4.4, P value 0.18). In previous studies hypothermia in patients spelled a bad prognosis, however the number in previous studies[3,19] and also in our study was too small to make any meaningful conclusion.

No significant association between mortality and abnormal heart rate was found. Bradycardia had a very high association with mortality in some studies;[3] six of seven patients with bradycardia at admission died in study of S. Singhi et al.[3] Tachycardia was also associated with increased risk of death and poor neurological outcome when compared with a normal heart rate in their study. In our study, there was no significant difference between patients with tachycardia and bradycardia at admission (P value 0.27), however the odds ratio of 6.0 meant a higher risk of death in patients with bradycardia. These findings were reproduced at 48 hours (Odds ratio 7.0, P value 0.4), however the numbers were too small for any significant conclusion.

Poor pulse volume was a significant predictor of mortality at admission (Odds ratio 6.7, P value 0.002) and also at 48 hours (Odds ratio 55.5, P value 0.003) in our study. 64.7% patients with poor pulse volume at admission and 75% of such patients at 48 hours died against the mortality figures of 21.4% at admission and 5.1% at 48 hours in patients with good pulse volume. This was in accordance with findings of S. Singhi et al.[3]

Hypotension was a poor prognostic sign; 80% of 10 children who were hypotensive at admission died against 20% of 10 with hypertension (Odds ratio 16.0, P value 0.023). These figures were 67% and 24% in the study of S. Singhi et al.[3] with a p value of 0.02. This is similar to the study by Johnston and Seshia,[19] wherein 14 of 15 hypotensive children died. However, similar correlation could not be reproduced at 48 hours. Hypertension at admission or at 48 hours was not associated with increased mortality. Various abnormalities in respiratory pattern may be seen in coma depending on the region of brain involved. It is therefore expected that an abnormal breathing pattern would predict a poorer outcome. The prognosis in our study was best with a normal respiratory pattern while abnormal breathing at admission had highest risk of mortality (Odds ratio 4.53, P value 0.02). This is similar to a study from Canada[19] and that of S Singhi et al.[3] These findings were reproduced at 48 hours (Odds ratio 24.7, P value 0.03).

Pupillary signs were also good predictors of survival and neurologic outcome. Abnormal pupils at admission as well as at 48 hours were strong predictors of a fatal outcome. In the study by Seshia et al.,[18] 68% of children with fixed dilated pupils for more than 2 hour died. Similar observations were made by Ogunmekan et al.[5] in a large retrospective study from Nigeria and also by S Singhi et al.[3] Of 39 patients with normal pupils at admission, 9 (23.1%) died and of 20 with abnormal pupils 11 (55%) died (Odds ratio 4.1, P value 0.02). At 48 hours, out of 36 patients with normal pupils, 3 (8.3%) died and of 7 with abnormal pupils 3 (42.9%) died (Odds ratio 8.3, P value 0.04).

Presence of the corneal reflex indicates functional interconnections in the pons. Absent corneal reflexes in children with deep coma prognosticated a poor outcome in our study; 80% patients with absent corneal reflex and 29.6% of others died (Odds ratio 9.5, P value 0.04). These figures were 100% and 7.3% at 48 hours (Odds ratio 53, P value 0.01), respectively. These findings are similar to those of S. Singhi et al.[3]

Presence of doll's eye movements suggests intact 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 therefore, generally indicates asymmetrical brain stem lesion in mid brain or pons while complete absence of doll's eye movements suggests bilateral structural brainstem abnormality or severe metabolic-toxic encephalopathies. In the present study, 28.8% of children, with preserved EOM at presentation, died whereas, 71.4% of children with absent EOM died (Odds ratio 6.2, P value 0.04). At 48 hours these figures were 5.3% and 60% (Odds ratio 27.0, P value 0.008). This goes with the earlier studies that suggest absent or impaired EOM as a sensitive index for prognosticating the outcome. Preserved EOM have been associated with favourable outcome in previous studies.[5,14] Seshia et al.[19] 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, abnormality of motor pattern was not predictive of higher risk of death (Odds ratio 2.3, P value 0.16). Same findings were reproduced at 48 hours (Odds ratio 8.67, P value 0.06). However, other studies[3,18] reported a significant association of abnormal motor

pattern with mortality. In our study, the mortality figures were 26.3% and 3.7% at admission and 48 hours, respectively in patients with normal motor pattern and 47.6% and 25%, respectively in patients with abnormal motor pattern. Though not significant, the risk of dying was more in patients with abnormal tone. There was no difference in mortality between patients with hypertonia or hypotonia.

Papilledema was significantly associated with increased mortality both at admission (Odds ratio 6.17, P value 0.04) and at 48 hours (Odds ratio 17.5, P value 0.01). This association is in coherence with the study by S. Singhi et al.[3]

In our patients, seizures were recorded in 72% at some time of their hospital stay which agrees with the figure of 68% given by Ogunmekan et al.[5] in their study. However, presence of seizures did not influence the outcome as was seen by S. Singhi et al.[3] in their study.

CONCLUSION

This study shows that clinical signs such as poor pulse volume, absent corneal reflex or abnormal respiratory pattern were strong predictors of death following severe non-traumatic coma. The study also reaffirms that clinical variables and GCS score remain the most readily available tools for assessment of non-traumatic coma, to identify those who are likely to die and those having the greatest potential for recovery. This is particularly helpful in resource-limited countries for directing the limited resources for maximal benefit.

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