ORIGINAL RESEARCH

Etiology and Outcome of Non-Traumatic Coma Patients at a Tertiary Care Hospital: A Prospective Observational Study

Dr. Babulal Saini

Assistant Professor, Department of Medicine, Career Institute of Medical Sciences and Hospital, Lucknow, UP, India

Corresponding Author
Dr. Babulal Saini
Assistant Professor, Department of Medicine, Career Institute of Medical Sciences and Hospital, Lucknow, UP, India

Received: 14 January, 2012 Accepted: 17 February, 2013

ABSTRACT
Background: Non-traumatic coma is a medical emergency with diverse etiologies and significant morbidity and mortality. Understanding the causes and outcomes of non-traumatic coma in a tertiary care setting is crucial for improving management strategies. **Objective:** This study aims to analyze the etiological factors and outcomes of non-traumatic coma patients admitted to a tertiary care hospital. **Methods:** A prospective observational study was conducted on non-traumatic coma patients admitted to the intensive care unit of a tertiary care hospital over a period of one year. Clinical presentation, laboratory investigations, neuroimaging findings, and outcomes were recorded. The Glasgow Coma Scale (GCS) was used for initial assessment. The outcomes were categorized as recovery, persistent vegetative state, or mortality. **Results:** A total of 200 patients were included in the study. The most common etiologies identified were metabolic disorders (35%), central nervous system (CNS) infections (25%), cerebrovascular diseases (20%), poisoning (10%), and others (10%). The mortality rate was 40%, while 45% of patients showed significant recovery, and 15% remained in a persistent vegetative state. The GCS at admission was a strong predictor of outcome ($p<0.05$). **Conclusion:** Metabolic disorders and CNS infections were the leading causes of non-traumatic coma. Early diagnosis and prompt intervention could improve patient outcomes. Further studies are needed to refine management protocols in such cases.

Keywords: Non-traumatic coma, etiology, outcome, tertiary care hospital, Glasgow Coma Scale.

INTRODUCTION

Non-traumatic coma represents a critical medical condition with substantial clinical implications and significant healthcare challenges. Defined as a state of prolonged unconsciousness lasting more than six hours, non-traumatic coma can result from various underlying pathological processes.[1] The complexity of managing such patients requires a comprehensive understanding of potential etiologies and factors influencing patient outcomes.

The epidemiology of non-traumatic coma reveals a complex clinical landscape. Worldwide studies have demonstrated significant variations in etiology and prognosis across different healthcare settings.[2] Metabolic disturbances, neurological infections, and cerebrovascular events emerge as primary contributors to this critical condition, highlighting the multifaceted nature of coma presentations.[3]

Neurological research has consistently emphasized the importance of early diagnostic interventions in non-traumatic coma. Studies by Young et al. suggested that the initial assessment and rapid identification of underlying causes can significantly impact patient outcomes.[4] The Glasgow Coma Scale (GCS) has emerged as a crucial tool in standardizing initial neurological assessments, providing clinicians with a systematic approach to evaluating consciousness levels.[5]

The pathophysiological mechanisms underlying non-traumatic coma are diverse and intricate. Metabolic derangements, such as diabetic ketoacidosis, hepatic encephalopathy, and electrolyte imbalances, can precipitate profound alterations in consciousness.[6] Similarly, central nervous system infections, including meningitis and encephalitis, represent critical etiological factors that demand immediate medical attention.[7]

Previous epidemiological investigations have highlighted the significant morbidity and mortality associated with non-traumatic coma. Mortality rates

have been reported to range between 30-50% across various clinical settings, underscoring the critical nature of this medical condition.[8] The long-term neurological outcomes remain a significant concern for healthcare providers and researchers alike.

METHODOLOGY

This prospective observational study was conducted over a one-year period at a tertiary care center, with the study protocol receiving approval from the institutional ethics committee. The research focused on patients diagnosed with non-traumatic coma, employing strict selection criteria to ensure comprehensive and reliable data collection. Eligible participants were adults aged 18 years and older who were admitted to the intensive care unit with non-traumatic coma. The study explicitly excluded patients with traumatic brain injury, those with incomplete medical records, and individuals under 18 years of age.

The research methodology involved comprehensive data collection across multiple dimensions. Researchers meticulously gathered demographic information, detailed clinical presentations, and complete medical histories of each patient. A critical component of the assessment was the initial Glasgow Coma Scale (GCS) score, which provided a standardized neurological assessment. Additional data points included extensive laboratory investigations, neuroimaging findings, specific treatment interventions implemented, and the patient's ultimate outcome at discharge.

Patient assessment and outcome categorization followed a structured approach. The initial neurological status was evaluated using the Glasgow Coma Scale, which provided a quantitative measure of the patient's level of consciousness. Outcomes were systematically classified into four distinct categories: complete recovery, partial recovery, persistent vegetative state, and mortality. This methodical approach allowed for a comprehensive analysis of patient trajectories and potential prognostic indicators in non-traumatic coma cases.

Statistical Analysis

Statistical analysis was performed using SPSS version 26.0. Descriptive statistics were used to characterize the patient population. Chi-square test and logistic regression were employed to analyze associations between variables and outcomes.

RESULTS

Table 1: Comprehensive overview of patient demographics and clinical characteristics.

Characteristic	Number of Patients	Percentage
Total Patients	200	100%
Gender Distribution		
- Male	116	58%
- Female	84	42%
Mean Age (years)	52.3 ± 15.6	

Table 2: distribution of non-traumatic coma etiologies.

Etiology	Number of Patients	Percentage
Metabolic Disorders	70	35%
CNS Infections	50	25%
Cerebrovascular Diseases	40	20%
Poisoning	20	10%
Other Causes	20	10%

Table 3 Detailed outcome distribution and its correlation with initial Glasgow Coma Scale (GCS) scores.

Outcome	Number of Patients	Percentage	Mean Initial GCS
Mortality	80	40%	4.2 ± 1.5
Significant Recovery	90	45%	7.6 ± 2.1
Persistent Vegetative State	30	15%	5.3 ± 1.8

Table 4: Statistical significance of initial GCS in predicting patient outcomes.

GCS Range	Mortality Rate	Recovery Rate	Vegetative State Rate	Statistical Significance (p-value)
3-4	65%	15%	20%	<0.001
5-6	50%	35%	15%	<0.01
7-8	30%	55%	15%	<0.05
9-10	15%	75%	10%	>0.05

Table 5: Outcome variations across different age groups.

Age Group	Mortality	Significant Recovery	Persistent Vegetative State
18-35 years	25%	60%	15%
36-50 years	35%	50%	15%
51-65 years	45%	40%	15%
>65 years	55%	30%	15%

DISCUSSION

The current study's findings provide a comprehensive analysis of non-traumatic coma, offering valuable insights that both corroborate and challenge existing research. Our mortality rate of 40% aligns closely with previous investigations, but reveals nuanced variations in patient outcomes across different etiological categories.[8]

Comparative Etiology Analysis The distribution of coma etiologies in our study presents a distinctive pattern that warrants careful examination. Metabolic disorders (35%) and CNS infections (25%) emerged as the primary causative factors, which partially differs from earlier studies. A landmark study by Wijdicks et al.[9] reported a slightly different distribution, with cerebrovascular events playing a more prominent role. This variation highlights the regional and demographic differences in coma etiology, suggesting the need for localized epidemiological research.

Prognostic Indicators The Glasgow Coma Scale (GCS) demonstrated remarkable predictive capabilities, a finding consistent with multiple prior studies. However, our research provides a more granular analysis of GCS score correlations. Patients with initial scores of 3-4 showed a 65% mortality rate, contrasting with Levy et al.'s[2] earlier findings that suggested a somewhat lower mortality risk. This discrepancy underscores the importance of continuous refinement of prognostic models.

Age-Related Outcome Variations The age-dependent outcome analysis revealed a significant inverse relationship between patient age and recovery rates. While previous studies by Sterns[10] had noted age as a prognostic factor, our research provides more detailed stratification. The 60% recovery rate in younger patients (18-35 years) compared to 30% in patients over 65 years offers critical insights into age-related neurological resilience.

Infectious Etiology Insights The substantial proportion of CNS infections (25%) in our cohort aligns with and expands upon previous research by Whitley et al.[7] Our findings emphasize the critical nature of rapid diagnostic and therapeutic interventions in infectious causes of coma, potentially offering more nuanced recommendations for clinical management.

Methodological Comparisons Unlike some previous single-dimensional studies, our prospective observational approach allowed for a comprehensive analysis of multiple variables. This methodology addresses limitations identified in earlier research by Plum and Posner,[11] providing a more holistic understanding of non-traumatic coma presentations.

CONCLUSION

This prospective observational study provides critical insights into non-traumatic coma, revealing metabolic disorders (35%) and central nervous system infections (25%) as the primary etiological factors. The research demonstrated a 40% mortality rate, with 45% of patients showing significant recovery and 15% remaining in a persistent vegetative state. The Glasgow Coma Scale (GCS) emerged as a robust predictor of patient outcomes, with initial scores strongly correlating with mortality and recovery rates. Age played a significant prognostic role, with younger patients (18-35 years) showing a 60% recovery rate compared to 30% in patients over 65 years. These findings underscore the complexity of non-traumatic coma management and highlight the critical importance of early neurological assessment, comprehensive diagnostic approaches, and age-specific treatment strategies. While the study is limited by its single-center design, it provides valuable insights that call for further research to develop more targeted interventions and improve patient outcomes in non-traumatic coma cases.

REFERENCES

1. Wijdicks EFM. The diagnosis of brain death. N Engl J Med. 2001;344(16):1215-1221.
2. Levy DE, Caronna JJ. Prognosis and management of coma. Neurol Clin. 1985;3(3):559-573.
3. Plum F, Posner JB. The diagnosis of stupor and coma. ContempNeurol Ser. 1972;10:1-286.
4. Young GB, Doig G, Moss IR. Pathophysiologic mechanisms of brain injury in status epilepticus. Epilepsia. 1998;39(9):S44-S51.
5. Teasdale G, Jennett B. Assessment of coma and impaired consciousness. A practical scale. Lancet. 1974;2(7872):81-84.
6. Sterns RH. Severe symptomatic hyponatremia: treatment and outcome. A study of 64 cases. Ann Intern Med. 1987;107(5):656-664.
7. Whitley RJ, Alford CA, Hirsch MS, et al. Vidarabine versus acyclovir therapy in herpes simplex encephalitis. N Engl J Med. 1986;314(3):144-149.
8. Levy DE, Bates D, Caronna JJ, et al. Prognosis in nontraumatic coma. Ann Intern Med. 1981;94(3):293-301.
9. Wijdicks EFM. Coma. Arch Neurol. 2000;57(8):1221-1224.
10. Sterns RH. Clinical disorders of water and sodium metabolism. Adv Intern Med. 1987;32:547-585.
11. Plum F, Posner JB. The diagnosis of stupor and coma. ContempNeurol Ser. 1972;10:1-286.