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ORIGINAL ARTICLE

Etiology, Clinical Features, and Outcomes of Coma Patients in Tertiary Care Settings

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ABSTRACT:
Background: Coma is a medical emergency with diverse etiologies ranging from structural brain injury to systemic and metabolic disturbances. Early identification of the cause is crucial for optimizing outcomes. **Aim:** To study the physical traits, prognoses, and etiology of coma patients admitted to a tertiary care hospital. **Material and Methods:** This cross-sectional study included 80 patients presenting with coma (GCS ≤8) over a 12-month period. Demographic data, comorbidities, clinical features, and etiological diagnoses were recorded. Investigations included routine laboratory tests, neuroimaging, and specialized studies as indicated. Outcomes were assessed at discharge. Data were analyzed using descriptive statistics and inferential tests with significance set at $p<0.05$. **Results:** Males constituted 55% of cases. Hypertension (40%) and diabetes (35%) were the most common comorbidities. Cerebrovascular events (45%) were the leading cause of coma, followed by metabolic encephalopathy (31.7%) and infective etiologies (11.7%). Status epilepticus, toxicological exposures, and other causes were less frequent. Vascular and metabolic causes together accounted for over three-quarters of cases. **Conclusion:** Cerebrovascular and metabolic etiologies dominate the spectrum of coma in tertiary care settings, often in the presence of vascular and metabolic comorbidities. Early diagnosis, aggressive management of reversible causes, and targeted intervention are key to improving neurological recovery and survival.
Keywords: Coma, Cerebrovascular events, Metabolic encephalopathy, Prognosis

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INTRODUCTION

Coma is a profound state of unconsciousness in which a patient is unresponsive to external stimuli and cannot be awakened, representing a medical emergency requiring immediate evaluation and management [1]. It is a manifestation of widespread brain dysfunction and may result from a wide range of etiologies, including traumatic brain injury, cerebrovascular events, metabolic disturbances, central nervous system infections, and toxic exposures [2]. Globally, coma accounts for a substantial proportion of intensive care admissions, with studies suggesting that up to 3–15% of emergency presentations in tertiary hospitals involve altered consciousness states [3].

The pathophysiology of coma varies according to its etiology but generally involves disruption of the reticular activating system in the brainstem or bilateral hemispheric dysfunction [4]. Advances in neuroimaging, laboratory diagnostics, and neurocritical care have significantly improved the ability to identify underlying causes promptly, allowing for targeted therapeutic interventions [5]. However, despite these developments, the prognosis of coma remains highly variable, influenced by factors such as etiology, age, duration of unconsciousness, neurological examination findings, and comorbid conditions [6].

Clinical evaluation of a comatose patient involves detailed neurological examination, often quantified using the Glasgow Coma Scale (GCS), which is widely used for initial assessment and prognostication [7]. The integration of GCS with neuroimaging findings, brainstem reflex testing, and electrophysiological studies enhances the accuracy of outcome predictions [8]. Recent literature has emphasized that non-traumatic causes of coma, such as hypoxic-ischemic encephalopathy, stroke, or metabolic derangements, may be associated with poorer functional outcomes compared to traumatic causes, despite similar initial severity scores [9].

In low- and middle-income countries, including India, resource limitations, delayed presentation, and inadequate pre-hospital care can further influence the prognosis of coma patients [10]. These challenges underscore the importance of comprehensive studies focusing on the etiology, clinical profile, and outcomes of coma in tertiary care settings to aid in better planning of critical care resources and improve patient outcomes.

The present study aims to evaluate the physical traits, etiologies, and prognoses of coma patients admitted to a tertiary care hospital, thereby providing insights that could guide timely diagnosis, optimize management strategies, and inform prognostic counseling for families.

MATERIAL AND METHODS

This observational cross-sectional study was conducted in the Department of Emergency Medicine and Neurocritical Care at a tertiary care hospital over a period of twelve months. A total of 120 patients presenting in a comatose state were included in the study. Coma was defined as a state of unarousable unresponsiveness, confirmed clinically by the absence of purposeful responses to external stimuli, with a Glasgow Coma Scale (GCS) score of ≤8 at presentation. Patients of all ages and both sexes were eligible for inclusion. Exclusion criteria comprised patients with transient loss of consciousness resolving within 30 minutes, those with brain death confirmed on arrival, and cases where consent could not be obtained from relatives.

On admission, a detailed history was obtained from relatives or caregivers regarding the onset, progression, and any precipitating events related to the coma. Particular attention was given to prior medical illnesses, history of trauma, exposure to toxins or drugs, and the presence of fever, seizures, or focal neurological symptoms. A thorough physical examination was conducted with emphasis on neurological assessment, including GCS scoring, cranial nerve examination, motor and sensory evaluation, and the presence of brainstem reflexes. Systemic examination findings were also recorded to identify evidence of metabolic, infectious, or systemic causes.

All patients underwent baseline investigations, including complete blood count, renal and liver function tests, serum electrolytes, random blood sugar, arterial blood gases, and, where indicated, toxicology screening. Neuroimaging was performed in all cases, with computed tomography (CT) scans obtained as the initial imaging modality. Magnetic resonance imaging (MRI) was performed in selected patients where additional anatomical or etiological clarification was required. Lumbar puncture was carried out in suspected cases of meningitis or encephalitis, after excluding raised intracranial pressure by imaging. Additional diagnostic studies, including electroencephalography (EEG) and carotid Doppler, were performed where clinically indicated.

Patients were managed according to established institutional protocols based on the identified cause of coma, with supportive care provided in the intensive care unit as required. Prognosis was assessed based on clinical improvement in GCS during hospitalization, survival at discharge, and neurological outcomes classified as full recovery, partial recovery with neurological deficits, persistent vegetative state, or death. Data were compiled in a structured proforma, and statistical analysis was performed using SPSS version 25.0. Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. Chi-square tests and t-tests were used to assess associations between clinical characteristics, etiology, and prognosis, with p-values <0.05 considered statistically significant.

RESULTS

As shown in **Table 1**, among the 120 coma patients included in the study, males constituted the majority with 66 cases (55%), while females accounted for 54 cases (45%). This distribution reflects a slightly higher prevalence of coma in male patients in the present cohort.

In **Table 2**, the distribution of comorbidities among the patients is presented. Hypertension was the most common comorbidity, seen in 48 patients, followed closely by type 2 diabetes mellitus in 42 patients. Chronic kidney disease was documented in 18 patients, while a history of cardiovascular accident was found in 14 cases. Coronary artery disease was present in 10 patients, whereas systemic lupus erythematosus was reported in 6 patients. There were no documented cases of glioma or tuberculoma in the study population. These findings suggest that vascular and metabolic comorbidities are predominant in coma patients in this setting.

Table 3 summarizes the etiological distribution of coma in the study. Cerebrovascular events accounted for the largest proportion of cases with 54 patients (45%), of which 32 (26.7%) were hemorrhagic strokes and 22 (18.3%) were ischemic strokes. Metabolic encephalopathy was the second most frequent cause, seen in 38 patients (31.7%), and included hypoxemic ischemic encephalopathy in 8 (6.7%), hyponatremia in 10 (8.3%), hypertensive encephalopathy in 3 (2.5%), hypoglycemic encephalopathy in 2 (1.7%), hyperglycemic encephalopathy in 2 (1.7%), and diabetic ketoacidosis in 5 (4.2%). Infective etiologies were identified in 14 patients (11.7%), comprising septic encephalopathy in 8 (6.7%) and meningoencephalitis in 6 (5%). Status epilepticus was responsible for coma in 8 patients (6.7%), organophosphorus poisoning in 4 (3.3%), and alcoholic intoxication in 2 (1.7%). These results indicate that vascular and metabolic causes together accounted for over three-quarters of all coma presentations in the present study.

Table 1: Gender distribution (N=120)

Gender	No. of patients	Percentage
Male	66	55.0%
Female	54	45.0%

Table 2: Total number of patients with comorbidities (N=120)

S. No	Comorbidities	No. of patients
1	Chronic kidney disease (CKD)	18
2	Systemic lupus erythematosus (SLE)	6
3	Type 2 Diabetes mellitus	42
4	Hypertension	48
5	Cardiovascular accident	14
6	Coronary artery disease	10
7	Glioma	0
8	Tuberculoma	0

Table 3: Patients with various coma aetiologies (N=120)

S. No	Etiology for poor GCS	No. of patients	Percentage
1	Cerebrovascular events	54	45.0%
1a	Hemorrhagic stroke	32	26.7%
1b	Ischemic stroke	22	18.3%
2	Metabolic encephalopathy	38	31.7%
2a	Hypoxemic Ischemic encephalopathy	8	6.7%
2b	Hyponatremia	10	8.3%
2c	Hypertensive encephalopathy	3	2.5%
2d	Hypoglycemic encephalopathy	2	1.7%
2e	Hyperglycemic encephalopathy	2	1.7%
2f	Diabetic Ketoacidosis	5	4.2%
3	Infective etiology	14	11.7%
3a	Septic encephalopathy	8	6.7%
3b	Meningoencephalitis	6	5.0%
4	Status epilepticus	8	6.7%
5	Alcoholic intoxication	2	1.7%
6	Organophosphorus poisoning	4	3.3%
7	Total	120	100.0%

DISCUSSION

The findings of this study demonstrate that cerebrovascular events and metabolic encephalopathy together accounted for the majority of coma cases in a tertiary care setting, consistent with recent global trends. Several recent investigations have emphasized that cerebrovascular diseases, particularly hemorrhagic stroke, are associated with higher initial severity of coma and poorer prognosis when compared to ischemic stroke, largely due to elevated intracranial pressure and extensive neuronal injury [11]. The prominence of vascular causes in our cohort reflects the high prevalence of hypertension and diabetes as comorbidities, both of which are well-established risk factors for stroke-related coma [12].

Metabolic encephalopathy emerged as the second most frequent etiology in our study, with hyponatremia, hypoxemic ischemic encephalopathy, and diabetic complications being notable contributors. Recent literature highlights that electrolyte imbalances and metabolic derangements can rapidly precipitate or worsen coma states, particularly in critically ill patients, and that early correction of these abnormalities is strongly linked to improved neurological recovery [13]. The high proportion of metabolic causes observed here may also be related to the prevalence of uncontrolled diabetes and chronic kidney disease in the population, conditions that predispose patients to metabolic instability.

Infective etiologies, though less common than vascular or metabolic causes, remain significant in regions where sepsis and central nervous system infections are prevalent. Studies have shown that septic encephalopathy and meningoencephalitis contribute to higher mortality rates in comatose patients, especially when diagnosis and antimicrobial therapy are delayed [14]. This underscores the need for prompt identification of infectious causes in comatose patients to improve outcomes.

Non-structural causes such as status epilepticus and toxicological exposures (alcoholic intoxication and organophosphorus poisoning) also featured in our findings. Contemporary reports indicate that although these causes are less frequent, they can be rapidly reversible with timely intervention, resulting in better long-term outcomes compared to structural brain lesions [15]. This highlights the importance of comprehensive diagnostic evaluation in coma patients to identify potentially reversible etiologies.

Our results align with the growing body of evidence suggesting that prognosis in coma is multifactorial, influenced not only by etiology but also by age, baseline neurological function, and the presence of systemic comorbidities. This reinforces the role of individualized management strategies and the

integration of rapid diagnostic protocols in tertiary care centers to optimize outcomes in comatose patients.

CONCLUSION

This study reveals that cerebrovascular events and metabolic encephalopathy are the predominant causes of coma in the tertiary care setting, with hypertension and diabetes as the leading comorbidities. Infective, epileptic, and toxicological causes, though less common, remain clinically important due to their potential reversibility. The findings emphasize the necessity for early etiological diagnosis, aggressive management of comorbid conditions, and targeted therapeutic interventions to improve survival and neurological recovery in coma patients.

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