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Original Research Article

Study of clinical profile, etiology and immediate outcome in Non-traumatic coma in children

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Abstract: Etiology of coma and clinical status at the time of presentation are likely predictors of outcome in cases of non-traumatic coma. The present study included of clinical profile, etiology of non-traumatic coma and immediate outcome in terms of survival and death in pediatric patients.This was prospective hospital based cohort study undertaken over a period of one year. A detailed history of all patients were taken and entered in case proforma. A complete clinical examination of patients was done. The etiology of coma was determined and the diagnosis of tuberculous meningitis was done. The data was analyzed using statistical software SPSS version 16. The clinical data was analyzed for the etiology of non-traumatic coma.Thirty one children survived and 19 died with mortality rate of 38%. Among survivors 20 (64%) were normal, 11(36%) had some disability (23%) mild disability, 10% moderately and 3% severely disabled. From the study, we got infections as the important cause of non-traumatic coma.
KeywordsNon traumatic coma, Pediatric patients, Intensive care

INTRODUCTION:

Coma is a relatively common condition in the pediatric intensive care unit, mainly divided into traumatic and non-traumatic coma. Non-traumatic coma in childhood is an important pediatric emergency accounting for. Etiology of coma and clinical status at the time of presentation are likely predictors of outcome [1]. Coma refers to a state in which the patient is unable to arouse or respond to noxious stimuli and is completely unaware of self and surroundings [2].

A very little information is available regarding non traumatic coma particularly from developing countries including India. In the Indian studies by Bansal *et al.;* in 2005, Buch *et al.;* in 2011, Gowda *et al.;* in 2014 the etiology of non-traumatic coma was mainly CNS infections [1, 3, 4].

A better understanding of causes and outcome is essential to help to plan rational management of non-traumatic coma. Therefore this study aims at learning the clinical profile, etiology of non-traumatic coma and immediate outcome in terms of survival and death.

METHODS:

This was prospective hospital based cohort study undertaken over a period of one year from May 2013 to April 2014 in the pediatric intensive care unit of a Tertiary care teaching Hospital in Mumbai. Institutional Ethics committee approval was taken prior to starting the study. Informed written consent was obtained from parents /guardian of the subjects prior to enrollment of subjects. Total 50 consecutive children between 1 month to 12 years of age, presenting with acute non traumatic coma excluding those known to have neurological abnormality prior to admission were included.

A detailed history of all patients were taken and entered in case proforma. A complete clinical examination of patients at the time of admission including vitals, general examination, and an anthropometry with WHO classification for malnutrition and systemic examination with particular emphasis on central nervous system was done.

The clinical variables recorded were vitals including heart rate, respiratory rate and pattern, blood pressure (average of three recordings using NIBP), Temperature. The neurological assessment was done

using James's Modified Glasgow coma scale [6], brain stem responses (pupillary size and response to light, posture, response to pain, oculocephalic reflex), Motor pattern (recorded by subjectively assessing the passive tone), Extra ocular movements, Corneal reflex, Seizures if any, type of seizures, Involuntary movements and Fundus examination. The investigation done in all patient were complete blood count, random blood sugar, liver function test, renal function test, serum electrolyte, blood gases analysis, HIV by ELISA.

The other investigationsdone were X-Rays, computerized tomography brain, magnetic resonance imaging brain, magnetic resonance angiography, electroencephalogram, blood ammonia, coagulative profile, CSF examination etc. The etiology of coma was determined on the basis of history, clinical examination and relevant laboratory investigations and imaging studies. Bacterial meningitis is diagnosed as acute febrile encephalitis with identification of microorganisms from the CSF culture or presence of three or more of the following abnormalities-

1) Polymorpho neuclear leukocytosis>100 cells/mm^3.
2) Glucose -40mg/dl or 50%of blood sugar.
3) Elevated proteins>40mg/dl
4) Microorganisms seen by gram staining.

Diagnosis of tuberculous meningitis was based on the criteria by Ahuja et al. Encephalitis was defined as acute febrile encephalitis with CSF leukocytosis with lymphocyte predominance (>5cells/cm3) and absence of bacteria on direct microscopy , culture , MRI brain findings where no other diagnosis was identifiable. Hypertensive encephalopathy was diagnosed on the basis of acute coma in the presence of blood pressure more than 95^{th} percentile for age and sex with or without retinal changes. Coma following hypoxic anoxic injury to brain following cardiorespiratory compromise or shock was considered to be hypoxic-ischemic. Those with metabolic derangements with clinical picture of acute coma were labeled as toxic-metabolic.

Demographic data and clinical data was recorded at the time of admission, Patients were reassessed at 24, 48 hours and at discharge to record the clinical data. They were further assessed at discharge to record the immediate outcome.ie survival with or without disability or death. Definition of Study variables were given in appendix 1.

The data was analyzed using statistical software SPSS version 16. The clinical data was analyzed for the etiology of non-traumatic coma. The clinical sign were further analyzed for prediction of outcome in the form of survival and death at admission, 24 hours, and 48 hours and at discharge. For qualitative data Pearson's chi square test and logistic regression analysis was used for test of significance.

Definition of Study variables were as follows,

Tachycardia: Heart rate above the upper limit for that age.

Bradycardia: Heart rate less than 60 per minute (as per age).

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^oC.

Hypothermia: Temperature below 35^oC.

Coma severity: Based on score obtained on the modified Glasgow coma scale [5].

Extra ocular movements (EOM):

(i) Normal - no impairment movement in any direction,
(ii) Abnormal - if lateral, medial, upward, downward or all movements of eyeballs were absent.

Corneal reflex: absent or present.

Outcome variable Defined as:

1. **No disability**: no motor deficit, ataxia, cranial nerve palsy, and functional level back to pre-illness state.
2. **Mild disability**: minimal alterations of tone/deep tendon reflexes, isolated cranial nerve palsy and motor weakness of muscle power grade 4 or ataxia.
3. **Moderate disability**: moderate motor weakness of muscle power grade 3 or ataxia, behavior disturbance and multiple cranial nerve involvement.
4. **Severe disability**: severe motor weakness of muscle power grade < 3 or ataxia and quadriplegia.

Etiology were defined as follows[6],

Pyogenic meningitis:

Acute febrile encephalopathy with culture of compatible microorganisms from the CSF or the presence of 2 or more of the following abnormalities in CSF –

(i) Polymorpho nucler leucocytosis
(ii) Glucose ~ 40 mg/dL or 50% of blood sugar
(iii) Micro-organisms seen by gram staining

Tuberculous meningitis*:*

Based on the criteria by Ahuja *et al.;* [7]

Viral meningoencephalitis:

Acute febrile encephalopathy along with CSF pleocytosis, with lymphocyte predominance (>5 cells/mm3) and absence of bacteria on direct microscopy culture; with no other alternative diagnosis identifiable

Enteric encephalopathy:

Encephalopathy with definitive investigational evidence of infection with Salmonella or Shigella (positive blood or stool culture for Salmonella or Shigella or positive serology for Salmonella)

Hypertensive encephalopathy:

Acute coma in association with blood pressure more than 95th percentile for age and sex with or without retinal changes

Hypoxic-ischemic encephalopathy:

Encephalopathy following hypoxic cerebral injury such as near drowning, accidental or homicidal hanging

Toxic encephalopathy:

Encephalopathy following ingestion of toxin containing substances (neem oil, kerosene, organophosphorus, etc)

Hepatic encephalopathy:

Spectrum of potentially reversible neuropsychiatric abnormalities seen in patients with liver dysfunction after exclusion of unrelated neurologic and metabolic abnormality

Dyselectrolytemia:

Acute afebrile encephalopathy with abnormal serum electrolyte levels (sodium) with normal CSF examination with no other alternative diagnosis identifiable

RESULTS

A total 50 comatose children (23 boys, 27girls) were included in the study, which fulfilled the inclusion criteria. Ten children were below the age of 1 year, 24 were between 1-5 years and 16 were above 5 years. Maximum children 24 (48%) were in 1 to 5 year age group with mean age of 4.2 years.

Central nervous system (CNS) infections accounted for (n=32) 64% of cases (Table1). Amongst non-infectious causes (n=18) hepatic coma constituted five (27.8%) leads followed by Diabetes ketoacidosis (2) and intracranial bleed (2) each (11.1%). Other causes were autoimmune encephalitis, brain tumour, inborn error of metabolism, snake bite, poisoning due to unknown substance consumption, electrolyte imbalance, status epilepticus, sagittal vein thrombosis and acute myeloid leukaemia (AML). Table 1 shows etiology of coma and neurological outcome in relation to etiology among survivors.

Table 1: Etiology of coma in the study population and neurological outcome in relation to etiology among the survivor

Diagnosis	**Frequency (n=50)**	**No. Died (n=19)**	**No. Survived (n=31)**	**Disability**			
				Normal	Mild	Moderate	Severe
Infectious Etiology	**32**	**13 (40.62%)**	**19(59.37%)**	**11**	**5**	**2**	**1**
Viral Encephalitis	10	5	5	1	3	1	0
Tubercular Meningitis	9	4	5	2	2	1	0
Pyogenic Meningitis	8	2	6	5	0	0	1
Sepsis	2	1	1	1	0	0	0
Cerebral Malaria	1	0	1	1	0	0	0
Dengue shock syndrome	1	0	1	1	0	0	0
Encephalopathy	1	1	0	0	0	0	0
Non-infectious etiology	**18**	**6 (33.33%)**	**12(66.67%)**	**9**	**2**	**1**	**0**
Hepatic coma	5	3	2	2	0	0	0
Diabetes Keto Acidosis	2	0	2	2	0	0	0
Intracranial Bleed	2	1	1	1	0	0	0
AML	1	0	1	0	1	0	0
Autoimmune encephalitis	1	0	1	0	0	1	0
Brain Tumor	1	0	1	0	1	0	0
IEM	1	1	0	0	0	0	0
Snake bite	1	0	1	1	0	0	0
Status Epilepticus	1	0	1	1	0	0	0
Electrolyte imbalance	1	1	0	0	0	0	0
Poisoning	1	0	1	1	0	0	0
Sagital Vein Thrombosis	1	0	1	1	0	0	0

Outcome:

Thirty one children survived and 19 died with mortality rate of 38%. Among survivors 20 (64%) were normal, 11(36%) had some disability. (23%)mild disability, 10% moderately and 3% severely disabled).Mortality rate was 6/10 (60%) amongst infants, 6/24 (25%) amongst 1-5 years and 7/16 (43%) among children >6 years-12years. Out of 31 survivors 20 had no disability and only 4 children had moderate to severe disability whereas 7 had mild disability. Nobody in infant group had moderate to severe

disability whereas 3 children in 1-5 years and one child in age >5years had moderate to severe disability.

Mortality among girls was 10/27 (37.03%) whereas in boys 9/23 (39.1%) which was not statistically significant. Mortality was highest in viral meningitis and tuberculous meningitis than other CNS infections but not statistically significant. Mortality was highest in hepatic coma than other causes in non-infectious group which was statistically significant.3/6 (50%). Table 2 shows clinical signs at admission, at 24 hours and 48 hours and their association with survival.

Table 2: Clinical signs at Admission and at 48 Hours and Their Association with Survival

variables	**At admission**					**At 48 hours**				
	Total	Survived	Died	Odd's ratio	'p' value	Total	Survived	Died	Odd's ratio	'p' value
Age										
<3 years	22	13	9		0.774	21	18	3		0.001
>3 years	28	18	10			25	23	2		
Gender										
Female	27	17	10		1	28	26	2		0.001
Male	23	14	9			27	1	26		
Temperature										
Normal	8	7	1		0.134	16	5	11		0.004
Hyperthermia	42	24	18			34	26	8		
Heart rate										
Normal	9	8	1		0.127	26	23	3		<0.001
Abnormal	41	23	18			24	8	16		
Pulse volume										
Good	35	21	14		0.757	41	30	11		0.001
Poor	15	10	5			9	1	8		
Blood pressure										
Normal	34	25	9		0.005	34	28	6		<0.001
Abnormal	16	6	10			26	3	13		
Pupils										
Normal	29	16	13		0.376	27	26	1		<0.0001
Abnormal	21	15	6			23	5	18		
Deep tendon reflexes										
Normal	6	5	1		0.428	22	21	1		<0.001
Abnormal	44	26	18			28	10	18		
Plantar reflexes										
Flexion	30	21	9		0.235	20	11	9		0.553
Extension	20	10	10			30	20	10		
Neck rigidity										
Present	21	13	8		1	7	3	4		0.404
Absent	29	18	11			43	28	15		
Papilloedema										
Present	1	0	1		0.38	2	1	1		1
Absent	49	31	18			48	30	18		
Cranial nerve palsy										
Present	11	7	4		1	8	4	4		0.459
Absent	39	24	15			42	27	15		
GCS										
≤ 5	2	1	1		1	19	2	17		<0.001
>5	48	30	18			31	29	2		
EOM										
Normal	41	25	16		0.614	37	26	11		0.093
Abnormal	9	6	3			13	5	8		
Motor pattern										
Normal	20	11	9		0.66	26	24	2		<0.001
Abnormal	30	20	10			24	7	17		
Power										
≤2	2	1	1		0.32	13	1	12		<0.001
>2	48	30	18			37	30	7		

Predictors of outcome at admission:

On bivariate analysis only abnormal blood pressure correlated significantly with mortality at admission whereas age, gender, modified GCS<5, temperature, poor pulse volume, bradycardia, abnormal respiratory pattern and apnoea, nonreactive pupil, deep tendon reflexes, neck rigidity, cranial nerve palsy, motor patterns did not show significant association.10/16 children with abnormal blood pressure died as compared to 9/34 who had normal blood pressure (p=0.005).

Predictors of outcome at 24 hours:

The significant predictors of death at 24 hours of admission were abnormal heart rate and abnormal blood pressure amongst vital parameters (p=0.004), (p=0.001) respectively. On neurological evaluation abnormal reflexes (0.002), abnormal pupil (0.008), MGCS score <5(0.04), abnormal motor pattern (0.009), and power grade <=2 w (0.03) were found to be significantly associated with fatality with p values <0.005.

Predictors of outcome at 48 hours:

The significant predictors of death after 48 hours stay in hospital on bivariate analysis were similar to the predictors at 24 hours except poor pulse pressure. These were abnormal heart rate and abnormal blood pressure amongst vital parameters (p=0.001), (p=0.0001) respectively. On neurological evaluation abnormal reflexes (0.001), abnormal pupils (0.001), MGCS score <5(0.001), abnormal motor pattern (0.001) and power grade <=2(0.001) were found to be significantly associated with fatality with p values = /<0.001.

In addition poor pulse volume at 48 hours (0.001) was significant variable in predicting fatality as compared to normal volume pulse. The assessment of GCS at 24 hours and 48 hours was confounded by various factors such as sedation for ventilation and use of anticonvulsants.

On logistic regression we did not get any significant individual parameter predicting fatality alone though GCS<5 was (0.07) somewhat close. Table 3 shows factors predicting fatal outcome at 48 hours on logistic regression.

Table 4 shows comparison of causes and mortality of Non-traumatic Coma in children among various studies.

Table 3: Predictors of fatal outcome based on clinical parameters at 48 hours

Variable	Odds Ratio	95.0% C.I. for EXP(B)		' p ' value
		Lower	Upper	
Hyperthermia	1.1	0.04	35.4	0.94
Tachycardia	5.1	0.15	167.0	0.36
Pulse Volume (Poor)	406712695.1	0.00	.	1.00
Hypertension & Hypotension	0.8	0.02	29.2	0.93
Abnormal Pupils	996713471.6	0.00	.	1.00
Abnormal Reflexes	3.7	0.04	362.4	0.57
GCS<5	21.5	0.78	595.3	0.07

Table 4: Comparison of Causes and Mortality of Acute non traumatic coma in childhood reported by various authors

Studies **Causes**	**Our Study (n=50) (India)**	**Gowada *et al.*[4] (n=104) (India)**	**Buch *et al.* [3] (n=94) (India)**	**Bansal *et al.*[1] (n=100) (India)**	**Ahmed *et al.*[10] (n=111) (Pakistan)**	**Sofiah *et al.* [9] (n=116) (Malaysia)**	**Vijaykumar *et al.*[8] (n=328) (UK)**	**Ibekwe *et al.*[11] (n=40) (Nigeria)**
CNS infection	32(64%)	68(65.3%)	34(36.1%)	60(60%)	59(53.1%)	80(69%)	164(50%)	28(70%)
Septicemia	1(2%)	-	34(36.1%)	-	5(4.5%)	-	-	4(10%)
Toxic & Metabolic	9(18%)	21(20.1%)	-	15(15%)	9(8.1%)	15(13%)	39(12%)	1(2.5%)
Status Epilepticus	1(2%)	6(5.77%)	-	10(10%)	5(4.5%)	-	88(27%)	2(5%)
Hypoxia	-	4(3.88%)	-	4(4%)	-	6(5%)	13(4%)	-
IC Bleed	2(4%)	-	-	7(7%)	-	4(3%)	-	-
Poisoning	1(2%)	1(0.96%)	-	-	5(4.5%)	-	-	2(5%)
Snake Envenomation	1(2%)	1(0.96%)	-	-	-	-	-	1(2.5%)
Respiratory tract infection	-	-	26(27.6%)	-	-	-	-	-

DISCUSSION:

Acute non-traumatic in pediatric intensive care is a significant condition accounting for an estimated 10-15% of all hospital admissions and major cause of morbidity and mortality. 73 Despite of its prevalence and associated morbidity and mortality a very little is known about paediatric coma and studies related to it. These studies are mostly retrospective stating the causes of non-traumatic coma which can differ from place to place. This prospective study helps to analyse the etiology and predictive values of various monitoring parameters in developing country like India.

The monitoring parameters studied were vital signs such as temperature, ,heart rate , blood pressure, pulse volume and neurological signs that helped in assessing the cerebral and brainstem functions and its integrity. The damage to the brain and its extent can be assessed by the vitals and neurological signs at the onset of coma and within 24-48 hours and are helpful in predicting the mortality and morbidity. The first 24-48 hours are critical for such patients. Hence the clinical signs which reflect extent and severity of cerebral hemisphere and brain stem were studied.

TheModified GCS is helpful in assessing coma severity, integrity of cerebral hemisphers in children. Brain stem function is assessed using brainstem reflexes such as eye movements, pupillary responses, and corneal responses. Breathing pattern and abnormal tone of limbs posturing indicates the severity of damage to the brain as whole [5].

From the study, we got CNS infections as the important cause of non-traumatic coma. There are other studies which also showed infection as the prominent cause of coma. In our study 24 out of 50 patients (48%) were in age group of 1 – 5 years with mean age of 4.2 years. Similar findings were seen in study by Bansal et al (59%), Gowada *et al.;* (48%), and Ibekwe *et al.;*(62.5%) [1, 4, 8]. A study by Ahmed *et al.;* and Sofiah *et al.;*had maximum infant [9, 10].

In our study, females formed 54% of the total study population so showing not much gender bias. Female predominance was seen in study by Ibekwe et al in Nigeria [77]. But majority of other studies done in Non-traumatic coma Bansal *et al.;* Gowada *et al.;* Ahmed*et al.;* and Sofiah *et al.;*found male. These findings were similar to studies done by Gowada *et al.;* Buch *et al.;* Bansal *et al.;* Ahmed *et al.;* [1, 3, 4, 10]. It is also supported by other studies wherein infection of CNS was found to be leading cause of non-traumatic coma [8,9,11,12].

In our study, the majority of infectious cause of non-traumatic coma comprised of Viral encephalitis (n=10), Tubercular meningitis (n=9) and pyomeningitis (n=8). Other studies like Gowada *et al.;* and Ahmed *et al.;* found Pyomeningitis as a most common cause of CNS infection of non-traumatic coma [4, 10]. But in study by Bansal *et al.;* tubercular meningitis was the leading cause of CNS infection [1].

However, the type of infection varies in different regions. Cerebral malaria was common in Africa [12], whereas dengue haemorrhagic fever was an important cause of coma in South-East Asia [9], while septicaemia was prominent in Saudia Arabia [13]. The importance of infective etiologies in children is in sharp contrast to adult-hospital based series where degenerative and cerebrovascular pathologies predominate [14]. Despite these small differences which may be attributed to sampling, it can be inferred that infective pathologies remain the prime reason for comatose pediatric patients in these countries as compared to western countries where metabolic reasons for coma are more common [14]. Among the non-infectious causes, toxic metabolic causes are commonest and were comparable in frequency with other studies [1,9,15,16].

The overall mortality in our study was 38%, which is comparable with other studies as in Bansal *et al.;* (35%)[1] and Sofiah *et al.*[9]*;* 35.7% was slightly higher as compared to other pediatric hospital based series (26.7%) from Nigeria and Canada [15, 16]. This was possibly due to reaching in late stage of disease to the hospital. However, mortality was considerably lower than that reported in adults; the adult mortality rates being 60% and neurological intact survival rates 10% [12].

Among survivors of nom-traumatic coma in our study, 35% had a disability. Viral encephalitis remain the leading cause of disability (36.3%), is comparable to Ahmed *et al.;*who got 41% disability [10]. Within the survivors, no statistically significant association between the etiology of coma and probability of disability could be established because of small size population.

In our Study, Mortality was more in male children compared to females, which is similar finding in Gowada *et al.*[4]*;* however it is not significant statistically. Earlier studies[17] had shown a greater mortality in male (42%) compared to female children (20%).

It is believed that prognosis in coma depends on its severity but there is rather inconclusive data on the use of GCS score and its predictive value in pediatric non-traumatic coma. In our study the modified GCS recorded at 48 hours had significant association with outcome ($p < 0.001$); mortality rates progressively increased with decreasing GCS.

In our study Hyperthermia at admission or at 48 hours not associated with higher risk of mortality. In our study no patient had hypothermia, which is believed to be indicator of poor outcome. In the study by Johnson and Seshia, all the hypothermic children died [17]. Hypothermia, regardless of etiology causes diminished cerebral metabolism and very low temperature may result in an isoelectric electroencephalogram.

While studying signs of circulatory instability we found that poor pulse volume at 48 hours predicted 89% death. Abnormal heart rate also had high association with mortality (66%). Among the survivors all had mild to moderate grade disability. Bansal *et al.;* also found significant relation between poor pulse volume, abnormal heart rate with mortality [1]. Hypotension at admission and at 48 hours also indicate fatal outcome in our study (87.5%). These findings correlate with finding of Bansal *et al.;* and study by Johnston and Seshia [1, 17]. We also found that mortality was higher in Hypertensive than normotensive patient, similar finding also got to Ahmed *et al* which is in contradictory to findings of Bansal *et al.;*[1, 10], Bansal *et al.;* found similar risk of mortality and neurodeficit in both Hypertensive and Normotensive patient.

On evaluating specific neurological sign for outcome, in our study abnormal pupillary reaction was strong predictive of fatal outcome (78.23%), and patient with abnormal pupillary reaction who survived, 60% had disability at the time of discharge. Similar results were seen in Bansal *et al.;* and Seshia *et al.;*[1, 15] Ogunmekan made similar observations in a large retrospective study from Nigeria [16].

In our study while assessing muscle tone we found out muscle flaccidity resulted significant mortality (77.77%) which is also supported by Ahmed *et al.;*[10], Bansal *et al.;* and Seshia *et al.;* who observed that 84% of those with a normal motor pattern had a normal outcome whereas 82% of those who were flaccid died [15]. Patient with Power grade < 3 shows high mortality (64%) in comparison to power grade >3, who all survived.

In our study abnormal deep tendon reflexes were associated with fatal outcome 3.7 times than normal DTRs. This is similar to study by Gowda *et al.;* they found deep tendon reflexes were associated with fatal outcome 1.7 times then the normal deep tendon reflexes [4]. This may be due to involvement of pyramidal tract and mid brain by metabolic and infective causes.

Presence of the corneal reflex indicates functional interconnections in the pons. Absent corneal reflexes in children with deep coma prognosticated a poor outcome. 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 our study, 61.5% of children with abnormal extra ocular movement died, and 80% had disability amongst survivors. This goes with the earlier studies that suggest absent or impaired EOM as a sensitive index for prognosticating the outcome. In study by Bansal *et al.;* 54% of children with absent EOM died [1]. Seshia *et al.;* observed that all those with absent EOM died and needed assisted ventilation [15].

CONCLUSION:

In our study we could not find significant relation of respiratory pattern, corneal reflex, pupillary size, papilloedema predicting outcome, which were seen in other studies. Factor such as papilloedema, Cranial nerve palsies, and abnormal plantar responses did not correlate with poor outcome in Ahmed *et al.;* this may be due to small sample size[10].

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