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Research Article

A STUDY OF INCIDENCE, ETIOLOGICAL FACTORS, CLINICAL PROFILE AND OUTCOME AMONG PATIENTS WITH METABOLIC ENCEPHALOPATHY

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Abstract:

Background: One of the most common causes of morbidity and death in intensive care units is metabolic encephalopathy, especially in patients who are elderly, immunocompromised, severely ill or have concomitant diseases. Therefore, we aim to investigate the prevalence, etiology, clinical characteristics, and outcome of metabolic encephalopathy patients admitted to tertiary care institution during the study period.

Materials and methods: There were 97 patients in this two-year prospective and observational research carried out in a Karnataka tertiary care facility. According to clinical suspicion, all patients underwent pertinent laboratory investigations such as CBC (Complete blood count), LFT (Liver function test) with Sr. ammonia RFT (Renal function test), RBS (Random blood sugar), CSF (Cerebrospinal fluid) analysis, brain MRI (Magnetic resonance imaging) and also, GCS (Glasgow coma scale) were used to study the incidence, various etiological factors, and clinical presentation of metabolic encephalopathy. Additionally, the results of different etiologies were examined in terms of length of hospital stay, morbidity, and mortality. All the data was analyzed using SPSS Software (Version 26).

Results: The mean age of study participants was 53.10 ± 13.21 years. Out of 97 patients, the highest frequency of encephalopathy was seen in those aged 51 to 60 old 29 (29.9%). Notably, the incidence was 1 (1%) in those under 20 years old and 3 (3.1%) in those between 71 - 80 years old. Of the 97 patients, 23 (23.7%) were female and 74 (76.3%) were male. Insidious onset occurred in 78 (80.4%) of patients, while abrupt onset occurred in 19 (19.6%) of patients. Fever accounted for 18 (18.6%) of patient presentations, followed by headaches 38 (39.2%), vomiting 47 (48.5%), convulsions 24 (24.7%), localized impairments 2 (2.1%), and altered

behavior 50 (51.5%), which was the most common mode of presentation seen in our patients. 10 (10.3%) of patients had loose stools, 25 (25.8%) experienced abdominal distension, 24 (24.7%) had jaundice, 43 (44.3%) experienced leg swelling, 24 (24.7%) had decreased urine production, and 25 (25.8%) had dyspnea. The majority of patients had a GCS of 9-12, while only 23 (23.75%) had a GCS of ≤8. Only 2 (2.06%) of patients needed to stay in the hospital for more than 15 days, compared to 12 (12.37%) who needed for less than 6 days and 83 (85.56%) of patients who needed for 7-14 days. After examining the causes of encephalopathy, hepatic encephalopathy was seen in 21 (21.6%) of cases, which was the most common. 16 (16%) of patients had hypoglycemia, 15 (15.5%) had uremia, 10 (10.3%) had hyponatremia, 8 (8.2%) had alcoholic withdrawal, 8 (8.2%) had hypercapnic encephalopathy, 7 (7.2%) had hyperglycemic hyperosmolar coma, 7 (7.2%) had hypertensive encephalopathy, and only 5.2% had septic encephalopathy.

Conclusion: We found that hepatic encephalopathy is a common cause of altered behavior and that metabolic encephalopathy often affects the elderly. In order to improve patient treatment, more multicentre research with a larger sample size enhances the early and accurate etiological identification of acute confusional episodes.

Keywords: CSF; Immunocompromise; Metabolic encephalopathy; MRI; Comorbidities; Altered sensorium

INTRODUCTION

Normal brain function depends on neuronal metabolism, which is connected to the systemic homeostasis of metabolites like glucose, electrolytes, amino acids, and ammonia. Failure of any organ system, including the kidneys, liver, lungs, respiratory system, heart, and circulatory system, results in a general reduction in brain function. We refer to the secondary brain impairment resulting from these systems' failures as "metabolic encephalopathy [1]. Metabolic encephalopathy is a prevalent occurrence in the intensive care unit setting. Wernicke encephalopathy, sepsis, renal failure, hepatic failure, and electrolyte imbalance are common causes [2]. Encephalopathies are recognized as the most frequent consequence of a broad range of illnesses managed in the intensive care unit; however, the onset of metabolic encephalopathy may serve as an indicator of organ failure or decline. Internal medicine research on the reversibility of metabolic encephalopathy is linked to effective treatment of systemic dysfunction [3]. There are two primary types of metabolic encephalopathies: those brought on by malfunctioning peripheral organs and those brought on by insufficient amounts of oxygen, glucose, or metabolic cofactors (which are primarily derived from vitamins) [4]. Hypoxic-ischemic conditions, various organ dysfunctions, systemic diseases, and toxic agents are the causes of metabolic encephalopathies. The most common exogenous toxic agent is alcohol [5].

Age-related encephalopathy is suggested by epidemiological and demographic data. After 65 years of age, the number of patients with encephalopathy rises. Individuals living in nursing homes who are over 75 years old have a 60% risk of developing encephalopathy; in people under 55, this risk is 1.1% [6]. 10-40% of hospitalized patients over 65 experience encephalopathy, while 8-70% experience septic encephalopathy. Depending on the degree of liver damage, Hepatic encephalopathy is a decline in brain function that occurs as a result of severe liver disease and occurs in 45–80% of cirrhosis patients [7]. The most prevalent kind of ME (Metabolic encephalopathy), septic encephalopathy, affects up to 70% of patients with bacteremia. Failure of the body to eliminate harmful nitrogenous waste products results in uremic

encephalopathy. If left untreated, severe electrolyte imbalances like hypo- or hypernatremia, hypocalcemia, hypercalcemia, hypomagnesemia, and hypophosphatemia can lead to ME. Other rather common causes of ME include post-transplantation encephalopathy and hypoxic-ischemic encephalopathy. The outcome of encephalopathy in adults is variable, ranging from complete recovery to permanent brain damage or death and depends on early diagnosis and appropriate management with the help of laboratory investigations and imaging. Medical literature has only scarce data regarding encephalopathy in adults, with even fewer studies in Indian adults. Hence, this study aims to investigate the incidence, etiological factors, clinical presentation, and outcomes of patients with metabolic encephalopathy admitted to a tertiary care hospital.

MATERIALS AND METHODS

A prospective observational study was conducted at the Karnataka Institute of Medical College Hospital and Research Centre, Hubli, Karnataka. Ethical clearance had been taken from the institution and respective university (Rajiv Gandhi University of Health Sciences Bengaluru, Karnataka) with letter ACA/DCD/SYN/KIMS-H/PG/2018-19 dated 15/11/2018. Over the course of two years, 97 patients took part in the study. Patients were included in the study if they presented with altered sensorium and had clinical suspicion of metabolic encephalopathy, underwent pertinent laboratory investigations such as CBC (Complete blood count), LFT (Liver function test) with Sr. ammonia RFT (Renal function test), RBS (Random blood sugar), CSF (Cerebrospinal fluid) analysis, brain MRI (Magnetic resonance imaging) and also, Glasgow coma scale [8]. Patients were selected through simple random sampling following strict adherence to predetermined inclusion and exclusion criteria.

Study Design

This was a prospective observational study conducted from 2018 to 2020.

Population

Patients admitted with suspected metabolic encephalopathy were included after fulfilling the inclusion criteria.

Statistical Analysis

All of the collected data was imported into Microsoft Excel, and the data were analyzed using SPSS version 26. Chi-square tests were used to compare categorical variables, while t-tests were employed for continuous variables. A P-value (Probability that the result is true) of <0.05 was considered statistically significant after assuming all the rules of statistical tests.

Inclusion criteria

- All patients admitted to medicine wards / ICU with altered sensorium with clinically suspected metabolic encephalopathy.
- Age $\geq$ 13 years.
- Both male and female patients.

Exclusion criteria

- Patients with proven organic causes of brain injury.
- Patients with neuro-infection.
- Patients are not giving consent to participate in the study.

Study method

Patients who were admitted with altered sensorium and satisfied the inclusion criteria were included in the study. A variety of scales and investigations, including the GCS score (It is a scale used to assess a patient's level of consciousness) [8]. Electrophysiological study (EEG), CT/MRI, laboratory investigations such as CBC, LFT, RFT, Sr. Electrolytes, ABG, Sr. Ammonia, RBS/HBA1c, viral markers, fundoscopy, and CSF analysis were used to study the incidence, various etiological factors, and clinical presentation of metabolic encephalopathy. Additionally, the results of different aetiologies were examined in terms of length of hospital stay, morbidity, and mortality.

RESULTS

As per table 1, metabolic encephalopathy was commonly seen in the age group between 51-60 years old, i.e., 29.9%, followed by 22.7% in 41-50 years old. Least commonly seen in ≤20 Years old. And the Mean Age (Years) of our study subjects was 53.10 ± 13.21 years

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Table-01: Age distribution of patients

Age	Count (percentage)
≤20 Years	1 (1.0%)
21-30 Year	2 (2.1%)
31-40 Years	15 (15.5%)
41-50 Years	22 (22.7%)
51-60 Years	29 (29.9%)
61-70 Years	20 (20.6%)
71-80 Years	5 (5.2%)
81-90 Years 3 (3.1%)	3 (3.1%)

As shown in table 2, the male gender was predominantly seen, i.e., 76.28%, maximum patients (80.4%) presented as insidious in the onset of altered sensorium, the most common symptom was altered behavior (51.5%), the less common presentation was a focal neurological deficit. The most common comorbidity was diabetes, seen in 47% of patients, followed by hypertension (45.4%). Most patients had GCS scores ranging from 9 to 12, consistent with Grade 2 hepatic encephalopathy and was seen in 45.2% of patients. As shown in the below table, patients with hepatic encephalopathy had a higher median hospital stay compared to those with hypoglycemic encephalopathy

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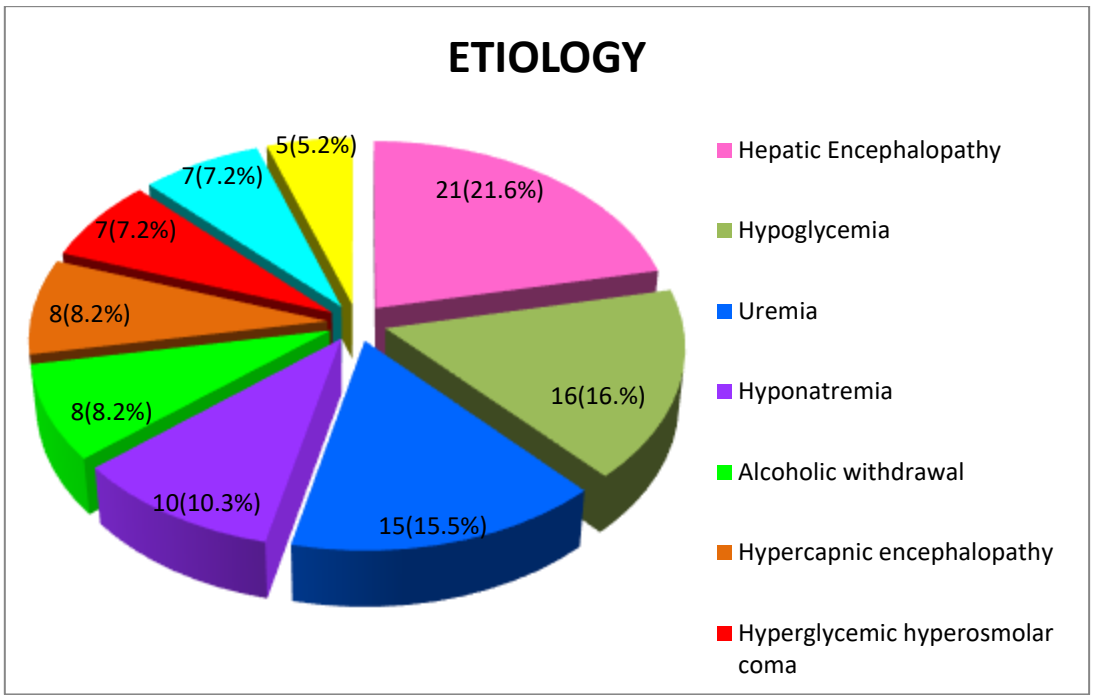
Table-02: Summary of results.

		Frequency	Percentage
Gender	Male	74	76.28%
	Female	23	23.71%
	Total	97	100.0%
Altered Sensorium: Onset	Sudden	19	19.6%
	Insidious	78	80.4
	Total	97	100%
Symptoms	Fever	18	18.6%
	Headache	38	39.2%
	Vomiting	47	48.5%
	Convulsion	24	24.7%
	Focal Deficits	2	2.1%
	Altered Behavior	50	51.5%
	Loose Stools	10	10.3%
	Abdominal Distension	25	25.8%
	Jaundice	24	24.7%
	Leg Swelling	43	44.3%
	Reduced Urine Output	24	24.7%
	Breathlessness	25	25.8%

Comorbidities	Diabetes	46	47%
	Hypertension	44	45.4%
	Epilepsy	5	5.2%
	Ischemic heart disease	4	4.1%
	Chronic liver disease	21	21.6%
	Chronic kidney disease	10	10.3%
	Chronic obstructive pulmonary disease	8	8.2%
GCS Score [8].	13-15	18	18.6%
	9-12	56	57.7%
	≤8	23	23.7%
Grading of hepatic encephalopathy	Grade-1	5	16.1%
	Grade-2	14	45.2%
	Grade-3	8	25.8%
	Grade-4	4	12.9%
Duration of hospital stay	≤6 days	12	12.37%
	7-14 days	83	85.56%
	≥15 days	2	2.06%

According to figure1, Hepatic encephalopathy was the leading cause of metabolic encephalopathy, accounting for 21.6% of cases, followed by hypoglycemia in 16%. While septic encephalopathy had the highest mortality rate, 39.1% of cases, even though it was less commonly seen (5.2%) of patients.

Figure 1



Pie diagram showing Hepatic encephalopathy was the most common ethology, seen in 21.6% of patients. Mortality was significantly higher in patients with septic encephalopathy ($p < 0.05$)

DISCUSSION

The fact that metabolic encephalopathy is often linked to secondary causes is what makes it so significant. If a good study of etiology is done, the presenting metabolic encephalopathy can be treated effectively with history, clinical examination, and appropriate investigations, including neuroimaging. This will lower the morbidity and mortality rates as well as the related societal repercussions. The study's patient population had an average age of 53.10 ± 13.21 years. Similar findings were found in studies by Swaroopa et al. [9], John B et al. [10], and Jali SN et al. [11], with ages of 48.7, 70.7, and 58 years, respectively. These studies are all comparable to the one being done now. The majority of patients (29%) in the current study were between the ages of 51 and 60. The gender distribution in our study revealed 76.28% male patients and 23.71% female patients; similarly, studies by Swaroopa et al. [9], Jhon B et al. [10], Jali SN et al. [11], and others showed 64% male and 36% female patients, and 62% male and 38% female patients, respectively. All of these studies are consistent with our findings.

After analyzing the patient demographics, we next examined the etiological factors contributing to metabolic encephalopathy. The most common symptom in our study was altered behavior, seen in 51.5% of patients; vomiting came in second with 48.5% of patients, and focal neurological deficit was the least common presentation, seen in 2.1% of patients. About 18.6% of the patients had a fever, compared to 25.6% in the studies by Swaroopa et al. [9], 34.6% in the study by Jhon B et al. [10], and 39.2% in our study. This is also the case for 57.3% of the patients in the study by Thula Nidhi et al. [12]. In our study population, 24.7% experienced convulsions, which is comparable to the 18.6% in Thula Nidhi et al.'s [12] study. Additional symptoms observed in our study included reduced urine output, breathlessness, bilateral leg swelling in 44.3% of cases, loose stools (10.3%), and abdominal distension (25.8%). Jaundice was observed. Similar to Swaroopa et al. [9], 63% of patients had jaundice, as did 24.7% of our patients. In our study, the most prevalent comorbidities were diabetes (47.4%), hypertension (45.4%), chronic kidney disease (10.3%), epilepsy (5.2%), infertile heart disease (4.1%), and COPD (8.2%). Studies by Hiremath et al. [13], which revealed 40% of subjects had diabetes, and Jhon B et al. [10], which showed 45.4% of subjects had diabetes, are comparable to the current study. 37.5% of participants in the Sarin SM study [14] had hypertension; this result is in line with what we found in our investigation. Comparable to our study, 18% of study participants in the Jhon B et al [10] study had chronic liver disease. Comparably, studies by Hiremath et al. [13] and Thula Nidhi et al. [12] found that 2% of patients had epilepsy; these results align with the present study.

A study by Jhon B et al. [10] found that 25.48% of participants had a GCS score between 13 and 15, which is similar to the 18.6% found in this study. 54.80% of subjects in a study by Jhon B et al. [10] had a GCS score below 8%. In research conducted by Hiremath et al. [13] and Thula Nidhi et al. [12], all participants have a GCS score of less than 8. Hepatic encephalopathy was found to be 29.12% in a study by Swaroopa et al. [9], 22.72% in a study by Hiremath et al. [13], and 22.58% in a study by Thula Nidhi et al. [12], all of which are comparable to the current study's finding of 21.6%. Hypoglycemic encephalopathy was 18.11% in a study by Jhon B et al. [10] and 14.28% in a study by Jali SN et al. [11]; these findings are similar to the 16.5% of hypoglycemic encephalopathy found in the current study. Uremic encephalopathy was 15.94% in research by Jhon B et al. [10] and 15.78% in a study by Sarin SM [14], which are comparable to the 15.5% of uremic

encephalopathy found in the current study. Similar to the 10.3% reported in this study, Hiremath et al.'s study [13] indicated that the prevalence of hyponatremia encephalopathy was 9.09. The present study's 7.2% incidence of hypercapnic encephalopathy is comparable to the 9.42% reported in a study by Jon B. et al. [10]. In research by Hiremath et al. [13], the prevalence of hypertensive encephalopathy was 2%, which is similar to the 7.2% reported in this study. Septic encephalopathy, a severe brain dysfunction caused by infection, accounted for 39.1% of deaths in this study. In a study by Swaroopa et al. [9], Septic Encephalopathy was 19.41% in a study by Swaroopa et al. [9], 10.52% in a study by Sarin SM [14], and 18.5% in a study by Bukhari R et al. [15]. These findings are not comparable to the 5.2% of Septic Encephalopathy found in the current study. Our findings underscore the need for early recognition and treatment of septic encephalopathy to reduce mortality rates. Future studies should focus on improving diagnostic protocols for metabolic encephalopathy.

Strengths of the study

The study is unique in that it assesses the many causes of ME, its clinical characteristics, and the patients' outcomes in terms of duration of stay, recovery, or death.

Multiple parameters were assessed in one study.

Limitations of the study

limited number of subjects due to the high expense of laboratory testing and the possibility of inconsistent results if conducted on a large number of subjects

It is a single-center study.

Being a hospital-based study, there is a bias factor in the selection of subjects.

The study population may not be representative of the general population because it only included symptomatic patients who came to the hospital.

CONCLUSION

In this study, the maximum number of patients had altered levels of consciousness on admission (51.5%), which was started insidiously (80.4%), followed by vomiting 48.5%. Male gender was predominantly affected in the age group of 51-60 years old (29.9%). Diabetes was the most common comorbidity seen in our cases. Maximum patients had GCS scores between 9-12 (57.7%). On average, most of the patients needed 7-14 days of hospitalization for recovery (85.56%). In order to improve patient treatment, more multicenter research with a larger sample size enhances the early and accurate etiological identification of acute altered sensorium episodes as many metabolic encephalopathies are completely recoverable.

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