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Study of Etiological Profile of Patients Presenting with Altered Sensorium to Emergency Department

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Abstract:
Altered sensorium is a common and potentially life-threatening presentation in the emergency department and may result from a wide range of neurological, metabolic, infective, toxic, and systemic conditions. Early identification of the underlying cause is essential for appropriate management and improving patient outcomes. This observational study was conducted over 18 months in the Emergency Department of Sri Siddhartha Medical College Hospital and Research Centre, Tumakuru, Karnataka, among 150 adult patients presenting with altered sensorium. Patients aged ≥18 years with a Glasgow Coma Scale score <15, disorientation, reduced response to verbal or physical stimuli, or somnolence were included. All patients underwent clinical and neurological assessment, relevant laboratory investigations, and neuroimaging as clinically indicated. Neurological etiologies were the most common cause of altered sensorium (43.3%), followed by metabolic (33.3%), infective (13.3%), toxic (6.7%), and other causes (3.3%). The mean age of the study population was 49.93±16.41 years, with a significant difference across etiological categories. Significant associations were observed between etiology and headache, GCS verbal response, vital parameters, selected biochemical investigations, and neuroimaging findings. ICU admission was required in 91.3% of patients, while 23.3% required ventilatory support. The overall mortality was 21.3%, with neurological etiologies accounting for 62.5% of deaths. Altered sensorium represents a high-risk emergency presentation associated with substantial critical care requirements and mortality. The predominance of neurological and metabolic causes highlights the importance of prompt and systematic neurological, metabolic, and systemic evaluation for early identification of the underlying etiology and appropriate management.
Keywords: Altered sensorium, altered mental status, neurological disorders, metabolic disorders, Glasgow Coma Scale, mortality.

1. Introduction

The interplay of genetics, skeletal-muscle properties, body fat composition, training history, nutrition and recovery are
Altered sensorium is a common and potentially life-threatening presentation in the emergency department [1], encompassing a broad spectrum of disturbances in consciousness ranging from confusion and disorientation to stupor and coma [2] [3]. It is a clinical manifestation rather than a diagnosis and may result from a wide range of neurological and systemic disorders. Altered mental status may account for approximately 4–10% of emergency department presentations [4], and acute changes in mental status require prompt evaluation because they may represent serious and potentially reversible conditions [5]. Normal consciousness depends on the coordinated functioning of the cerebral cortex, thalamus, brainstem, and ascending reticular activating system (ARAS) [6], together with adequate cerebral perfusion, oxygenation, glucose availability, and metabolic homeostasis [7], [8], [9]. Disturbances affecting these mechanisms may produce altered sensorium through either primary neurological injury or systemic dysfunction [10]. The etiological spectrum is broad and includes cerebrovascular disease, intracranial hemorrhage, traumatic brain injury, infections, metabolic and electrolyte abnormalities, hepatic or renal dysfunction, toxicological exposure, medication effects, and systemic illnesses [11], [12]. In many patients, more than one contributing factor may coexist, further complicating the diagnostic process.
Neurological disorders represent an important cause of altered sensorium, particularly in patients with cerebrovascular disease, intracranial hemorrhage, traumatic brain injury, or space-occupying lesions [4], [13], [14]. Vascular events may impair consciousness through direct involvement of structures responsible for arousal or through secondary effects such

as cerebral edema and raised intracranial pressure [16]. Similarly, central nervous system infections may produce altered consciousness through inflammation, cerebral dysfunction, seizures, or systemic complications [17],[15]. These conditions require early recognition because delays in diagnosis and treatment can result in permanent neurological injury and increased mortality [18]. Metabolic and systemic abnormalities are also important and potentially reversible causes [19]. Hypoglycaemia, hyperglycemia, sodium disturbances, renal or hepatic dysfunction, acid–base abnormalities, hypoxia, hypercapnia, and systemic infections can all impair cerebral function. Toxicological causes, including alcohol and drug intoxication, withdrawal syndromes, medication toxicity, and poisoning, may present with a similar clinical picture [20], [21],[22],[23]. The broad differential diagnosis therefore requires clinicians to consider neurological, metabolic, infectious, toxicological, and systemic causes simultaneously rather than focusing on a single diagnostic category[24].

The diagnostic evaluation of altered sensorium is particularly challenging because affected patients may be unable to provide a reliable history [25]. Information from relatives, attendants, previous medical records, and the circumstances surrounding symptom onset may therefore be essential [26]. A systematic clinical assessment, including evaluation of airway, breathing, circulation, neurological status, blood glucose, vital signs, and focused neurological examination, should be performed promptly [27]. Laboratory investigations and neuroimaging are then guided by the clinical presentation and suspected etiology [28],[29]. History and physical examination remain important components of the diagnostic process, while neuroimaging is particularly valuable when an intracranial structural or vascular cause is suspected. The etiological pattern of altered sensorium can vary according to age, geographical region, disease prevalence, referral patterns, and healthcare resources [30]. Previous emergency department studies have demonstrated considerable variation in the relative contribution of neurological, toxicological, traumatic, infectious, metabolic, and psychiatric causes [27]. Importantly, patients presenting with altered sensorium may require substantial healthcare resources and have considerable rates of hospital admission and mortality [31].

Despite the broad differential diagnosis and clinical importance of altered sensorium, locally relevant data on its etiological distribution and associated outcomes remain valuable for emergency care [32],[33]. Understanding the predominant causes and their relationship with clinical findings, laboratory abnormalities, neuroimaging results, and hospital outcomes may assist clinicians in prioritizing early investigations and appropriate escalation of care [4], [34]. Therefore, the present study was undertaken to evaluate the etiological profile of adult patients presenting with altered sensorium to the emergency department and to assess their clinical characteristics and outcomes during hospitalization.

2.Methodology

Study design: An observational study design was adopted for this research. This design was deemed most appropriate as it involved observing and analyzing the characteristics, etiological factors, and outcomes of patients presenting with altered sensorium without any intervention or manipulation of variables by the investigator. The study was purely descriptive and analytical in nature, aiming to document the existing scenario and establish associations between presenting features and final diagnoses.

Study setting: The study was conducted in the Emergency Department (ED) of Sri Siddhartha Medical College Hospital and Research Centre, located in Agalakote, Tumakuru, Karnataka, India. This institution is a tertiary care teaching hospital that caters to a large and diverse patient population from Tumakuru and the surrounding districts. The high patient inflow into the ED provided a robust and representative setting for recruiting an adequate number of cases presenting with altered sensorium, ensuring a wide spectrum of etiologies was captured.

Study duration: The total duration for the conduct of the study, including patient recruitment, data collection, and preliminary analysis, was 18 months. This extended period was necessary to achieve the required sample size, given the specific inclusion criteria and the need to encompass seasonal variations in diseases that could cause altered sensorium, such as infectious etiologies.

Inclusion and exclusion criteria: The study population consisted of adult patients who presented to the emergency department with a chief complaint of altered sensorium. A strict set of criteria was applied to ensure the selection of appropriate participants for the study. Inclusion Criteria (i) Patients aged 18 years and above. (ii) Patients presenting with a Glasgow Coma Scale (GCS) score of less than 15. (iii) Patients who were socially withdrawn and exhibited disorientation to place and time. (iv) Patients with a diminished or absent response to verbal commands or physical stimuli and (v) Patients who were somnolent.

Exclusion Criteria: (i) Patients below 18 years of age. (ii) Pregnant and lactating women and (iii) Patients with a known history of chronic altered sensorium due to pre-existing psychiatric or neurodegenerative conditions, such as Alzheimer's disease, schizophrenia, and other major psychiatric disorders. This was to exclude those with long-standing, stable cognitive deficits and focus on acute alterations.

Study sampling: A non-probability purposive sampling method was utilized for this study. Every consecutive patient who presented to the emergency department during the study period and met the predefined inclusion and exclusion criteria was invited to participate until the desired sample size was achieved. This method was chosen for its practicality and efficiency in a busy clinical setting like the emergency department, where random sampling can be challenging to implement.

Study sample size: The sample size was calculated based on the primary objective of determining the proportion of a specific etiology, which was stroke, with an assumed prevalence of 16.9% from previous literature. The calculation employed the standard formula for estimating a sample size for a proportion in a descriptive study:

$n=z1-a/2\ 2[p1-p]d2$

Where, Z = Z-value for a 95 % confidence interval (1.96), p = Expected proportion (prevalence of Strock = 0.06) and d = absolute precision (6% = 0.06)

Study parameters: The study parameters were categorized into various sections to ensure a holistic data collection process: (i) Sociodemographic Details: Age, sex, occupation, and address. (ii) Clinical Presentation: Chief complaints, history of presenting illness, past medical history, comorbidities, family history, and personal history (including diet, alcohol intake, and smoking). (iii) Vital Signs and Initial Assessment: Parameters from the ABCDE approach Airway, Breathing (Respiratory Rate, SpO2), Circulation (Pulse Rate, Blood Pressure), Disability (GCS score, Pupillary reaction, Random Blood Sugar), and Exposure (Temperature). (iv) General and Systemic Examination: Findings from general physical examination (e.g., pallor, icterus, clubbing) and detailed systemic examination of the pulmonary, cardiovascular, and central nervous systems (including higher mental functions, cranial nerves, motor system, sensory system, reflexes, and meningeal signs). (v) Investigative Parameters: Results of all advised investigations, including GRBS, ECG, Complete Blood Count (CBC), Serum Electrolytes, Renal Function Tests (RFT), Liver Function Tests (LFT), Arterial Blood Gas (ABG) analysis, and neuroimaging (NCCT or MRI Brain). (vi) Outcome Parameters: The final diagnosis (etiology) and the outcome of the patient during their hospital stay (e.g., discharged, left against medical advice, expired).

Study procedure: The procedure for each participant began with immediate stabilization following the ABCDE (Airway, Breathing, Circulation, Disability, Exposure) approach. This was critical to address any immediate life-threatening conditions and to simultaneously identify and manage reversible causes of altered sensorium, such as hypoxia or hypoglycemia. Following stabilization, a detailed history was obtained from the patient (if possible) and/or accompanying relatives or bystanders. A thorough general physical and systemic examination, with an emphasis on a detailed neurological examination, was then performed. Based on the clinical assessment, a provisional diagnosis was made, and a tailored set of investigations was advised to confirm the etiology. All patients were managed as per the standard protocols of the emergency department irrespective of their participation in the study.

Study data collection: Data collection was performed using a pre-designed, semi-structured questionnaire (the clinical proforma provided in the synopsis). This proforma captured all the study parameters in a structured manner. The principal investigator was responsible for filling out the proforma for each enrolled participant by interviewing the attendants, examining the patient, and reviewing the patient's medical records and investigation reports. The collected data was then transferred and entered into a Microsoft Excel spreadsheet that was designed and validated for this study to ensure accuracy and minimize data entry errors.

Data analysis: The data entered into the Excel spreadsheet was imported into Epi Info software, version 3.4.3, for statistical analysis. Descriptive statistics were employed to summarize the data: (i) Quantitative data (e.g., age, GCS score, laboratory values) were presented as mean and standard deviation. The student's t-test was planned to be used as a test of significance for comparing means between groups, if applicable. (ii) Categorical data (e.g., etiological categories, gender, outcome) were expressed as frequencies and percentages.

Inferential statistics were applied to explore associations between variables. The Chi-square test was specified to be used to compare proportions, such as the prevalence of certain etiologies between different demographic groups. To quantify the strength of any associations found, Odds Ratios (OR) with 95% Confidence Intervals (CI) were to be computed. A p value of less than 0.05 was established as the threshold for statistical significance throughout the analysis.

Ethical considerations: Formal ethical clearance for the study was sought from the Institutional Ethical Committee of Sri Siddhartha Medical College Hospital and Research Centre. The approval was awaited at the time of the synopsis submission. The study adhered to all fundamental ethical principles. Written informed consent was obtained from the legally authorized representative (LAR) of each participant before enrolment, given that the participants had altered mental status and lacked decision-making capacity. The consent process involved explaining the study's purpose, procedures, potential risks (which were minimal to zero), and benefits using a detailed information sheet in a language understandable to the LAR. It was explicitly stated that participation was voluntary and that refusal to participate would not affect the standard of care provided. Confidentiality was rigorously maintained by anonymizing data; no personally identifiable information was used in the data analysis or any subsequent presentation or publication of the results. All study-related documents were stored securely, with access limited to the research team.

3.Results

Baseline Demographic Characteristics: As shown in Table 1, the overall mean age of the study population was 49.93 ± 16.41 years. The mean age was highest among patients with neurological etiology (53.77 ± 16.35 years), followed by infective (53.00 ± 12.81 years), metabolic (46.80 ± 16.50 years), other (45.00 ± 14.14 years), and toxic etiologies (37.00 ± 16.19 years). The difference in mean age across etiological categories was statistically significant (p = 0.011). Neurological etiology was the most common among both females (48.3%) and males (40.2%), followed by metabolic etiology (32.8% and 33.7%, respectively), with no statistically significant association between sex and etiological category (p = 0.599). Similarly, neurological etiology was predominant among both rural (44.2%) and urban (41.8%) residents, followed by metabolic etiology (32.6% and 34.5%, respectively), and the association between residence and etiological category was not statistically significant (p = 0.832). Neurological etiology was also the most frequent across all occupational groups, ranging from 35.7% among laborers to 50.0% among service/clerical workers. Metabolic etiology was particularly common among service/clerical workers (41.7%) and those in other occupations (41.7%). However, no statistically significant association was observed between occupation and etiological category (p = 0.423).

Etiological Distribution and Clinical Characteristics: As shown in Table 2, all 150 patients presented with altered sensorium, with neurological etiology being the most common (43.3%), followed by metabolic (33.3%), infective (13.3%), toxic (6.7%), and other etiologies (3.3%). Fever was present in 40.0% of patients, with metabolic etiology being most frequent among those with fever (40.0%); however, the association between fever and etiological category was not statistically significant ($p = 0.498$). Headache was reported in 16.7% of patients and was significantly associated with etiological category ($p = 0.033$), with metabolic etiology predominating among patients with headache (44.0%). Seizures were present in 30.0% of patients, with neurological etiology being the most common (46.7%), although the association was not statistically significant ($p = 0.767$). Vomiting was reported in 33.3% of patients and showed no significant association with etiological category ($p = 0.535$). Weakness was present in 26.7% of patients and was not significantly associated with etiology ($p = 0.465$). Confusion or disorientation was observed in 53.3% of patients, with neurological etiology being predominant (43.8%); however, the association was not statistically significant ($p = 0.949$).

Glasgow Coma Scale and Vital Parameters: As shown in Table 3, neurological etiology was predominant across the GCS eye and motor response categories. The association between GCS eye response and etiological category was not statistically significant ($p = 0.372$), and similarly, no significant association was observed for GCS motor response ($p = 0.887$). In contrast, GCS verbal response showed a statistically significant association with etiological category ($p = 0.028$), with neurological etiology predominating across most verbal response categories. Among patients with total GCS scores of 3–8, metabolic etiology was most frequent (46.5%), whereas neurological etiology predominated among those with GCS scores of 9–15 (52.0%); however, the association between total GCS score and etiological category was not statistically significant ($p = 0.491$).

Significant differences across etiological categories were observed for all assessed vital parameters. Pulse rate was highest in the other etiological group (97.20 ± 15.32 beats/min) and lowest in toxic etiology (85.20 ± 13.90 beats/min; $p = 0.006$). Systolic blood pressure differed significantly across etiologies ($p = 0.002$), with the highest mean value observed in metabolic etiology (140.22 ± 26.42 mmHg). Diastolic blood pressure was also significantly different ($p = 0.014$), with the lowest mean value observed in the other etiological group (72.20 ± 7.66 mmHg). Respiratory rate was highest among infective etiologies (26.35 ± 5.60 breaths/min; $p = 0.022$), while body temperature was highest in toxic etiologies (38.52 ± 0.91 °C; $p = 0.005$). Mean SpO_2 was lowest among toxic etiologies (81.00 ± 6.68%; $p = 0.012$), and MAP was lowest in toxic etiologies (80.40 ± 13.28 mmHg; $p = 0.040$).

Biochemical Parameters: Biochemical analysis demonstrated statistically significant differences across etiological categories for all assessed parameters (Table 4). The overall mean random blood sugar was 140.18 ± 79.26 mg/dL, with the highest mean value observed in metabolic etiology (159.60 ± 129.33 mg/dL; $p = 0.026$). Mean serum sodium was significantly lower in the metabolic group (133.70 ± 9.80 mEq/L), with an overall mean of 138.17 ± 6.94 mEq/L ($p < 0.001$). Serum potassium also differed significantly across etiological categories ($p = 0.026$), although the differences in mean values were relatively small. The overall mean ABG pH was 7.323 ± 0.069, with the lowest mean pH observed in the toxic group (7.303 ± 0.070; $p = 0.007$). Serum ammonia levels were highest among patients with infective etiology (97.40 ± 43.66 µmol/L), with an overall mean of 86.95 ± 38.64 µmol/L, and the difference across etiological categories was statistically significant ($p = 0.035$).

Neuroimaging Findings: Neuroimaging findings showed a statistically significant association with etiological category (Table 5). On NCCT brain, all patients with hemorrhagic stroke, ischemic stroke, and mass lesions were classified under neurological etiology (100% each), while among patients with a normal NCCT, metabolic etiology was most frequent (48.5%), followed by infective (19.4%) and neurological (17.5%) etiologies ($p < 0.001$). MRI was performed in 60 patients, of whom 16 (80.0%) with infective etiology demonstrated infection. MRI findings were significantly associated with etiological category ($p = 0.003$)

Table 1. Baseline Demographic Characteristics of Patients Presenting with Altered Sensorium According to Etiological Category (n = 150)

Characteristic	Infective n (%)	Metabolic n (%)	Neurological n (%)	Other n (%)	Toxic n (%)	Total n (%)	p-value
Age (years)	20.00	50.00	65.00	05.00	10.00	150.00	
Mean ± SD	53.00 ± 12.814	46.80 ± 16.499	53.77 ± 16.347	45.00 ± 14.142	37.00 ± 16.193	49.93 ± 16.415	0.011
Sex							0.599
Female	08 (13.8 %)	19 (32.8 %)	28 (48.3 %)	1 (1.7 %)	2 (3.4 %)	58 (100 %)	
Male	12 (13.0 %)	31 (33.7 %)	37 (40.2 %)	4 (4.3 %)	8 (8.7 %)	92 (100 %)	
Residence							0.832
Rural	13 (13.7 %)	31 (32.6 %)	42 (44.2 %)	4 (4.2 %)	5 (5.3 %)	95 (100 %)	
Urban	7 (12.7 %)	19 (34.5 %)	23 (41.8 %)	1 (1.8 %)	5 (9.1 %)	55 (100 %)	
Occupation							0.423
Farmer	6 (21.4 %)	6 (21.4 %)	11 (39.3 %)	3 (10.7 %)	2 (7.1 %)	28 (100 %)	
Housewife	8 (13.8 %)	19 (32.8 %)	28 (48.3 %)	1 (1.7 %)	2 (3.4 %)	58 (100 %)	
Laborer	4 (14.3 %)	10 (35.7 %)	10 (35.7 %)	0 (0.0 %)	4 (14.3 %)	28 (100 %)	
Other	2 (8.3 %)	10 (41.7 %)	10 (41.7 %)	1 (4.2 %)	1 (4.2 %)	24 (100 %)	
Service/Clerk	0 (0.0 %)	5 (41.7 %)	6 (50.0 %)	0 (0.0 %)	1 (8.3 %)	12 (100 %)	

Table 2. Etiological Distribution and Clinical Characteristics of Patients Presenting with Altered Sensorium (n = 150); *Statistically significant.

Clinical characteristic	Category	Infective n (%)	Metabolic n (%)	Neurological n (%)	Other n (%)	Toxic n (%)	Total n (%)	p-value
Chief complaint	Altered sensorium	20 (13.3 %)	50 (33.3 %)	65 (43.3 %)	05 (3.3 %)	10 (06.7 %)	150 (100)	N/A
Fever	No	11 (12.2 %)	26 (28.9 %)	42 (46.7 %)	04 (4.4 %)	07 (07.8 %)	90 (100)	0.498
	Yes	09 (15.0 %)	24 (40.0 %)	23 (38.3 %)	01 (1.7 %)	03 (05.0 %)	60 (100)	
Headache	No	19 (15.2 %)	39 (31.2 %)	58 (46.4 %)	03 (2.4 %)	06 (04.8 %)	125 (100)	0.033*
	Yes	01 (04.0 %)	11 (44.0 %)	7 (28.0 %)	02 (8.0	04 (16.0	25 (100)	

					%)	%)		
Seizures	No	16 (15.2 %)	35 (33.3 %)	44 (41.9 %)	04 (3.8 %)	06 (05.7 %)	105 (100)	0.767
	Yes	04 (8.9 %)	15 (33.3 %)	21 (46.7 %)	01 (2.2 %)	04 (08.9 %)	45 (100)	
Vomiting	No	12 (12.0 %)	32 (32.0 %)	47 (47.0 %)	02 (2.0 %)	07 (07.0 %)	100 (100)	0.535
	Yes	08 (16.0 %)	18 (36.0 %)	18 (36.0 %)	03 (6.0 %)	03 (06.0 %)	50 (100)	
Weakness	No	15 (13.6 %)	38 (34.5 %)	49 (44.5 %)	03 (2.7 %)	05 (04.5 %)	110 (100)	0.465
	Yes	05 (12.5 %)	12 (30.0 %)	16 (40.0 %)	02 (5.0 %)	05 (12.5 %)	40 (100)	
Confusion/disorientation	No	08 (11.4 %)	25 (35.7 %)	30 (42.9 %)	02 (2.9 %)	05 (07.1 %)	70 (100)	0.949
	Yes	12 (15.0 %)	25 (31.3 %)	35 (43.8 %)	03 (3.8 %)	05 (06.3 %)	80 (100)	

Table 3. Glasgow Coma Scale and Vital Parameters According to Etiological Category (n = 150); Values are presented as n (%) or mean ± SD. BP, blood pressure; GCS, Glasgow Coma Scale; MAP, mean arterial pressure; SD, standard deviation; SpO_2, peripheral oxygen saturation. GCS Eye Response: 1 = No eye opening; 2 = Eye opening to pain; 3 = Eye opening to voice; 4 = Spontaneous eye opening; GCS Verbal Response: 1 = No verbal response; 2 = Incomprehensible sounds; 3 = Inappropriate words; 4 = Confused conversation; 5 = Oriented; GCS Motor Response: 1 = No motor response; 2 = Extension
3 = Abnormal flexion; 4 = Withdrawal; 5 = Localizes pain; 6 = Obeys commands; *Statistically significant.

Parameter	Category	Infective n (%)	Metabolic n (%)	Neurological n (%)	Other	Toxic	Total	p-value
GCS Eye Response	1	03 (10.0 %)	06 (20.0 %)	20 (66.7 %)	00 (00.0 %)	01 (03.3 %)	30 (100 %)	0.372
	2	05 (13.5 %)	17 (45.9 %)	11 (29.7 %)	02 (05.4 %)	02 (05.4 %)	37 (100 %)	
	3	07 (17.5 %)	13 (32.5 %)	15 (37.5 %)	01 (02.5 %)	04 (10.0 %)	40 (100 %)	
	4	05 (11.6 %)	14 (32.6 %)	19 (44.2 %)	02 (04.7 %)	03 (07.0 %)	43 (100 %)	
GCS Verbal Response	1	04 (18.2 %)	09 (40.9 %)	08 (36.4 %)	00 (00.0 %)	01 (04.5 %)	22 (100 %)	0.028*
	2	09 (25.7 %)	05 (14.3 %)	15 (42.9 %)	02 (05.7 %)	04 (11.4 %)	35 (100 %)	
	3	02 (08.3 %)	10 (41.7 %)	11 (45.8 %)	01 (04.2 %)	00 (00.0 %)	24 (100 %)	
	4	00 (00.0 %)	10 (28.6 %)	22 (62.9 %)	00 (00.0 %)	03 (08.6 %)	35 (100 %)	
	5	05 (14.7 %)	16 (47.1 %)	09 (26.5 %)	02 (05.9 %)	02 (05.9 %)	34 (100 %)	
GCS Motor Response	1	04 (12.1 %)	11 (33.3 %)	16 (48.5 %)	01 (03.0 %)	01 (03.0 %)	33 (100 %)	0.887
	2	03 (13.0 %)	08 (34.8 %)	11 (47.8 %)	01 (04.3 %)	00 (00.0 %)	23 (100 %)	

	3	03 (13.0 %)	08 (34.8 %)	10 (43.5 %)	00 (00.0 %)	02 (08.7 %)	23 (100 %)	
	4	01 (04.5 %)	09 (40.9 %)	10 (45.5 %)	00 (00.0 %)	02 (09.1 %)	22 (100 %)	
	5	06 (18.8 %)	10 (31.3 %)	12 (37.5 %)	01 (03.1 %)	03 (09.4 %)	32 (100 %)	
	6	03 (17.6 %)	04 (23.5 %)	06 (35.3 %)	02 (11.8 %)	02 (11.8 %)	17 (100 %)	
Total GCS	3–8	10 (14.1 %)	33 (46.5 %)	26 (36.6 %)	03 (4.2 %)	03 (04.2 %)	75 (100 %)	0.491
	9–15	10 (13.3 %)	17 (22.7 %)	39 (52.0 %)	02 (2.7 %)	07 (09.3 %)	75 (100 %)	
Pulse rate (beats/min)	Mean ± SD	91.55 ± 19.72	90.20 ± 16.77	88.54 ± 17.52	97.20 ± 15.32	85.20 ± 13.90	89.56 ± 17.21	0.006
Systolic BP (mmHg)	Mean ± SD	139.60 ± 22.23	140.22 ± 26.42	131.29 ± 28.74	133.00 ± 41.78	134.50 ± 26.37	135.65 ± 27.46	0.002
Diastolic BP (mmHg)	Mean ± SD	83.95 ± 12.89	88.64 ± 13.93	86.74 ± 14.39	72.20 ± 7.66	84.20 ± 12.03	86.35 ± 13.92	0.014
Respiratory rate (breaths/min)	Mean ± SD	26.35 ± 5.60	24.42 ± 6.61	23.82 ± 6.79	19.80 ± 2.39	23.90 ± 7.48	24.23 ± 6.56	0.022
Body temperature (°C)	Mean ± SD	38.21 ± 1.23	37.96 ± 1.16	37.97 ± 1.15	37.88 ± 1.18	38.52 ± 0.91	38.03 ± 1.15	0.005
SpO_2 (%)	Mean ± SD	88.95 ± 8.61	85.66 ± 8.60	84.46 ± 8.88	85.20 ± 7.26	81.00 ± 6.68	85.25 ± 8.67	0.012
MAP (mmHg)	Mean ± SD	81.65 ± 14.26	84.76 ± 15.68	85.09 ± 14.96	81.20 ± 15.66	80.40 ± 13.28	84.08 ± 14.93	0.040

ICU Admission, Ventilatory Support and Clinical Outcomes: ICU admission was required in 137 (91.3%) patients and was significantly associated with etiological category ($p = 0.031$). ICU admission was required in all patients with infective and other etiologies, followed by neurological (92.3%), toxic (90.0%), and metabolic (86.0%) etiologies.

Ventilatory support was required in 35 (23.3%) patients, with neurological etiology accounting for the largest proportion (45.7%), followed by metabolic (31.4%) and infective (17.1%) etiologies. However, the association between ventilatory support and etiological category was not statistically significant ($p = 0.701$). Among patients who died, neurological etiology accounted for the largest proportion (62.5%), followed by metabolic (21.9%) and toxic (9.4%) etiologies. Among recovered and discharged patients, metabolic etiology was most frequent (39.8%), followed by neurological (33.0%) and infective (17.0%) etiologies. The association between clinical outcome and etiological category was not statistically significant ($p = 0.143$).

Clinical Outcomes According to Etiological Category: Among deaths, neurological etiology constituted 62.5%, followed by metabolic causes at 21.9% and infective causes at 9.4% (Figure 1). Among patients who recovered and were discharged, metabolic etiology was most frequent (39.8%), followed by neurological causes (33.0%) and infective causes (17.0%). The association between clinical outcome and etiological category was not statistically significant ($p = 0.143$).

Discussion

In the present study involving 150 patients presenting with altered sensorium, neurological etiologies constituted the largest proportion (43.3%), followed by metabolic causes (33.3%), infective etiologies (13.3%), toxic causes (6.7%), and other causes (3.3%), indicating that acute central nervous system pathology and systemic metabolic disturbances accounted for the majority of cases. This distribution is broadly consistent with the previous findings [35], who reported neurological causes as the most common discharge diagnosis (28%) among emergency department patients with altered mental status, although toxicologic causes were more prominent in their cohort (21%) compared with the present study (6.7%) 85. In contrast [36], documented a much higher predominance of neurological etiologies (71.6%) and markedly lower proportions of metabolic (6.1%) and toxic causes (1.5%), suggesting geographic and healthcare system–related variability in etiological patterns. Delirium-focused studies, while not categorizing broad etiologies, emphasize that altered mentation frequently reflects underlying neurological, infectious, or metabolic precipitants. Kennedy et al. (2014) reported delirium in 9% of older emergency patients, commonly associated with infection and

Table 4. Biochemical Parameters According to Etiological Category (n = 150) *Statistically significant.

Biochemical parameter	Infective(n=20)	Metabolic(n=50)	Neurological (n=65)	Other (n=5)	Toxic (n=10)	Total (n=150)	p-value
Random blood sugar (mg/dL)	129.55 ± 27.61	159.60 ± 129.33	133.12 ± 30.70	110.80 ± 22.76	124.90 ± 31.01	140.18 ± 79.26	0.026*
Serum sodium (mEq/L)	139.65 ± 3.07	133.70 ± 9.80	140.52 ± 3.20	142.20 ± 1.10	140.30 ± 3.50	138.17 ± 6.94	<0.001*
Serum potassium (mEq/L)	4.27 ± 0.48	4.20 ± 0.41	4.25 ± 0.46	4.28 ± 0.65	4.15 ± 0.38	4.23 ± 0.44	0.026*
ABG pH	7.327 ± 0.079	7.328 ± 0.072	7.320 ± 0.064	7.342 ± 0.075	7.303 ± 0.070	7.323 ± 0.069	0.007*
Serum ammonia (µmol/L)	97.40 ± 43.66	87.32 ± 41.25	84.75 ± 36.37	83.60 ± 36.32	80.10 ± 33.10	86.95 ± 38.64	0.035*

Figure 5: Neuroimaging Findings According to Etiological Category (n = 150) *Statistically significant.

Neuroimaging finding	Infective n (%)	Metabolic n (%)	Neurological n (%)	Other n (%)	Toxic n (%)	Total n (%)	p-value
NCCT Brain							<0.001*
Hemorrhagic stroke	00 (00.0 %)	00 (00.0 %)	12 (100.0 %)	00 (0.0 %)	00 (0.0 %)	12 (100 %)	
Ischemic stroke	00 (00.0 %)	00 (00.0 %)	20 (100.0 %)	00 (0.0 %)	00 (0.0 %)	20 (100 %)	
Mass lesion	00 (00.0 %)	00 (00.0 %)	15 (100.0 %)	00(0.0 %)	00 (0.0 %)	15 (100 %)	
Normal	20 (19.4 %)	50 (48.5 %)	18 (17.5 %)	05 (4.9 %)	10 (9.7 %)	103 (100 %)	
Total	20 (13.3 %)	50 (33.3 %)	65 (43.3 %)	05 (3.3 %)	10 (6.7 %)	150 (100 %)	
MRI Brain							0.003*
Infection	16 (80.0 %)	18 (36.0 %)	22 (33.8 %)	01 (20.0 %)	03 (30.0 %)	60 (100 %)	
Not done	04 (20.0 %)	32 (64.0 %)	43 (66.2 %)	04 (80.0 %)	07 (70.0 %)	90 (100 %)	
Total	20 (13.3 %)	50 (33.3 %)	65 (43.3 %)	05 (3.3 %)	10 (6.7 %)	150 (100 %)	

Figure 6: ICU Admission, Ventilatory Support and Clinical Outcomes According to Etiological Category (n = 150)

Parameter	Category	Infective n (%)	Metabolic n (%)	Neurological n (%)	Other n (%)	Toxic n (%)	Total n (%)	p-value
ICU admission	No	00 (00.0 %)	07 (14.0 %)	05 (07.7 %)	00 (00.0 %)	01 (10.0 %)	13 (100 %)	0.031*
	Yes	20 (100.0 %)	43 (86.0 %)	60 (92.3 %)	05 (100.0 %)	09 (90.0 %)	137 (100 %)	
Ventilatory support	Yes	06 (17.1 %)	11 (31.4 %)	16 (45.7 %)	00 (0.0 %)	02 (05.7 %)	35 (100 %)	0.701
	No	14 (12.2 %)	39 (33.9 %)	49 (42.6 %)	05 (4.3 %)	08 (07.0 %)	115 (100 %)	
Clinical outcome	Death	02 (06.3 %)	07 (21.9 %)	20 (62.5 %)	00 (0.0 %)	03 (09.4 %)	32 (100 %)	0.143
	Recovered & discharged	18 (15.3 %)	47 (39.8 %)	39 (33.0 %)	05 (4.2 %)	09 (07.6 %)	118 (100 %)	

intracranial pathology. Overall, the etiological profile observed highlights the heterogeneous nature of altered sensorium and underscores the importance of a broad yet prioritized diagnostic approach in emergency settings.

The overall mean age of patients in the present study was 49.93 ± 16.41 years, with a statistically significant variation across etiological categories (p = 0.011). Higher mean ages were observed in neurological (53.77 ± 16.35 years) and infective (53.00 ± 12.81 years) etiologies, whereas toxic causes occurred at a younger mean age (37.00 ± 16.19 years), and metabolic etiologies showed an intermediate mean age (46.80 ± 16.49 years). This age-related distribution suggests that cerebrovascular disease and infections contribute more prominently to altered sensorium in older individuals, while toxic exposures are more common in younger populations. Comparable age profiles were reported [35], where the mean age was 49 years, closely matching the present findings 85. Similarly, previously reported [36] a mean age of 45.6 years, reinforcing that altered mental status is predominantly encountered in middle-aged and older adults. Geriatric emergency department studies further contextualize these observations. Han et al. [37] demonstrated delirium in 8.3% of patients aged 65 years and above, highlighting older age as a major risk factor for altered mentation 86. The present findings therefore support age as a significant determinant influencing etiological distribution in altered sensorium presentations.

Male patients constituted 61.3% of the study population; however, the distribution of etiological categories did not differ significantly between sexes (p = 0.599). Among females, neurological etiology was most common (48.3%), followed by metabolic (32.8%) and infective causes (13.8%), while among males, neurological causes also predominated (40.2%), followed by metabolic (33.7%) and infective etiologies (13.0%). Toxic etiologies were slightly more frequent in males (8.7%) compared with females (3.4%), though this difference was not statistically significant. These findings suggest that sex does not independently influence etiological patterns once patients present with altered sensorium. Similar male predominance was observed by Kanich et al. [35], where 57% of patients were men, without evidence of sex-specific etiological variation. Kekec et al. [36] also reported a near-balanced sex distribution (52.3% male) with neurological causes dominating in both sexes. Delirium studies have likewise demonstrated that sex is less influential than age, baseline cognitive status, and acute illness severity in determining altered mentation. Kennedy et al. [38] did not identify sex as an independent predictor of delirium compared with factors such as infection or prior stroke 90. Overall, the findings indicate that etiological evaluation of altered sensorium should be guided by clinical and biochemical parameters rather than sex alone.

Clinical symptom analysis revealed selective associations with etiological categories. Fever was present in 40.0% of patients, but no statistically significant association with etiology was observed (p = 0.498). Neurological etiologies predominated in both fever-absent (46.7%) and fever-present (38.3%) groups, while metabolic causes were relatively more frequent among febrile patients (40.0%). In contrast, headache demonstrated a statistically significant association with etiological category (p = 0.033). Among patients presenting with headache, metabolic etiologies were most frequent (44.0%), followed by neurological (28.0%) and toxic causes (16.0%). Patients without headache showed a predominance of neurological etiologies (46.4%) and a much lower proportion of toxic causes (4.8%). These findings suggest that headache may serve as a useful discriminating symptom in early etiological assessment. Kanich et al. [35] emphasized that history of the presenting event contributed the highest diagnostic yield (51%) compared with investigations, highlighting the importance of symptom-based evaluation 85. Furthermore, Hustey and Meldon [39] demonstrated that impaired mental status was frequently under recognized due to inadequate clinical documentation, reinforcing the value of careful symptom assessment. The present findings indicate that while fever alone is nonspecific, headache has greater etiological relevance in altered sensorium.

Neurological status assessed using the Glasgow Coma Scale showed differential associations with etiological categories. GCS eye response (p = 0.372) and motor response (p = 0.887) did not demonstrate statistically significant associations, whereas GCS verbal response showed a significant association with etiology (p = 0.028). Neurological etiologies increased with better verbal response, reaching 62.9% in patients with verbal score 4, while metabolic etiologies were more frequent in lower verbal response categories. When total GCS score was analyzed, patients with severe impairment (GCS 3–8) demonstrated a higher proportion of metabolic etiologies (46.5%), whereas those with GCS 9–15 showed predominance of neurological causes (52.0%), though this difference was not statistically significant (p = 0.491). These findings suggest that profound depression of consciousness is more commonly associated with metabolic disturbances. Structured mental status assessment has been emphasized in prior literature. Han et al. [40] demonstrated that a two-step delirium screening approach achieved high sensitivity (98%) and specificity (up to 96.9%), improving recognition of altered mentation. Similarly, Sharma et al. (2017) identified universal disturbances in attention and thought processes among delirious ICU patients, regardless of motor activity. The present findings reinforce the importance of detailed neurological and verbal assessment in guiding etiological evaluation of altered sensorium.

Analysis of vital parameters demonstrated significant physiological variation across etiological categories, reflecting differing pathophysiological mechanisms underlying altered sensorium. The overall mean pulse rate was 89.56 ± 17.21 beats/min, with significantly higher values in other etiologies (97.20 ± 15.32) and infective causes (91.55 ± 19.72), and lower values in toxic etiologies (85.20 ± 13.90) (p = 0.006). Mean systolic blood pressure was 135.65 ± 27.46 mmHg, highest in metabolic (140.22 ± 26.42 mmHg) and infective etiologies (139.60 ± 22.23 mmHg), while neurological causes showed comparatively lower values (131.29 ± 28.74 mmHg), with a statistically significant difference (p = 0.002). Diastolic blood pressure also varied significantly (p = 0.014), being lowest in other etiologies (72.20 ± 7.66 mmHg). Respiratory rate showed significant elevation in infective etiologies (26.35 ± 5.60 breaths/min) compared with other groups (p = 0.022). These findings support the observation by Kanich et al. [35] that physiological instability is common in altered mental status and contributes substantially to hospital admission and resource utilization. The

marked variability across etiologies highlights the importance of early vital parameter assessment in etiological stratification and risk assessment.

Body temperature, oxygen saturation, and mean arterial pressure showed statistically significant variation across etiological categories. The overall mean temperature was 38.03 ± 1.15 °C, with higher values in toxic (38.52 ± 0.91 °C) and infective etiologies (38.21 ± 1.23 °C) compared with neurological and metabolic causes ($p = 0.005$). Mean oxygen saturation was 85.25 ± 8.67%, with the lowest values observed in toxic etiologies (81.00 ± 6.68%) and relatively higher saturation in infective causes (88.95 ± 8.61%), showing a statistically significant difference ($p = 0.012$). Mean arterial pressure differed significantly ($p = 0.040$), with lower values in toxic (80.40 ± 13.28 mmHg) and infective etiologies (81.65 ± 14.26 mmHg). These findings indicate greater hemodynamic and respiratory compromise in toxic and infective causes of altered sensorium. The importance of recognizing systemic physiological derangements is emphasized in delirium literature, where Rieck et al. [41] highlighted that timely identification of underlying medical precipitants is critical to improving outcomes. The present results reinforce that abnormal temperature, hypoxia, and hypotension serve as important clinical indicators of etiology and severity in altered sensorium.

Biochemical analysis revealed significant differences across etiological categories, underscoring the contribution of metabolic derangements to altered sensorium. The overall mean random blood sugar was 140.18 ± 79.26 mg/dL, with markedly higher levels in metabolic etiologies (159.60 ± 129.33 mg/dL) ($p = 0.026$). Serum sodium showed a highly significant variation ($p < 0.001$), with metabolic etiologies demonstrating lower mean sodium levels (133.70 ± 9.80 mEq/L) compared with neurological and other causes, reflecting the role of dysnatremias. Serum potassium also differed significantly ($p = 0.026$), though absolute differences were modest. Arterial blood gas pH showed significant variation ($p = 0.007$), with lower mean pH values in toxic etiologies (7.303 ± 0.070), suggesting acid–base disturbances. Serum ammonia levels were highest in infective etiologies (97.40 ± 43.66 µmol/L) ($p = 0.035$). Kanich et al.[35] noted that routine laboratory tests individually had low diagnostic yield but emphasized their value when clinically targeted. The present findings support selective biochemical evaluation focused on metabolic and toxic causes as an essential component of altered sensorium assessment.

Neuroimaging demonstrated strong and statistically significant associations with etiological categories. All patients with hemorrhagic stroke, ischemic stroke, or mass lesions on NCCT brain (100% each) were classified under neurological etiology, establishing imaging as a definitive diagnostic tool ($p < 0.001$). Among patients with normal NCCT findings ($n = 103$), metabolic etiologies predominated (48.5%), followed by infective (19.4%) and neurological causes (17.5%), indicating that a normal scan increases the likelihood of systemic etiologies. MRI findings also showed a significant association ($p = 0.003$), with infective etiologies accounting for 80.0% of MRI-detected infections. These results align with the observations of Ginde et al. (2008), who reported widespread CT availability (96%) but more limited MRI access (66%), potentially influencing diagnostic yield across settings. Kanich et al. [35] emphasized that ancillary investigations should be guided by clinical suspicion rather than used indiscriminately. The present findings reinforce the value of targeted neuroimaging in confirming neurological and infective causes of altered sensorium.

The present study demonstrated substantial critical care utilization and adverse outcomes among patients with altered sensorium. ICU admission was required in 91.3% of patients and showed a statistically significant association with etiological category ($p = 0.031$), with 100% ICU admission in infective and other etiologies and over 90% in neurological and toxic causes. Ventilatory support was required in 23.3% of patients, though its distribution across etiologies was not statistically significant ($p = 0.701$). Mortality occurred in 21.3% of patients, with neurological etiologies accounting for 62.5% of deaths, followed by metabolic (21.9%) and infective causes (9.4%). This mortality rate is comparable to the 20.1% reported by Kekec et al. [36], where cerebrovascular disease was the leading cause of death 91. In contrast, Kanich et al. (2002) reported lower mortality (sssss9%) but still highlighted high admission rates and resource utilization 85. Delirium outcome studies similarly demonstrate worse prognosis and increased ICU use in patients with altered mentation, as shown by Kennedy et al.[38]. These findings underscore altered sensorium as a high-risk emergency presentation requiring aggressive monitoring and early etiological management.

Evaluation of comorbid conditions revealed no statistically significant association between most chronic illnesses and etiological categories of altered sensorium. Hypertension, present in 26.7% of patients, did not significantly influence etiological distribution ($p = 0.261$), with neurological causes predominating in both hypertensive (42.5%) and non-hypertensive (43.6%) groups. Diabetes mellitus, noted in 23.3%, similarly showed no significant association ($p = 0.576$), although neurological etiologies were more frequent among diabetic patients (54.3%) compared with non-diabetics (40.0%). Chronic kidney disease demonstrated a trend toward metabolic etiologies (50.0%) but lacked statistical significance ($p = 0.619$), while chronic liver disease showed higher neurological contribution (60.0%) without significant association ($p = 0.368$). These findings indicate that chronic comorbidities, while clinically important, do not independently determine etiological classification in altered sensorium. This observation aligns with Kanich et al. (2002), who emphasized that past medical history contributes to diagnostic reasoning but does not singularly predict etiology 85. Delirium literature similarly identifies comorbidities as risk modifiers rather than direct etiological determinants. Kennedy et al. [38] included dementia and prior stroke as risk factors for delirium but emphasized acute precipitants as primary drivers 90. The present findings reinforce that comorbidities should be interpreted in conjunction with acute clinical and biochemical findings rather than in isolation.

Renal and hepatic function status demonstrated variable but non-significant associations with etiological categories. Patients with impaired renal function showed a higher proportion of metabolic etiologies (40.0%), followed by neurological (36.0%) and infective causes (12.0%), whereas those with normal renal function demonstrated neurological predominance (47.0%) with metabolic causes accounting for 30.0%; the association was not statistically significant ($p =$

0.451). Similarly, impaired liver function was associated with neurological etiologies (45.3%) and metabolic causes (37.7%), while patients with normal liver function also showed neurological predominance (42.3%), without significant association ($p = 0.424$). These findings indicate that although renal and hepatic dysfunction contribute to metabolic encephalopathy, they do not independently define etiological categorization in altered sensorium presentations. Kanich et al. [38] reported renal and gastrointestinal causes to account for only 1% each of AMS etiologies, highlighting their relatively smaller contribution as primary diagnoses 85. Delirium-focused reviews emphasize that renal and hepatic dysfunction act as precipitating factors rather than standalone etiologies. Rieck et al. [41] stressed that identification and correction of underlying medical abnormalities, including renal and hepatic derangements, are essential for improving outcomes. The present findings underscore the supportive rather than defining role of organ dysfunction in etiological stratification.

A strong and statistically significant concordance was observed between definitive diagnoses and etiological categories. All patients with documented neurological diagnoses were classified under neurological etiology (100%; $p < 0.001$). Similarly, all patients with metabolic diagnoses, infective diagnoses, toxic diagnoses, and other miscellaneous diagnoses were classified under their corresponding etiological categories (100% each; $p < 0.001$). Among patients without neurological diagnosis, metabolic etiologies constituted 58.8%, followed by infective (23.5%) and toxic causes (11.8%). This high concordance validates the etiological classification framework used in the study. Comparable observations were reported by Kanich et al. (2002), who demonstrated that final discharge diagnoses largely reflected the dominant clinical and diagnostic findings established during evaluation. The importance of accurate diagnostic classification is further emphasized in delirium research, were misclassification and under-recognition lead to adverse outcomes. Han et al. [40] reported that 76% of delirium cases were unrecognized by emergency physicians, while Hustey and Meldon [39] documented explicit delirium recognition in only 17% of affected elderly patients. The present findings demonstrate that when definitive diagnoses are established, etiological classification is robust and clinically coherent.

Neurological examination parameters such as pupillary status and meningeal signs did not show statistically significant associations with etiological categories. Among patients with equal and reactive pupils, neurological etiology predominated (46.4%), followed by metabolic (32.7%) and infective causes (10.9%). In patients with non-reactive pupils, neurological and metabolic etiologies each accounted for 30.0%, with infective causes at 20.0%; however, overall association was not significant ($p = 0.391$). Similarly, meningeal signs did not demonstrate significant etiological association ($p = 0.984$), with neurological etiologies remaining predominant in both meningeal sign–present (39.1%) and absent (44.1%) groups. These findings indicate that while pupillary abnormalities and meningeal signs are critical for immediate clinical decision-making, they lack specificity for etiological differentiation when considered alone. Kanich et al. [35] noted that physical examination contributed 41% to diagnostic yield, reinforcing its value as part of a composite assessment rather than a sole determinant 85. Delirium phenomenology studies, such as Sharma et al. [42], also demonstrated that cognitive and attentional disturbances dominate presentations irrespective of focal neurological signs 94. The present findings support comprehensive neurological examination integrated with imaging and laboratory data.

Socio-contextual variables such as residence and occupation did not demonstrate statistically significant associations with etiological categories. Neurological etiologies were predominant among both rural (44.2%) and urban (41.8%) residents, with comparable distributions of metabolic and infective causes ($p = 0.832$). Occupational analysis showed neurological etiologies ranging from 35.7% in laborers to 50.0% in service/clerical workers, with metabolic causes also common across categories; however, no significant association was observed ($p = 0.423$). These findings suggest that altered sensorium presents with similar etiological patterns across residential and occupational strata once patients reach emergency care. Kanich et al, [35] similarly observed that AMS constituted a small but resource intensive fraction of ED visits across diverse patient populations 85. Imaging availability studies by Ginde et al. [43] highlighted that rural and smaller hospitals may face diagnostic resource limitations 88, yet the present findings indicate that etiological distribution remains broadly consistent across settings. The results underscore that socio-contextual factors influence access and exposure but do not independently dictate etiological patterns in altered sensorium, reinforcing the universality of this emergency presentation.

This study possesses several important strengths that enhance its clinical relevance and academic value. A relatively large sample size of 150 patients presenting with altered sensorium provided adequate representation of diverse etiological categories encountered in emergency settings. The systematic evaluation of patients using standardized clinical assessment tools, including detailed neurological examination, Glasgow Coma Scale scoring, comprehensive laboratory investigations, and neuroimaging, allowed for robust etiological classification. Inclusion of a wide range of clinical parameters demographic variables, vital signs, biochemical markers, radiological findings, and outcomes enabled multidimensional analysis of altered sensorium. The study design facilitated identification of statistically significant associations between etiological categories and key clinical variables such as age, vital parameters, biochemical derangements, neuroimaging findings, and ICU admission. Consistent documentation and uniform diagnostic criteria strengthened internal validity and minimized classification bias. The high proportion of critically ill patients provided valuable insights into ICU utilization, ventilatory support requirements, and mortality patterns. Furthermore, the study reflects real-world emergency department practice, thereby increasing its external applicability to similar tertiary care centers. Collectively, these strengths contribute to a comprehensive understanding of altered sensorium and reinforce the study's utility in guiding early diagnostic prioritization and resource allocation.

Limitations of this study: Despite its strengths, the study has certain limitations that should be acknowledged. Being conducted at a single tertiary care center, the findings may not be fully generalizable to primary or secondary healthcare

settings with different patient demographics or resource availability. The observational design limits causal inference between etiological factors and outcomes. Some etiological subgroups, such as toxic and other causes, had relatively small sample sizes, which may have reduced statistical power for subgroup analysis. The study focused on in-hospital outcomes, and long-term neurological or functional outcomes were not assessed. Potential referral bias may have contributed to the high proportion of severe cases requiring ICU admission. Additionally, although extensive investigations were performed, variability in timing of assessments and interventions could have influenced certain results. These limitations suggest that findings should be interpreted in the context of the study setting and design.

Future scope: Future research should focus on multicenter studies to improve generalizability and capture regional variations in etiological patterns of altered sensorium. Prospective studies incorporating standardized diagnostic algorithms may help determine the impact of early targeted interventions on outcomes. Long-term follow-up assessing neurological recovery, cognitive outcomes, and quality of life would provide valuable insights into the broader impact of altered sensorium. Integration of validated delirium screening tools and severity scoring systems could enhance early recognition, particularly in elderly patients. Future studies may also explore predictive models combining clinical, biochemical, and imaging variables to aid rapid risk stratification. Evaluation of cost-effectiveness of structured emergency department protocols could further support implementation at institutional levels. 109 Advances in point-of-care diagnostics and artificial intelligence assisted decision support systems may also play a role in improving early etiological identification. Overall, continued research is essential to refine management strategies and reduce morbidity and mortality associated with altered sensorium.

Conclusion

The present study demonstrates that altered sensorium is a common, clinically heterogeneous, and high-risk emergency presentation requiring prompt and systematic evaluation. Among the 150 patients studied, neurological causes were the predominant etiology (43.3%), followed by metabolic (33.3%), infective (13.3%), toxic (6.7%), and other causes (3.3%). The findings indicate that clinical assessment, Glasgow Coma Scale evaluation, vital parameters, targeted biochemical investigations, and appropriate neuroimaging are complementary and essential for identifying the underlying cause. Significant associations were observed between etiology and age, headache, GCS verbal response, vital parameters, biochemical abnormalities, and neuroimaging findings. In particular, neuroimaging was highly useful in identifying structural neurological causes, while biochemical evaluation helped recognize metabolic and toxic contributors. The study also highlights the substantial severity and resource requirements associated with altered sensorium. ICU admission was required in 91.3% of patients, ventilatory support in 23.3%, and the overall mortality was 21.3%. Neurological etiologies accounted for the largest proportion of deaths (62.5%), emphasizing the need for early recognition and aggressive management of potentially life-threatening neurological conditions. Overall, the study supports a structured, multidisciplinary, and etiology-directed approach to altered sensorium in the emergency department, with early stabilization followed by focused neurological examination, metabolic assessment, and appropriately selected neuroimaging. Early identification and correction of reversible causes, together with timely escalation of care, may help reduce complications and mortality. The findings also provide useful evidence for emergency-care prioritization and resource allocation in tertiary-care settings.

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Conflict of interest statement: The authors declare that they have no conflict of interest related to this study or its publication.

Ethical declarations: The study protocol was reviewed and approved by the Institutional Ethics Committee of Sri Siddhartha Medical College Hospital and Research Centre before commencement of the study. Written informed consent was obtained from all participants or their legally authorized representatives prior to enrolment. All procedures were conducted in accordance with the ethical principles of the Declaration of Helsinki and institutional guidelines. Participant confidentiality was maintained throughout the study, and no additional interventions beyond routine clinical management were performed.

Reference

[1] U. Budumuru et al., “A Study of the Etiology, Clinical Profile, and Outcome of Nontraumatic Cases of Impaired/Altered Sensorium in Patients Attending the Emergency Department in a South Indian Tertiary Care Hospital,” J. Assoc. Physicians India, vol. 73, no. 12, pp. 34–36, Dec. 2025, doi: 10.59556/japi.73.1278.

[2] S. N. Jali, S. N. Nayak, B. K. Alexander, D. Tripathy, and B. K. Behera, “Clinical and investigational study for the aetiological evaluation of patients in nontraumatic altered sensorium and its outcome,” Int. J. Res. Med. Sci., vol. 7, no. 4, p. 1113, Mar. 2019, doi: 10.18203/2320-6012.ijrms20191309.

[3] S. Kulkarni and M. A. Hadimani, “Bilateral Transverse Abdominis Plane Block as a Sole Anaesthetic Technique in a Patient with Peripartum Cardiomyopathy Posted for Emergency Caesarean Section,” Karnataka Anaesth. J., pp. 29–31, Apr. 2019, doi: 10.18311/kaj/2018/v16i1/141995.
[4] K. T. Kim and Y. W. Cho, “Altered Level of Consciousness in a Tertiary Emergency Department: Etiologies, Mortality, and Outcomes,” J. Clin. Med., vol. 15, no. 5, p. 2037, Mar. 2026, doi: 10.3390/jcm15052037.
[5] S. C. LaHue and V. C. Douglas, “Approach to Altered Mental Status and Inpatient Delirium,” Neurol. Clin., vol. 40, no. 1, pp. 45–57, Feb. 2022, doi: 10.1016/j.ncl.2021.08.004.
[6] N. D. Schiff, “Cognitive Motor Dissociation Following Severe Brain Injuries,” JAMA Neurol., vol. 72, no. 12, p. 1413, Dec. 2015, doi: 10.1001/jamaneurol.2015.2899.
[7] F. Hyder et al., “Uniform distributions of glucose oxidation and oxygen extraction in gray matter of normal human brain: No evidence of regional differences of aerobic glycolysis,” J. Cereb. Blood Flow Metab., vol. 36, no. 5, pp. 903–916, May 2016, doi: 10.1177/0271678X15625349.
[8] Manjunatha et al., “Insights into the Age-Dependent Variation in Nutrition-Related Trace Elements in Diabetes Blood Using Total Reflection X-Ray Fluorescence,” Biol. Trace Elem. Res., vol. 203, no. 3, pp. 1277–1286, Mar. 2025, doi: 10.1007/s12011-024-04253-x.
[9] S. Kulkarni and S. G S, “COMMON PERONEAL NEUROPATHY AFTER LITHOTOMY POSITION AND ITS TREATMENT MODALITIES,” J. Evol. Med. Dent. Sci., vol. 4, no. 51, pp. 8944–8946, Jun. 2015, doi: 10.14260/jemds/2015/1296.
[10] D. Arciniegas, Md, L. Adler, Md, J. Topkoff, E. Cawthra, Rn, C. M. Filley, Md, and M. Reite, Md, “Subject Review: Attention and memory dysfunction after traumatic brain injury: cholinergic mechanisms, sensory gating, and a hypothesis for further investigation,” Brain Inj., vol. 13, no. 1, pp. 1–13, Jan. 1999, doi: 10.1080/026990599121827.
[11] A. Karunakaran, W. M. Ilyas, S. F. Sheen, N. K. Jose, and Z. T. Nujum, “Risk factors of mortality among dengue patients admitted to a tertiary care setting in Kerala, India,” J. Infect. Public Health, vol. 7, no. 2, pp. 114–120, Mar. 2014, doi: 10.1016/j.jiph.2013.09.006.
[12] K. S. Bharathi, S. Kulkarni, K. S. Sadananda, and C. L. Gurudatt, “Takotsubo cardiomyopathy precipitated by negative pressure pulmonary oedema following total thyroidectomy,” Indian J. Anaesth., vol. 60, no. 3, pp. 202–205, Mar. 2016, doi: 10.4103/0019-5049.177872.
[13] R. A. Aziz et al., “Chemotherapy-induced cognitive impairment (CICI): Cisplatin’s effects on neurotransmitter regulation and oxidative stress in Drosophila melanogaster,” NeuroToxicology, vol. 112, p. 103365, Jan. 2026, doi: 10.1016/j.neuro.2025.103365.
[14] R. Hanumanthappa et al., “Recent advances in therapeutic strategies for Alzheimer’s and Parkinson’s disease using protein/peptide co-modified polymer nanoparticles,” Neuroprotection, p. nep3.60, Nov. 2024, doi: 10.1002/nep3.60.
[15] R. Hanumanthappa et al., “A Levodopa-Encapsulated Poly-ε-Caprolactone Nanocomposite Improves the Motor Symptoms and Neurochemical Changes in a Rotenone-Induced Mouse Model of Parkinson’s Disease,” ACS Omega, p. acsomega.5c00691, May 2025, doi: 10.1021/acsomega.5c00691.
[16] J. L. Saver et al., “Number Needed to Treat to Benefit and to Harm for Intravenous Tissue Plasminogen Activator Therapy in the 3- to 4.5-Hour Window: Joint Outcome Table Analysis of the ECASS 3 Trial,” Stroke, vol. 40, no. 7, pp. 2433–2437, Jul. 2009, doi: 10.1161/STROKEAHA.108.543561.
[17] D. Van De Beek, J. De Gans, A. R. Tunkel, and E. F. M. Wijdicks, “Community-Acquired Bacterial Meningitis in Adults,” N. Engl. J. Med., vol. 354, no. 1, pp. 44–53, Jan. 2006, doi: 10.1056/NEJMra052116.
[18] R. Sonneville et al., “Understanding brain dysfunction in sepsis,” Ann. Intensive Care, vol. 3, no. 1, p. 15, 2013, doi: 10.1186/2110-5820-3-15.
[19] J. S. Kass and R. V. Rose, “Criminal Culpability for Acts Committed During a Possible Episode of REM Sleep Behavior Disorder,” Continuum, vol. 26, no. 4, pp. 1070–1074, Aug. 2020, doi: 10.1212/CON.0000000000000875.
[20] G. K. Isbister and N. A. Buckley, “The Pathophysiology of Serotonin Toxicity in Animals and Humans: Implications for Diagnosis and Treatment,” Clin. Neuropharmacol., vol. 28, no. 5, pp. 205–214, Sep. 2005, doi: 10.1097/01.wnf.0000177642.89888.85.
[21] S. Arunachala et al., “Effectiveness of motivational interviewing for improvement of hand hygiene compliance and reduction of hospital acquired infection in intensive care unit,” Am. J. Infect. Control, vol. 54, no. 7, pp. 772–777, Jul. 2026, doi: 10.1016/j.ajic.2026.01.015.
[22] S. Kulkarni, C. L. Gurudatt, D. Prakash, and J. A. Mathew, “Effect of spinal flexion and extension in the lateral decubitus position on the unilaterality of spinal anesthesia using hyperbaric bupivacaine,” J. Anaesthesiol. Clin. Pharmacol., vol. 34, no. 4, pp. 524–528, 2018, doi: 10.4103/joacp.JOACP_99_17.
[23] S. Jayaram, S. Kulkarni, and R. Hegde, “Effect of oral clonidine premedication on the onset and duration of spinal anesthesia with hyperbaric bupivacaine,” J. Evol. Med. Dent. Sci., vol. 3, no. 46, pp. 11262–11271, 2014.
[24] S. C. LaHue and V. C. Douglas, “Approach to Altered Mental Status and Inpatient Delirium,” Neurol. Clin., vol. 40, no. 1, pp. 45–57, Feb. 2022, doi: 10.1016/j.ncl.2021.08.004.
[25] K. P. Hill, “Medical Use of Cannabis in 2019,” JAMA, vol. 322, no. 10, p. 974, Sep. 2019, doi: 10.1001/jama.2019.11868.
[26] S. C. LaHue and V. C. Douglas, “Approach to Altered Mental Status and Inpatient Delirium,” Neurol. Clin., vol. 40, no. 1, pp. 45–57, Feb. 2022, doi: 10.1016/j.ncl.2021.08.004.

[27] A. T. Smith and J. H. Han, “Altered Mental Status in the Emergency Department,” in Neurologic Emergencies, L. Ganti and J. N. Goldstein, Eds., Cham: Springer International Publishing, 2018, pp. 209–232. doi: 10.1007/978-3-319-64523-0_11.
[28] J. A. Edlow and L. R. Caplan, “Avoiding Pitfalls in the Diagnosis of Subarachnoid Hemorrhage,” N. Engl. J. Med., vol. 342, no. 1, pp. 29–36, Jan. 2000, doi: 10.1056/NEJM200001063420106.
[29] D. Chilakabathina, J. K. J. Shrinivas Kulkarni, R. Ravikumara, and J. Jose, “Diagnostic Utility of B-Lines in Lung Ultrasound for Differentiating Cardiac and Pulmonary Causes of Acute Dyspnea,” Eur. J. Cardiovasc. Med., vol. 15, pp. 10–15, 2025.
[30] Sudhir, Avinash Hr, and Yashawant Ns, “Study of etiology, clinical profile, and outcome of altered sensorium patients admitted in a tertiary care hospital,” Asian J. Med. Sci., vol. 15, no. 10, pp. 93–99, Oct. 2024, doi: 10.71152/ajms.v15i10.4224.
[31] S. Arunachala et al., “Factors Affecting Survival in Severe and Very Severe COPD after Admission in ICUs of Tertiary Care Centers of India (FAST COPD): Study Protocol for a Multicentric Cohort Study,” Indian J. Crit. Care Med. Peer-Rev. Off. Publ. Indian Soc. Crit. Care Med., vol. 28, no. 6, pp. 552–560, Jun. 2024, doi: 10.5005/jp-journals-10071-24728.
[32] U. Budumuru et al., “A Study of the Etiology, Clinical Profile, and Outcome of Nontraumatic Cases of Impaired/Altered Sensorium in Patients Attending the Emergency Department in a South Indian Tertiary Care Hospital,” J. Assoc. Physicians India, vol. 73, no. 12, pp. 34–36, Dec. 2025, doi: 10.59556/japi.73.1278.
[33] J. Jose, S. Kulkarni, S. Hussain, R. R, and J. K. Jogannachar, “ACCURACY OF S REMS AS COMPARED TO RAPS IN PREDICTING THE PROGNOSIS IN NON-SURGICAL PATIENTS PRESENTING TO EMERGENCY DEPARTMENT IN A TERTIARY CARE HOSPITAL,” Genet. Mol. Res., vol. 25, no. 14 (S), Dec. 2026, doi: https://doi.org/10.4238/5d8mng66.
[34] S. Arunachala and J. Kumar, “mNUTRIC Score in ICU Mortality Prediction: An Emerging Frontier or Yet Another Transient Trend?,” Indian J. Crit. Care Med. Peer-Rev. Off. Publ. Indian Soc. Crit. Care Med., vol. 28, no. 5, pp. 422–423, May 2024, doi: 10.5005/jp-journals-10071-24713.
[35] W. Kanich et al., “Altered mental status: Evaluation and etiology in the ED,” Am. J. Emerg. Med., vol. 20, no. 7, pp. 613–617, Nov. 2002, doi: 10.1053/ajem.2002.35464.
[36] Z. Kekec, V. Senol, F. Koc, and G. Seydaoglu, “Analysis of Altered Mental Status in Turkey,” Int. J. Neurosci., vol. 118, no. 5, pp. 609–617, Jan. 2008, doi: 10.1080/00207450701849133.
[37] J. H. Han et al., “Diagnosing Delirium in Older Emergency Department Patients: Validity and Reliability of the Delirium Triage Screen and the Brief Confusion Assessment Method,” Ann. Emerg. Med., vol. 62, no. 5, pp. 457–465, Nov. 2013, doi: 10.1016/j.annemergmed.2013.05.003.
[38] M. Kennedy, R. A. Enander, S. P. Tadiri, R. E. Wolfe, N. I. Shapiro, and E. R. Marcantonio, “Delirium Risk Prediction, Healthcare Use and Mortality of Elderly Adults in the Emergency Department,” J. Am. Geriatr. Soc., vol. 62, no. 3, pp. 462–469, Mar. 2014, doi: 10.1111/jgs.12692.
[39] F. M. Hustey and S. W. Meldon, “The prevalence and documentation of impaired mental status in elderly emergency department patients,” Ann. Emerg. Med., vol. 39, no. 3, pp. 248–253, Mar. 2002, doi: 10.1067/mem.2002.122057.
[40] J. H. Han et al., “Diagnosing Delirium in Older Emergency Department Patients: Validity and Reliability of the Delirium Triage Screen and the Brief Confusion Assessment Method,” Ann. Emerg. Med., vol. 62, no. 5, pp. 457–465, Nov. 2013, doi: 10.1016/j.annemergmed.2013.05.003.
[41] K. M. Rieck, S. Pagali, and D. M. Miller, “Delirium in hospitalized older adults,” Hosp. Pract., vol. 48, no. sup1, pp. 3–16, Mar. 2020, doi: 10.1080/21548331.2019.1709359.
[42] A. Sharma, S. Malhotra, S. Grover, and S. K. Jindal, “Symptom profile as assessed on delirium rating scale-revised-98 of delirium in respiratory intensive care unit: A study from India,” Lung India Off. Organ Indian Chest Soc., vol. 34, no. 5, pp. 434–440, 2017, doi: 10.4103/lungindia.lungindia_416_14.
[43] A. A. Ginde, A. Foianini, D. M. Renner, M. Valley, and C. A. Camargo, Jr, “Availability and Quality of Computed Tomography and Magnetic Resonance Imaging Equipment in U.S. Emergency Departments,” Acad. Emerg. Med., vol. 15, no. 8, pp. 780–783, Aug. 2008, doi: 10.1111/j.1553-2712.2008.00192.x.