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Original Research Article

CLINICAL PROFILE, ETIOLOGY, AND OUTCOME OF NON-TRAUMATIC ALTERED MENTAL STATUS -A PROSPECTIVE OBSERVATIONAL STUDY IN A TERTIARY CARE CENTRE IN CHENNAI

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Abstract

Background: Altered mental status" (AMS) comprises a group of clinical symptoms rather than a specific diagnosis, and includes cognitive disorders, attention disorders, arousal disorders, and decreased level of consciousness.[1] Drugs (both illegal and prescribed), infections, metabolic disarray, trauma, neoplasms, stroke, convulsions, and a variety of organ system dysfunctions can all contribute to acute mental status changes in emergency department patients.[2] Coma is frequently a transitory state that can lead to the recovery of consciousness, the vegetative state, the state of limited consciousness, or even brain death. Patients were grouped based on the severity of their AMS symptoms, which ranged from complete loss of consciousness to intense excitement. The patients were classed as follows: 1) Inhibition of the central nervous system, which can manifest as coma, drowsiness, and disorientation; 2) stimulation of the central nervous system, which can manifest as irritability and aggressiveness; and 3) deviant conduct. We conducted this study to determine the etiology, clinical profile and outcome of patients with non-traumatic coma in a tertiary care centre. GCS score, Pupil reactivity score, GCS P score (GCS- Pupil reactivity score), and SIRS score were used to categorize the patients. The most prevalent cause of non-traumatic coma, according to our data, is cerebrovascular accident(specifically CVT followed by SAH) and metabolic causes(most common of which is hypoxia), toxins(which included drugs, OPC poisoning, snake bite), and infections.[3]

Keywords: Coma, Clinical profile, Sepsis, CVA

Introduction

The evaluation and management of altered mental state cover a wide range of topics and require a thorough history as well as a physical examination in order to rule out potentially fatal conditions. Alterations in one's state of consciousness can be broken down into three categories: alterations in arousal, alterations in the content of consciousness, or a mix of the two. Arousal can be described as hypoactivity or hyperactivity, and it also involves

ISSN: 0975-3583,0976-2833 VOL14, ISSUE 01, 2023

wakefulness and/or alertness. Changes in the content of consciousness can lead to changes in self-awareness, expression, language, and emotions. [4,5]

Alterations in mental status can be classified as delirium, which is an acute change in arousal and content; depression, which is a chronic change in arousal; dementia, which is a chronic change in arousal and content; and coma, which is a state of complete unresponsiveness (dysfunction of arousal and content).[6]

The differential diagnosis includes: primary intracranial diseases, systemic diseases that affect the central nervous system (CNS), exogenous poisons, and drug withdrawal. In infants and children, the most prevalent causes of mental status changes include infections, injuries, metabolic changes, and the consumption of hazardous substances. .[7,8] The most common causes of changed mental status in young adults are the consumption of hazardous substances or the effects of trauma. Alterations in mental state in the elderly are most frequently brought on by a stroke, infection, drug-drug interactions, or changes in the living environment. When it comes to senior people, anywhere from 10 to 25 percent of hospitalised patients will already be suffering from delirium .[9,10]

Objective

To study the Clinical profile, Etiology, and outcome of non-traumatic altered mental status in a tertiary care centre in Chennai.

Methodology

After obtaining Institutional Ethical committee approval, this prospective observational study was conducted among 130 patients admitted in the Emergency department and medical wards in Government Stanley Medical College and Hospital during the period of 6 months from August 2021 to January 2022.

The information regarding the study was explained to the patients. Written and informed consent were obtained from them. Patients with GCS P score ≤12 at admission were selected and followed up during their hospital stay. Clinical profile, etiology of altered sensorium, and their outcome was assessed.

After collection, the data were compiled and entered in Microsoft Excel sheet. Analysis were done using Statistical software version 16. All continuous variables were expressed as Mean and Standard Deviation and chi square test was used for continuous variables. ANOVA test and Z test were used as test of significance.P value of <0.05 Confidence Interval(CI).

Results

Table 1: Distribution of age among the study participants (N=130)

Sl no	Age	Frequency	Percentage
1	18-30	17	13.1
2	31-40	25	19.2
3	41-50	19	14.6
4	51-60	28	21.5
5	61-70	26	20
6	71-80	13	10
7	>80	2	1.5
Mean±SD		50.98±17.52	18-86

ISSN: 0975-3583,0976-2833 VOL14, ISSUE 01, 2023

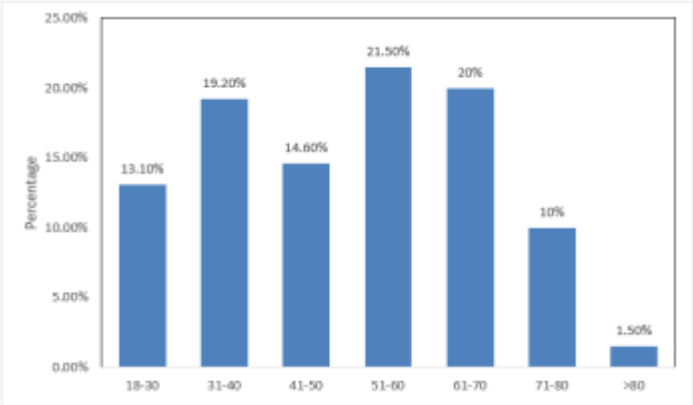

Figure 1: Distribution of age among the study participants (N=130)

Around 13.1% were in the age group of 18-30 years, 19.2% 31-40 years, 14.6% 41-50 years, 21.5% 51-60 years, 20% 61-70 years, 10% 71-80 years and 1.5% >80 years.

Table 2: Distribution of gender among the study participants (N=130)

Sl. No	Gender	Frequency	Percentage
1	Male	71	54.6
2	Female	59	45.4

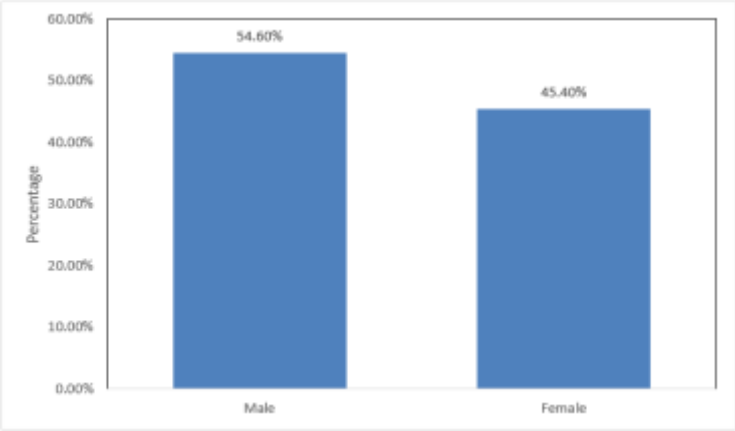

Figure 2: Distribution of gender among the study participants (N=130)

Around 54.6% were males and 54.4% were females.

Table 3: Distribution of symptoms among the study participants (N=130)

Sl no	Symptoms	Frequency	Percentage
1	Headache	70	53.8
2	Vomiting	92	70.8
3	Fever	48	36.9
4	Convulsions	23	17.7
5	Speech disturbances	15	11.5
6	Vertigo	18	13.8
7	Neck pain	10	7.7
8	Neuro deficit	37	28.5
9	Jaundice	19	14.6
10	GI bleeding	8	6.2

ISSN: 0975-3583,0976-2833 VOL14, ISSUE 01, 2023

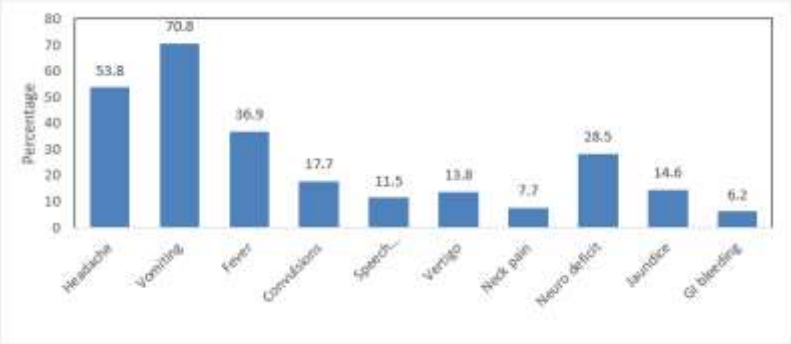

Figure 3: Distribution of symptoms among the study participants (N=130)

Around 53.8% had headache, 70.8% vomiting, 36.9% fever, 17.7% convulsions, 11.5% speech disturbances, 13.8% vertigo, 7.7% neck pain, 28.5% neuro deficit, 14.6% jaundice and 6.2% GI bleeding.

The onset among the study participants were abrupt in 57.7% and were gradual in 42.3%.

Table 4: Distribution of smoking among the study participants (N=130)

Sl No	Smoking	Frequency	Percentage
1	Present	35	26.9
2	Absent	95	73.1

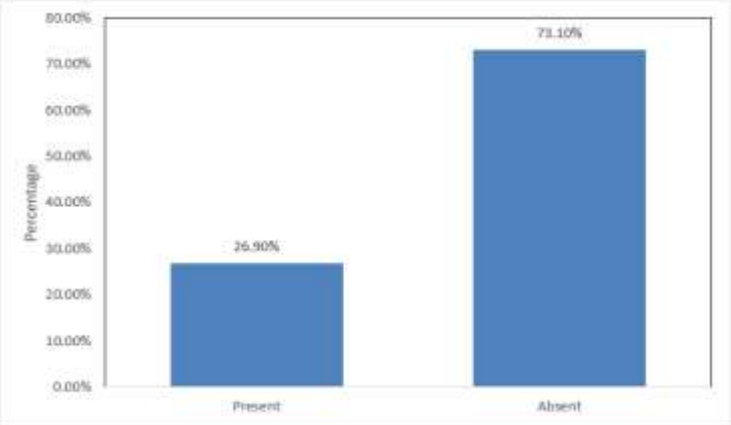

Figure 4: Distribution of smoking among the study participants (N=130)

Around 26.9% were smokers.

Table 5: Distribution of alcoholism among the study participants (N=130)

Sl no	Alcoholism	Frequency	Percentage
1	Present	41	31.5
2	Absent	89	68.5

ISSN: 0975-3583,0976-2833 VOL14, ISSUE 01, 2023

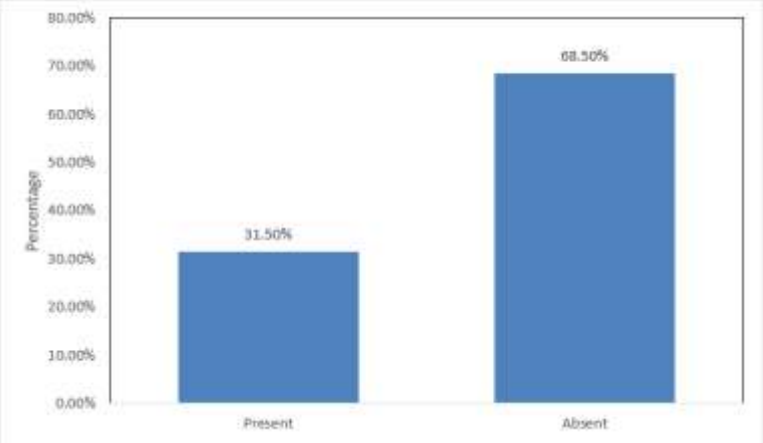

Figure 5: Distribution of alcoholism among the study participants (N=130)

Table 6: Distribution of GCS P Score among the study participants (N=130)

Sl no	GCS P Score	Frequency	Percent
1	1	2	1.5
2	2	2	1.5
3	3	4	3.07
4	4	26	20
5	5	17	13.1
6	6	19	14.6
7	7	41	31.5
8	8	7	5.4
9	9	7	5.4
10	10	5	3.8
11	11	4	3.7
12	12	6	4.6

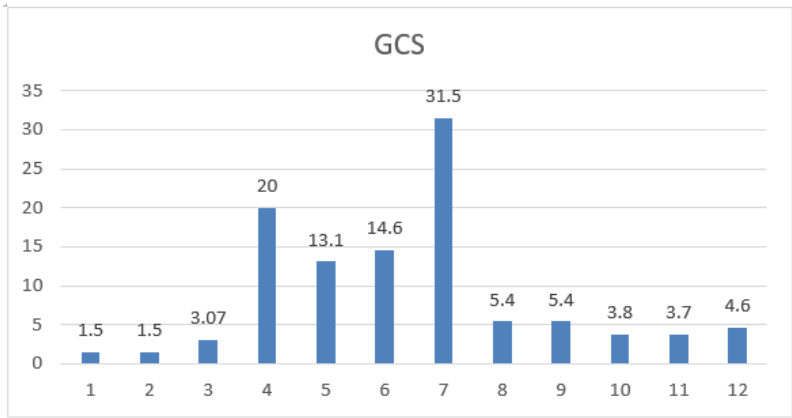

Figure 6: Distribution of GCS P Score among the study participants (N=130)

Around 1.5% had GCS of 1, 1.5 % had GCS of 2, 4.6% had GCS of 3, 21.6% GCS of 4, 13.1% GCS of 5, 14.6% GCS of 6, 31.5% GCS of 7, 5.4% GCS of 8, 5.4% GCS of 9, 3.8% GCS of 10, 3.7% GCS of 11 and 4.6% GCS of 12. Majority of patients had GCS between 4 and 7.

ISSN: 0975-3583,0976-2833 VOL14, ISSUE 01, 2023

Table 7: Distribution of past history among the study participants (N=130)

Sl no	Variable	Frequency	Percentage
1	Hypertension	33	25.4
2	Diabetes mellitus	37	28.5
3	CAD	17	13.1
4	Dyslipidemia	29	22.3
5	Tuberculosis	9	6.9
6	COPD	8	6.2
7	Bronchial asthma	9	6.9
8	Old CVA	11	8.5
9	Liver disease	12	9.2
10	CKD	7	5.4

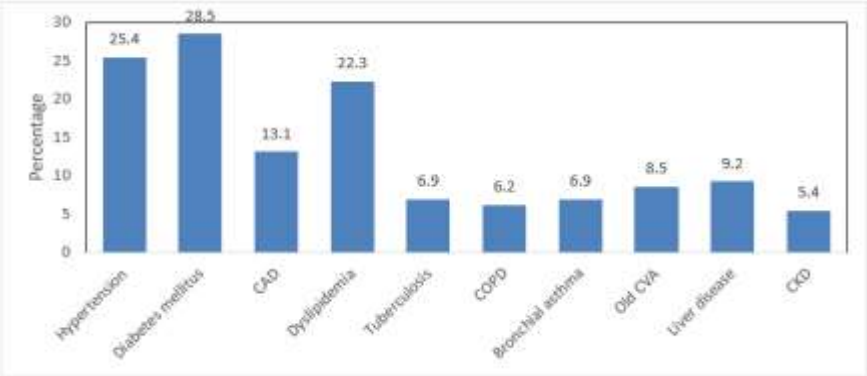

Figure 7: Distribution of past history among the study participants (N=130)

Around 25.4% were having hypertension, 28.5% diabetes mellitus, 13.1% CAD, 22.3% Dyslipidemia, 6.9% tuberculosis, 6.2% COPD, 6.9% bronchial asthma, 8.5% old CVA, 9.2% liver disease and 5.4% CKD. Around 6.2% had drug overdose/poisoning and around 6.9% were exposed to STD's.

Diabetes, hypertension and dyslipidemia were the most prevalent comorbidities.

Table 8: Distribution of etiology among the study participants (N=130)

Sl no	Etiology	Frequency	Percentage
1	**Cerebrovascular accident**	28	21.5
	Infarct	3	2.3
	Intra cerebral hemorrhage	6	4.6
	Subarachnoid hemorrhage	8	6.2
	Cerebral venous thrombosis	11	8.5
2	**Metabolic**	28	21.5
	Hepatic coma	3	2.3
	Uremic coma	3	2.3
	Hypoglycemia	4	3.1
	Hypoxia	9	6.9
	Hypercapnia	1	0.8
	Hyponatremia	3	2.3
	Diabetic Ketoacidosis	5	3.8
3	**Infection**	19	14.6
	Bacterial meningoencephalitis	5	3.8

ISSN: 0975-3583,0976-2833 VOL14, ISSUE 01, 2023

	Tuberculous meningitis	6	4.6
	Cerebral malaria	1	0.8
	Viral encephalitis	4	3.1
	Others	5	3.8
4	**Drug over dosage and poisoning** Drug over dose	25 8	19.2 6.2
	OP poisoning	5	3.8
	Snake bite	6	4.6
	Others	6	4.6
5	**Intracranial neoplasm**	12	9.2
6	**Others**	18	13.8

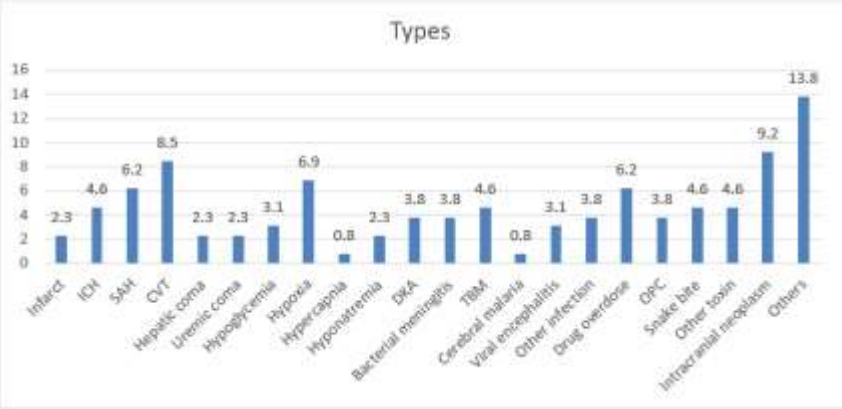

Figure 8: Distribution of etiology among the study participants (N=130)

There were about 21.5% with cerebrovascular accident, 21.5% metabolic, 14.6% infection, 19.2% drug overdosage and poisoning, 9.2% intracranial neoplasm and 13.8% others. The incidence of metabolic and vascular encephalopathy was comparable

Table 9: Distribution of outcome among the study participants (N=130)

Sl no	Outcome	Frequency	Percentage
1	Death	53	40.8
2	Recovery with functional disability	25	19.2
3	Good recovery	52	40.0

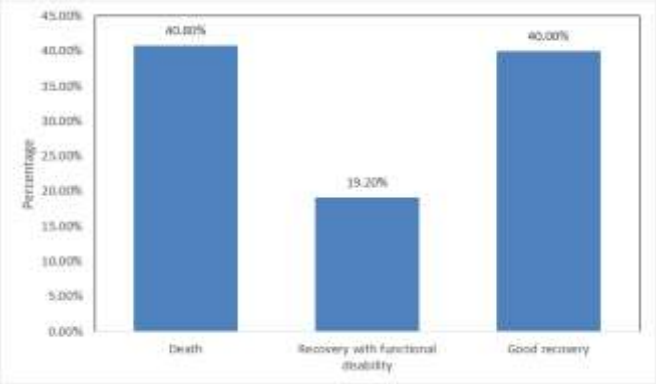

Figure 9: Distribution of outcome among the study participants (N=130)

Around 40.8% died, 19.2% had Recovery with functional disability and 40% had good recovery.

ISSN: 0975-3583,0976-2833 VOL14, ISSUE 01, 2023

Table 10: Association of outcome with study variables (N=130)

Sl no	Variable	I	II	III	p
1	**Respiratory pattern**				
	Normal	1	18	30	<0.001
	Abnormal	52	7	22	
2	**Spontaneous eye movement**				
	Absent	25	0	0	
	Conjugate deviation to left	4	9	1	
	Conjugate deviation to right	4	4	2	
	Roving conjugate	19	12	48	<0.001
	Roving dysconjugate	1	0	0	
3	**Fundus**				
	Diabetic retinopathy	2	2	5	
	Hypertensive retinopathy	7	3	4	0.006
	Normal	25	16	40	
	Papilledema	19	4	3	
4	**Oculocephalic reflex**				
	Present	25	25	50	<0.001
	Absent	28	0	2	
5	**Corneal reflex**				
	Present	25	25	48	<0.001
	Absent	28	0	4	
6	**Pupillary reflex**				
	Present	25	25	49	<0.001
	Absent	28	0	3	
7	**GCS P Score**				
	1-5	39	4	8	
	6-8	20	17	30	<0.001
	9-12	8	4	10	

Respiratory pattern, Spontaneous eye movement, Fundus, Oculocephalic reflex, Corneal reflex, Pupillary reflex and GCS score were significantly associated with outcome(mortality).

Mortality was more when the GCS P score was less/ repiratory pattern was abnormal/ absence of corneal, pupillary and oculocephalic reflex/ absence of spontaneous eye movements.

Table 11: Association of outcome with etiology among the study participants (N=130)

Sl no	Etiology	Frequency	I	II	III	P
1	**Cerebrovascular accident**	28(21.5)	4(3.0)	18(13.8)	6(4.61)	0.71
	Infarct	3(2.3)	2(1.53)	1(0.76)	0	
	Intra cerebral hemorrhage	6(4.6)	2(1.53)	4(3.07)	0	
	Subarachnoid hemorrhage	8(6.2)	0	4(3.07)	4(3.7)	
	Cerebral venous thrombosis	11(8.5)	0	9(6.92)	2(1.53)	
2	**Metabolic**	28(21.5)	16(12.3)	0	12(9.2)	0.67
	Hepatic coma	3(2.3)	3(2.3)	0	0	
	Uremic coma	3(2.3)	2(1.53)	0	1(0.76)	

ISSN: 0975-3583,0976-2833 VOL14, ISSUE 01, 2023

	Hypoglycemia	4(3.1)	0	0	4(3.0)	
	Hypoxia	9(6.9)	6(4.61)	0	3(2.3)	
	Hypercapnia	1(0.8)	1(0.76)	0	0	
	Hyponatremia	3(2.3)	2(1.53)	0	1(0.76)	
	Diabetic Ketoacidosis	5(3.8)	2(1.53)	0	3(2.3)	
3	**Infection**	19(14.6)	6(4.6)	4(3.07)	9(6.9)	0.58
	Bacterial meningoencephalitis	5(3.8)	2(1.53)	0	3(2.3)	
	Tuberculous meningitis	6(4.6)	2(1.53)	2(1.53)	2(1.53)	
	Cerebral malaria	1(0.8)	1(0.76)	0	0	
	Viral encephalitis	4(3.1)	0	0	4(3.0)	
	Others	5(3.8)	2(1.53)	2(1.53)	1(0.76)	
4	**Drug over dosage and poisoning**	25(19.2)	12(9.2)	0	13(10)	0.93
	Drug over dose	8(6.2)	4(3.0)	0	4(3.0)	
	OP poisoning	5(3.8)	3(2.3)	0	2(1.53)	
	Snake bite	6(4.6)	2(1.53)	0	4(3.0)	
	Others	6(4.6)	3(2.3)	0	3(2.3)	
5	**Intracranial neoplasm**	12	6(4.61)	1(0.76)	5(3.8)	
6	**Others**	18	6(4.61)	3(2.3)	9(6.9)	

P value was more than 0.05(by ANOVA test) among all etiologies of various types of encephalopathies. There is no statistical significance among death, disability and recovery among the different etiologies of various types of encephalopathies.

Discussion

Coma is defined as a prolonged state of unconsciousness in which an individual cannot be roused by external stimuli or internal need. Non-traumatic coma is one of the most common issues seen in the Emergency Room. The onset of non-traumatic coma is caused by a vast array of aetiologies.[11,12]

In order to successfully and systematically treat such patients, a physician must be knowledgeable about all of these causes. The prognosis of non-traumatic coma patients depends on their initial level of consciousness (GCS), duration of altered sensorium, aetiology, age, etc.[13,14]

Non-traumatic coma is one of the most significant presentations to a medical emergency department, and early diagnosis and treatment are of the utmost importance to the prognosis of the patient. .[15,16] Although studies on non-traumatic coma are available in the international literature, it appears that India's adult population is understudied. In this study, we describe the etiological profile of non-traumatic coma patients presenting to a tertiary care centre in India, as well as the clinical factors that can be used to predict the prognosis of a comatose patient at presentation.[17,18]

Hence, we conducted this study to determine the etiology, clinical profile and outcome of patients with non-traumatic coma in a tertiary care centre.In our study around 13.1% were in the age group of 18-30 years, 19.2% 31-40 years, 14.6% 41-50 years, 21.5% 51-60 years, 20% 61-70 years, 10% 71-80 years and 1.5% >80 years, and around 54.6% were males and 54.4% were females.

The most prevalent cause of non-traumatic coma, according to our data, is cerebrovascular accident(specifically CVT followed by SAH) and metabolic causes(most common of which is hypoxia). This is followed by toxins(which included drugs, OPC

ISSN: 0975-3583,0976-2833 VOL14, ISSUE 01, 2023

poisoning, snake bite), then infections. The majority of international studies have a comparable etiological profile, with cerebrovascular accident being the most prevalent cause [19,20]. The most common cause of non-traumatic coma in children, according to comparable studies, is central nervous system infection[21,22]

The Study by *Venugopal Suganth et al.i*[23] revealed that symptoms were convulsions (35 cases), fever (34), headache (26), vomiting (23), cough (5), fast breathing (4), abdominal pain (4), diarrhea (2), visual symptom-blindness (1 case). Most of the children presented with more than one symptom. Convulsions (35, 90%) and fever (34, 68%) were the most common symptoms. In our study 53.8% had headache, 70.8% vomiting, 36.9% fever, 17.7% convulsions, 11.5% speech disturbances, 13.8% vertigo, 7.7% neck pain, 28.5% neuro deficit, 14.6% jaundice and 6.2% GI bleeding.

Poisoning is an additional major cause of nontraumatic coma. The prevalence of intoxications in the included studies ranged from <1% to 39%. The in-hospital mortality rate was low (2.4%), whereas the one-year mortality rate was 10.9%. In a recent large observational study of ICU-admitted intoxicated patients, it was found that the proportion of patients with comorbid alcoholism was significantly Similar hospital mortality rates (2,1%) were observed by Brandenburg *et al.*[23]., but mortality rates during follow-up were lower. In our current study around 6.2% had drug overdose/poisoning.

In the study by Kamat *et al.*[24]., patients with metabolic encephalopathy fared better than those with hepatic encephalopathy, which was associated with a 60% mortality rate. Patients with diabetic ketoacidosis, uraemic encephalopathy, hypernatremia, and hypertensive encephalopathy had full recoveries, contrary to the results of our study. There was no significant association of outcome with type of encephalopathy. Disability was noted in patients with cerebrovascular accidents when compared to other types of encephalopathy though death was comparable in our study.

The examination of the brain stem includes the respiratory pattern, pupil size, light response, oculocephalic reflexes, tone, and posture. Diencephalic herniation can be imitated by drugs, toxins, metabolic abnormalities, and intra- and postictal states. If even a few signs of diencephalic, uncal, or midbrain/upper pontine phases of central herniation are observed in children, emergency management of presumed elevated ICP is required. If a patient has reached the lower pontine or medullary stage, recovery is extremely unlikely. In our study the respiratory pattern, spontaneous eye movement, fundus, Oculocephalic reflex, Corneal reflex, Pupillary reflex and GCS P score were significantly associated with outcome.

Conclusion

This study provides the etiological profile of patients presenting with non-traumatic coma and the outcome pattern for different aetiologies. It also emphasises that clinical factors such as coma duration, GCS score, motor response, and brain stem reflexes can provide vital prognostic information regarding the ultimate outcome of these patients. We should advocate for the need for more standardised patient cohort studies to confirm which aetiologies are most prevalent in a non-traumatic coma and to register morbidity and mortality using standardised endpoints.

These studies should include all patients presenting to the hospital with non-traumatic coma, regardless of the cause of non-traumatic coma, and should utilise fixed outcome endpoints. Only then will future opportunities for improving patient outcomes in patients with non-traumatic coma be identified.

Such knowledge of expected functional outcomes is extremely relevant in resource-constrained nations and will assist us in better planning and resource utilisation. Early

ISSN: 0975-3583,0976-2833 VOL14, ISSUE 01, 2023

detection and initiation of appropriate treatment may bring down the rate of disability in patients presenting with various causes of altered mental status.

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Conflict of Interest
The authors declare that there is no conflict of interest.

Authors' Contribution
All authors listed have made a substantial, direct and intellectual contribution to the work and approved it for publication.

References
1. American College of Emergency Physicians. Clinical policy for the initial approach to patients presenting with altered mental status. AnnEmerg Med. 1999;33:251–280
2. Morandi A, Pandharipande P, Trabucchi M, *et al.* Understanding international differences in terminology for delirium and other types of acute brain dysfunction in critically ill patients. Intensive Care Med. 2008;34(10):1907–1915
3. Clarfield AM. The decreasing prevalence of reversible dementias: an updated meta-analysis. Arch Intern Med. 2003;163(18):2219–2229
4. Rummans TA, Evans JM, Krahn LE, Fleming KC. Delirium in elderly patients: evaluation and management. Mayo Clin Proc. 1995 Oct;70(10):989-98.
5. Grover S, Kate N. Assessment scales for delirium: A review. World J Psychiatry. 2012 Aug 22;2(4):58-70.
6. Inouye SK. Delirium in older persons. N Engl J Med. 2006 Mar 16;354(11):1157-65.
7. Hosker C, Ward D. Hypoactive delirium. BMJ. 2017 May 25;357:j2047.
8. Medical Aspects of the Persistent Vegetative State (1): The Multi-Society Task Force on PVS. N Engl J Med. 1994; 330:1499–1508.
9. Giacino J, Whyte J. The Vegetative and Minimally Conscious States: Current Knowledge and Remaining Questions. J Head Trauma Rehabil. 2005;20:30–50.
10. Giacino JT, Ashwal S, Childs N, *et al.* The Minimally Conscious State: Definition and Diagnostic Criteria. Neurology. 2002;58:349–53.
11. Task Force for the Determination of Brain Death in Children. Guidelines for the Determination of Brain Death in Children. Pediatrics. 1987;80:298.
12. Laureys S, Owen AM, Schiff ND. Brain Function in Coma, Vegetative State, and Related Disorders. Lancet Neurol. 2004;3:537–46.
13. Wade S. Smith, Joey D. English. Cerebrovascular diseases. In: Dan L. Longo, Dennis L. Kasper, J. Larry Jameson, Anthony S. Fauci, Stephen L. Hauser, Joseph Loscalzo, eds. Harrison's Principles of Internal Medicine18e. McGraw-Hill. 2011.3270-99.
14. Shawcross DL, OldeDamink SW, Butterworth RF, *et al.*: Ammonia and hepatic encephalopathy: The more things change, the more they remain the same. Metab Brain Dis 2005; 20:169-79.
15. Sathyasaikumar KV, Swapna I, Reddy PV, *et al.*: Fulminant hepatic failure in rats induces oxidative stress differentially in cerebral cortex, cerebellum and pons medulla. Neurochem Res 2007; 32:517-24.
16. Cordoba J, Blei AT: Brain edema and hepatic encephalopathy. Semin Liver Dis 1996; 16:271-80.

ISSN: 0975-3583,0976-2833 VOL14, ISSUE 01, 2023

17. Pereira AA, Weiner DE, Scott T, *et al.*: Cognitive function in dialysis patients. Am J Kidney Dis 2005; 45:448-462.
18. Cooper JD, Lazarowitz VC, Arieff AI: Neurodiagnostic abnormalities in patients with acute renal failure. Evidence for neurotoxicity of parathyroid hormone. J Clin Invest 1978; 61:1448-1455.
19. Hamel, M. B., Goldman, L., Teno, J., Lynn, J., Davis, R. B., Harrell, F. E., ... & Phillips, R. S. (1995). Identification of comatose patients at high risk for death or severe disability. *JAMA*, *273*(23), 1842-1848.
20. Bustos, J. L., Surt, K., & Soratti, C. (2006, December). Glasgow coma scale 7 or less surveillance program for brain death identification in Argentina: Epidemiology and outcome. In *Transplantation proceedings* (Vol. 38, No. 10, pp. 3697-3699). Elsevier.
21. Wong, C. P., Forsyth, R. J., Kelly, T. P., & Eyre, J. A. (2001). Incidence, aetiology, and outcome of non-traumatic coma: a population based study. *Archives of disease in childhood*, *84*(3), 193-199.
22. Brandenburg, R., Brinkman, S., De Keizer, N. F., Meulenbelt, J., & De Lange, D. W. (2014). In-hospital mortality and long-term survival of patients with acute intoxication admitted to the ICU. *Critical care medicine*, *42*(6), 1471-1479.
23. Brandenburg, R., Brinkman, S., De Keizer, N. F., Meulenbelt, J., & De Lange, D. W. (2014). In-hospital mortality and long-term survival of patients with acute intoxication admitted to the ICU. *Critical care medicine*, *42*(6), 1471-1479.
24. Kamat, G. (2019). Clinical Profile, Etiology and Outcome of Non Traumatic Coma in a Tertiary Care Centre.