

To study the different etiologies of non traumatic causes of Altered Sensorium and its correlation with the outcome.

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ABSTRACT

BACKGROUND: Altered sensorium is a measure of a person's arousability and responsiveness to stimuli from the environment. It can result from a variety of factors, including alterations in the chemical environment of the brain (e.g. exposure to poisons or intoxicants), insufficient oxygen or blood flow in the brain and excessive pressure within the skull. Investigations may be equivocal or not available all time and at all places to come to a diagnosis and predict the outcome. The treating physician should identify the clinical signs, interpret and act accordingly to get an idea about the prognosis and outcome in a patient with Altered Sensorium. This study is undertaken to study the various etiologies and compare the same with the outcome in patients with altered sensorium.

METHODS: The study was conducted on 150 patients with altered sensorium of non traumatic origin, over a period of one year at Koppal Institute of Medical Sciences, Koppal after taking consent from patient relatives and ethical committee clearance. All patients of altered sensorium of non-traumatic origin, aged more than 18 years were included in this study. Glasgow coma scale (GCS) is used for primary assessment and also to compare the outcome.

RESULTS: Among total cases of altered sensorium in study aged between 11 to 75 years, majority of them were in age group of 31-40 years with male 31% and female 22%. Different clinical symptoms were being fever (36%), headache (56%), vomiting (53%), weakness (14%), pain abdomen (12%), abdominal distension (5%), yellowish discoloration (8%), blurring vision (22%), convulsion (18%). The most common cause of altered sensorium is infective (32%) followed by Cerebro-Vascular Accident (CVA), metabolic and other causes. Mortality was 100% in ICH and SAH. Complete recovery (100%) was there in patients with hypoglycemia, ketotic encephalopathy, and Wernicke Encephalopathy. Ischemic stroke, CVT, SOL, infections, Hepatic encephalopathy, hyponatremia, Uremic encephalopathy, Hypoxia, Hypercapnia, status epilepticus has all the outcomes. Mortality was 100% in patients with GCS 3. Among patients with GCS 4-8, mortality was 25%, 8% recovered partially and 66% recovered completely. Patients with GCS more than 8 had 70.5% complete recovery, 25.5% had partial recovery and 6% mortality

CONCLUSION: Younger age indicate a better prognosis and unconsciousness in older patient is an unfavorable sign. Most common presenting complaint is vomiting headache, and fever. Infective followed by metabolic are the majority of causes of altered sensorium and both showed good recovery. Among hepatic coma, patient with acute fulminant hepatitis have poor recovery than compared to chronic liver disease with altered sensorium. Patient with metabolic coma which includes hypoglycemia, hyponatremia, ketosis, uremia showed good recovery with proper timely intervention. Alteration of consciousness in patients with cerebrovascular accidents carries a high mortality in which, patients with Intracranial bleed and ischemic infarct with altered sensorium showed poor recovery. Patients with GCS score of 3 had worst prognosis, and 100% mortality.

KEY WORDS: Altered Sensorium, Glasgow coma scale, Cerebro-Vascular Accident, Sub arachnoid Hemorrhage, Intra cranial Hemorrhage.

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I. Introduction

A number of patients with ALTERED SENSORIUM attend the hospitals, identifying the etiology in these patients in altered sensorium is a real challenge to the doctor who is attending to them, for one should know the differential diagnosis of Altered Sensorium. Patients may be in various stages of Altered Sensorium, from delirious state to Coma. There may not be a reliable attendant of the patient to give the complete history of

the patient's disease. The doctor should take possible minimum time, as it may be a totally reversible state and can save the patient with appropriate intervention. So, it is perplexing for the doctor who faces a patient in Altered Sensorium.¹

An altered sensorium is a measurement of a person's arousability and responsiveness to stimuli from the environment¹. A mildly depressed level of consciousness or alertness may be classed as **lethargy**; someone in this state can be aroused with little difficulty.¹ People who are **obtunded** have a more depressed level of consciousness and cannot be fully aroused.^{1, 2} Those who are not able to be aroused from a sleep-like state are said to be **stuporous**.^{1, 2} **Coma** is the inability to make any purposeful response.^{1, 2} Scales such as the Glasgow coma scale have been designed to measure the level of consciousness.

An altered level sensorium can result from a variety of factors, including alterations in the chemical environment of the brain (e.g. exposure to poisons or intoxicants), insufficient oxygen or blood flow in the brain and excessive pressure within the skull. Prolonged unconsciousness is a sign of a medical emergency.³ A deficit in the level of consciousness suggests injury to both the cerebral hemispheres or the reticular activating system.⁴ A decreased level of consciousness correlates to increased morbidity (sickness) and mortality (death).⁵ Thus it is a valuable measure of a patient's medical and neurological status in fact, some sources consider level of consciousness to be one of the vital signs.^{3, 6}

Investigations may be equivocal or not available all time and at all places to come to a diagnosis and predict the outcome. The treating physician should identify the clinical signs, interpret and act accordingly to get an idea about the prognosis and outcome in a patient with Altered Sensorium.

A study of patients in Altered Sensorium, especially in coma in the western world is a matter of great controversy involving the financial burden on the society and ethical demand of doing everything for the patient who would be in a vegetative state. But in India, it is not such a practical problem, yet as the interest is to gauge the outcome whether it is the death or the recovery with or without deficit as the outcome. So this study was undertaken to observe the varied clinical and lab parameter of patients who came with altered sensorium and to study these parameters with respect to diagnosis and clinical outcome.⁶

OBJECTIVES

1. To study etiology of non traumatic cause of altered sensorium.
2. To compare the etiological parameters with the outcome.
3. To study the clinical profile of non traumatic cause of altered sensorium.

II. Methodology

The study was conducted on 150 patients with altered sensorium of non traumatic origin, over a period of one year at Koppal Institute of Medical Sciences, Koppal after taking consent from patient relatives and ethical committee clearance. It is a type of descriptive study.

Inclusion criteria

All adult patients (>18years) presenting with altered sensorium lasting for more than 6 hours to Kims Koppal.

Exclusion criteria

1. Patients with altered sensorium due to head trauma.
2. Transient unresponsiveness due to syncope/hysteria.
3. Unresponsiveness of imminent death

All patients underwent full medical and neurologic clinical evaluation at the time of admission (time of admission to study was arbitrarily taken as time of first neurological assessment). Neurologic condition was judged by evaluating forebrain and brainstem function using Glasgow coma scale (GCS).⁷

Table 1: GCS scoring

Verbal response		Eye opening		Motor response	
Oriented	5	Spontaneously	4	Obedient	6
Confused	4	To noise	3	Localizing	5
Inappropriate words	3	To pain	2	Withdrawal	4
Incomprehensible	2	None	1	Flexor	3
None	1			Extensor	2
				None	1

The total coma score of the patient was calculated by adding the three scores and it was taken to relate the depth of consciousness (lower the score deeper the coma). The patients were subjected to relevant

investigation available in our setup. All available clinical and laboratory data were used to ascertain cause of the altered sensorium.

The following outcome were identified viz..., Death, Persistent vegetative state(awake but unaware), severe disability(dependent but conscious), moderate disability (independent but disabled) and good recovery(jennet and Bond).⁸

The history and general examination, the neurologic profile at the time of admission, important positive investigation findings, the diagnosis of all the cases studied are presented in a tabular form and analyzed using appropriate statistical test.

III. Results

The age and sex distribution of 150 cases out of which 96 were males and 54 were females.

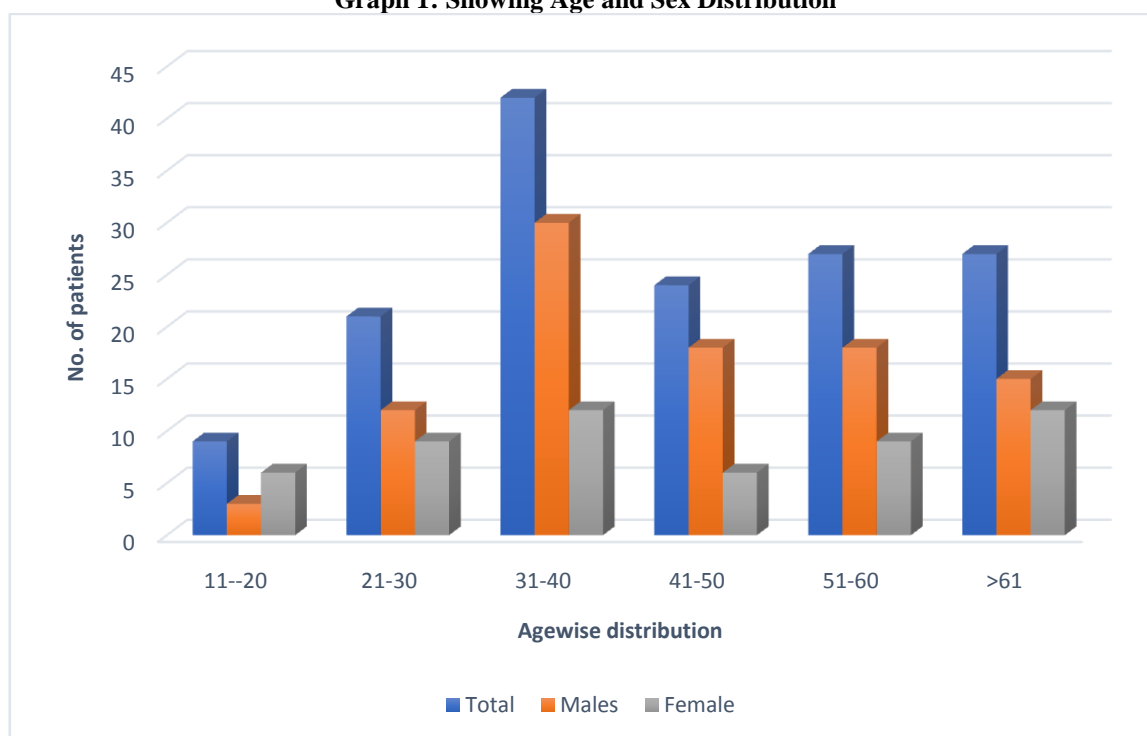
Table 2: Sex distribution

Total	Males	Females
150	96	54

Table 3: Age and sex distribution

	Total n=150	Male n=96	Female n=54
11-20	9	3	6
21-30	21	12	9
31-40	42	30	12
41-50	24	18	6
51-60	27	18	9
>61	27	15	12

Graph 1: Showing Age and Sex Distribution



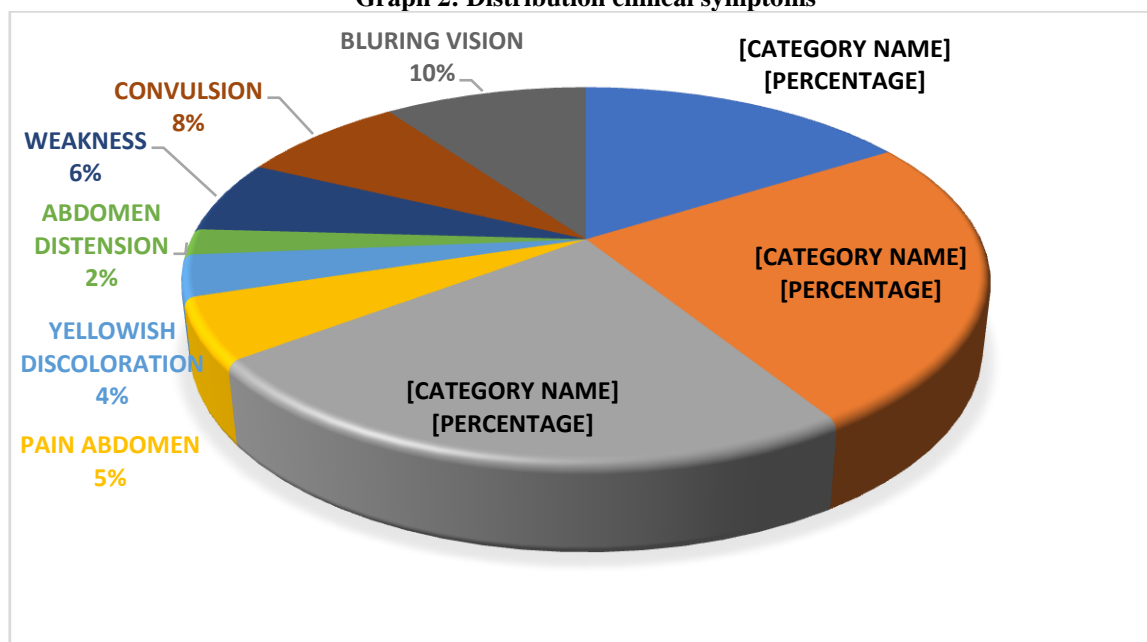
Among total cases of altered sensorium in study aged between 11 to 75 years, majority of them were in age group of 31-40 years with male 31% and female 22%.

Table 4: Clinical Presentation of patients

SYMPTOMS	TOTAL	PERCENTAGE
FEVER	55	36
HEADACHE	85	56
VOMITING	80	53
PAIN ABDOMEN	18	12
YELLOWISH DISCOLORATION	12	8

ABDOMEN DISTENSION	8	5
WEAKNESS	36	14
CONVULSION	28	18
BLURING VISION	34	22

Graph 2: Distribution clinical symptoms



Patients in altered sensorium present with different clinical symptoms viz fever(36%), headache(56%), vomiting(53%), weakness(14%), pain abdomen(12%), abdominal distension(5%), yellowish discoloration(8%),blurring vision(22%), convulsion(18%). Presentation in each patient varies with the pathology and etiology of diseases, most common symptoms is headache followed by vomiting, fever,blurring of vision etc.

Table 5: Etiological distribution of patients with altered sensorium

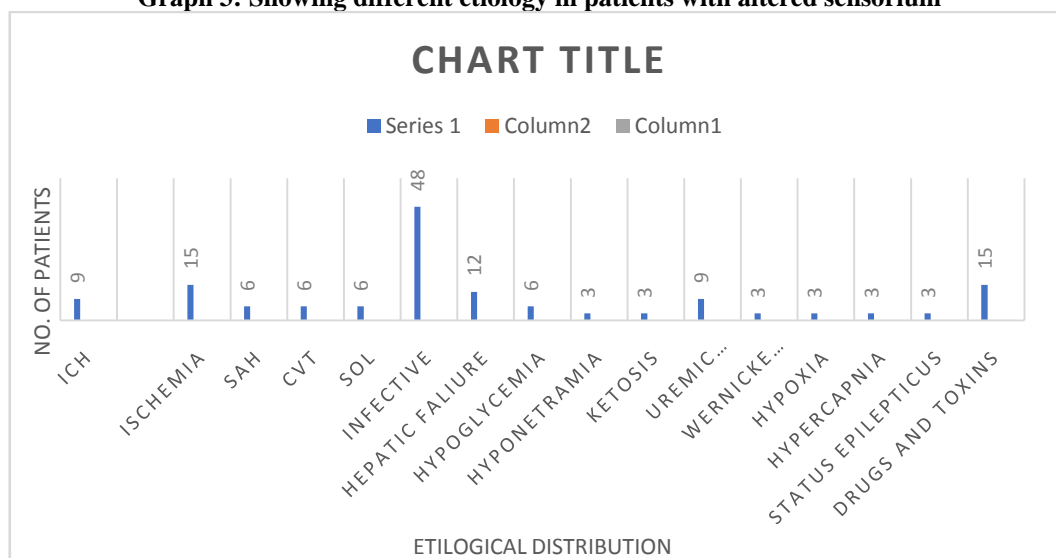
ETIOLOGY	NO. CASES(n=150)
INFECTIVE	48(32%)
CVA	36(24%)
METABOLIC	42(28%)
OTHERS	23(15%)

Among 150 patients, the most common cause of altered sensorium is infective (32%) followed by Cerebro-Vascular Accident (CVA), metabolic and other causes.

Table 6: Showing different etiology in patients with altered sensorium

ETIOLOGY	No . of cases(n=150)
ICH	9
ISCHEMIC STROKE	15
SAH	6
CVT	6
SOL	6
INFECTIVE	48
HEPATIC FALIURE	12
HYPOGLYCEMIA	6
HYPONATREMIA	3
KETOSIS	3
UREMIC ENCEPATHLOPATHY	9
WERNICKE ENCEPHALOPATHY	3
HYPOXIA	3
HYPERCAPNIA	3
STATUS EPILEPTICUS	3
DRUGS AND TOXINS	15

Graph 3: Showing different etiology in patients with altered sensorium



Different etiologies in altered sensorium were being, Intra Cerebral Haemorrhage (ICH), ischemic stroke, sub arachnoid haemorrhage (SAH), Cortical Venous Thrombosis (CVT), Space occupying lesion (SOL), infective cause, Hepatic failure, hypoglycemia, hyponatremia, ketosis, Uremic encephalopathy, Wernicke Encephalopathy, Hypoxia/ Hypercapnia, status epilepticus, drugs and toxin exposure as described above.

Table 7: Association of etiology with outcome of patients

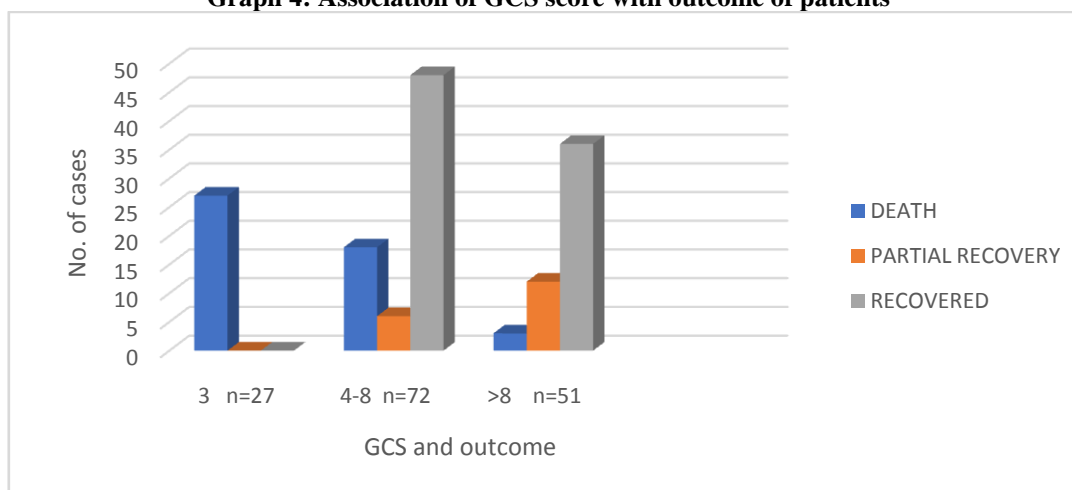
DIAGNOSIS	No. of cases	Deaths	%	Partially recovered	%	Recovered	%
ICH	9	9	100	0	0	0	0
Ischemia	15	3	20	3	20	9	60
SAH	6	6	100	0	0	0	0
CVT	6	1	17	0	0	5	83
SOL	6	3	50	0	0	3	50
Infection	48	12	25	6	12.5	30	62.5
Drugs/toxins	15	6	40	0	0	9	60
Hepatic encephalopathy	12	3	25	0	0	9	75
Ketotic Encephalopathy	3	0	0	0	0	3	100
Uremic encephalopathy	9	3	33.3	0	0	6	66.6
Wernicke's encephalopathy	3	0	0	0	0	3	100
Hypoglycemia	6	0	0	0	0	6	100
Hyponatremia	3	1	33.3	0	0	2	66.6
Hypoxia	3	0	0	3	100	0	0
Hypercapnia	3	3	100	0	0	0	0
Status epilepticus	3	1	33.3	0	0	2	66.6

Mortality was 100% in ICH and SAH. Complete recovery (100%) was there in patients with hypoglycemia, ketotic encephalopathy, and Wernicke Encephalopathy. Ischemic stroke, CVT, SOL, infections, Hepatic encephalopathy, hyponatremia, Uremic encephalopathy, Hypoxia, Hypercapnia, status epilepticus has all the outcomes as described above.

Table 8: Association of GCS score with outcome of patients

GCS->	3 (n=27)	%	4-8 (n=72)	%	>8 n=51	%
DEATH	27	100	18	25	3	6
PARTIAL RECOVERY	0	0	6	8	12	25.5
RECOVERED	0	0	48	66	36	70.5

Graph 4: Association of GCS score with outcome of patients



Considering GCS, mortality was 100% in patients with GCS 3. Among patients with GCS 4-8, mortality was 25%, 8% recovered partially and 66% recovered completely. Patients with GCS more than 8 had 70.5% complete recovery, 25.5% had partial recovery and 6% mortality.

IV. Discussion

In present study, 150 cases of altered sensorium were taken and descriptive study with outcome was determined. Owing to the small number of subjects the results can only be considered as preliminary and no more than trends.

Etiology

The relative frequency of different disease entities that were responsible for altered sensorium in the present study has been compared with other different studies.

The figure in our series is comparable with that of Plum and Posner⁷, but differs with that of Srinivasan in terms of metabolic causes (all causes taken together). The common cause of altered sensorium in the present series is INFECTIVE (32%) followed by metabolic. The next in order of frequency are cardiovascular accident (Hemorrhage, Ischemia, SAH) (24%), drug/exogenous toxins (10%), metabolic (Ketosis Hepatic, Hypoxia, uremia) [34%]. The order of frequency in Plum and Posner series is exogenous toxins (29.8%), CVA (13.2%), hepatic coma (3.4%) and infections (2.6%).

In our case study, CVA is the third main cause of altered sensorium, but in the K Srinivasan study infection (50%) is the main cause.⁹ Infective causes were much less in Plum and Posner series. This reflects the difference in geographic distribution of infections particularly that of viral encephalitis which is more rampant in this country along with poor socioeconomic condition of patient.

In non-traumatic coma study by Arun, Bansal and Sunith¹⁰ infection is the most common cause followed by metabolic and CVA which coincides with our study. Similarly the outcome (mortality) was also comparable with our study. Maximum mortality is by CVA (50%). Most common presenting complaint with patients with altered sensorium will be vomiting, headache followed by fever which itself shows the commonest etiology to be infective.

Etiology and Outcome

The relation of the cause of altered sensorium to outcome in the present study has been outlined below. The number of cases in each group is comparatively very small to draw conclusion, or to compare with other studies which are larger studies done in specialized centers.

Presence of any degree of altered sensorium substantially reduces the chance of a good outcome of patients with cerebral hemorrhage. Among Marquardson's 769 patients less than 1% of those admitted in unresponsive altered sensorium (coma) survived beyond 5 days, and even among those who were nearly drowsy (71% died within three weeks). These poor results reflect a high proportion of acute cerebral hemorrhage.

Among 153 patients studied by Carter 75% of those in coma had intracranial bleeding, and all showed a high mortality rate. Similar finding was noted in our study in which all 9 patients with intracranial bleed showed 100% mortality. Obtundation, stupor, coma with ischemic stroke showed a poor outcome as equally as intracranial bleed. Carter reported that among 95 patients with cerebral infarction in coma for less than 24 hours, 44% died within 4 weeks.¹¹ Similarly in our study patient who presented with infarction and altered

sensorium not lasting more than 24 hours showed good recovery. This was true among our study in which 12 patients with ischemic infarction showed good recovery. Long standing unresponsiveness for 24 - 48 hours led to mortality of 86% and when more than 48 hours led to mortality of 95%. In present study, among 9 cerebral hemorrhage all 9 died, with a mortality of 100% and among ischemic stroke only 3 died of 15 with mortality of 20%. In case of ischemic stroke mortality was more when patients were deeply comatose or wide area of infarction.

Level of consciousness is of major importance in outcome from subarachnoid hemorrhage. According to Richardson the mortality in the first 6 months is 29% for alert patients, 55% for drowsy patients, 71% for stuporous patients and 90% for patients in coma. Age is important, young and alert patients have a mortality one third that of elderly (McKissock et al). In the present study, 6 patients who had SAH with altered sensorium at the time of presentation died with a mortality of 100%.

The cause of hepatic coma importantly influences the outcome, prognosis being far worse in fulminant hepatic failure than in coma associated with chronic cirrhosis of liver (Prytz Ritt).¹² In Bate's study, none of the comatose patients with acute hepatitis survived and among 48 patients in coma secondary to subacute or chronic hepatic disease 22 of them made a moderate/good recovery.¹³ In the present study, 3 of 12 cases died which were cases of fulminant hepatic failure with altered sensorium. The other 9 cases showed good recovery were cirrhosis with portal hypertension with a chronic liver pathology.

GCS helps to predict outcome and identification of comatose patients at high risk for death or severe disability. Patients with abnormal brain stem response, absent verbal response, absent withdrawal response to pain showed more mortality. Patient with absent brain stem reflexes died early which was also the conclusion in a study done in CMC VELLORE by George John.¹⁴

V. Conclusion

This study was concerned to assess the outcome and prognosis of patients presenting to emergency in altered sensorium with minimal history and clinical data including GCS and neurological examination.

Younger age indicated a better prognosis and unconsciousness in older patient is an unfavorable sign. Most common presenting complaint is vomiting headache, and fever. Infective followed by metabolic are the majority of causes of altered sensorium and both showed good recovery. Among hepatic coma, patient with acute fulminant hepatitis have poor recovery than compared to chronic liver disease with altered sensorium. Patient with metabolic coma which includes hypoglycemia, hyponatremia, ketosis, uremia showed good recovery with proper timely intervention. Alteration of consciousness in patients with cerebrovascular accidents carries a high mortality in which, patients with Intracranial bleed and ischemic infarct with altered sensorium showed poor recovery. Patients with GCS score of 3 had worst prognosis, and 100% mortality.

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