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Original Research

# Etiologic and Clinical Profile and Outcome of Encephalopathy Patients at a tertiary care hospital

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***ABSTRACT:***

**Background**: This study was conducted to assess the Etiologic and Clinical Profile and Outcome of Encephalopathy Patients at a tertiary care hospital. **Material and methods**: This was a retrospective, hospital-based study. Overall, 100 subjects were recruited. 70 subjects were males and 30 subjects were females. Acute or subacute onset of altered mental status (AMS), elevated antithyroid antibodies, a rapid response in mental status with corticosteroids, and the absence of structural, infectious, or other metabolic factors that could explain AMS and its response to steroids were the criteria used to diagnose HE.The following conditions were excluded from the study: (a) illness with an infectious, metabolic, toxic, or vascular etiology; (b) illness with a structural lesion or traumatic brain injury; (c) postoperative encephalopathy; and (d) patients under the age of 18 years. Out of the 100 patients who were examined, 30 had high TPO antibody levels (>60 IU/mL) and were diagnosed with encephalopathy. **Results**: Out of 100 subjects, 70 subjects were males and 30 were females. Cognitive impairment was seen in 43/100 subjects. Sleep disturbances, insomnia, ataxia and headache were observed in 19,10,9 as well as 7 out of 100 subjects. Seizures as well as tremors were observed in 5 as well as 4 subjects. Neurological aetiology was seen in 49 (49%) patients. Hyponatremia was present in 27(27%) patients and hypoglycaemia was seen in 14 (14%) patients. Pneumonia was the commonest infection present in 10(10%) patients. **Conclusion**: It is important to screen each patient with hepatic encephalopathy. Physicians, endocrinologists, and neurophysicians may benefit from earlier diagnosis and treatment with positive clinical outcomes if they are more aware of this clinical entity.

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## INTRODUCTION

Hepatic encephalopathy (HE) or portosystemic encephalopathy (PSE) is a reversible syndrome of impaired brain function occurring in patients with advanced liver failure. However, HE is not a single clinical entity. It may reflect either a reversible metabolic encephalopathy, brain atrophy, brain edema or any combination of these conditions. The mechanisms causing brain dysfunction in liver failure are still unknown. These factors are directly related to liver failure (e.g. decreased metabolism of ammonia). Unless the underlying liver disease is successfully treated, HE is associated with poor survival and a high risk of recurrence [1,2]. Even in its mildest form, HE reduces health-related quality of life and is a risk factor for bouts of severe HE [3,4].

HE produces a wide spectrum of nonspecific neurological and psychiatric manifestations [5]. In its lowest expression [6,7], HE alters only psychometric tests oriented towards attention, working memory, psychomotor speed and visuospatial ability, as well as electrophysiological and other functional brain measures [8,9].

As HE progresses, personality changes, such as apathy, irritability and disinhibition, may be reported by the patient's relatives [10], and obvious alterations in consciousness and motor function occur.

Disturbances of the sleep–wake cycle with excessive daytime sleepiness are frequent [11], whereas complete reversal of the sleep–wake cycle is less consistently seen [12,13]. Patients may develop progressive disorientation to time and space, inappropriate behavior, acute confusional state with agitation or somnolence, stupor and, finally, coma [14].

Hence, this study was conducted to assess the Etiologic and Clinical Profile and Outcome of Encephalopathy Patients at a tertiary care hospital.

**Material and methods**

This is a retrospective, hospital-based study. Overall, 100 subjects were recruited. 70 subjects were males and 30 subjects were females. Acute or subacute onset of altered mental status (AMS), elevated antithyroid antibodies, a rapid response in mental status with corticosteroids, and the absence of structural, infectious, or other metabolic factors that could explain AMS and its response to steroids were the criteria used to diagnose HE.The following conditions were excluded from the study: (a) illness with an infectious, metabolic, toxic, or vascular etiology; (b) illness with a structural lesion or traumatic brain injury; (c) postoperative encephalopathy; and (d) patients under the age of 18 years. Out of the 100 patients who were examined, 30 had high TPO antibody levels (>60 IU/mL) and were diagnosed with encephalopathy. As a result of the exclusion criteria, 9 patients were excluded. 23 cases were discovered to match the established HE criteria. We carefully inspected the included patients' case records. Age, gender, past medical history, medications, antithyroid antibody levels, brain magnetic resonance imaging (MRI), electroencephalogram (EEG), CSF analysis, course of treatment, and outcome data were all gathered. Utilizing mean, median, standard deviation, and percentage, statistical analysis was conducted.

**Results**

Out of 100 subjects, 70 subjects were males and 30 were females. Cognitive impairment was seen in 43/100 subjects. Sleep disturbances, insomnia, ataxia and headache were observed in 19,10,9 as well as 7 out of 100 subjects. Seizures as well as tremors were observed in 5 as well as 4 subjects. Neurological aetiology was seen in 49 (49%) patients. Hyponatremia was present in 27(27%) patients and hypoglycaemia was seen in 14 (14%) patients. Pneumonia was the commonest infection present in 10(10%) patients.

**Table 1: Gender-wise distribution of subjects**

| Gender | Number of subjects | Percentage |
|---|---|---|
| Males | 70 | 70% |
| Females | 30 | 30% |
| Total | 100 | 100% |

**Table 2: Clinical findings in subjects with encephalopathy.**

| Clinical Findings | Number of subjects (n=100) |
|---|---|
| Cognitive impairment | 43 |
| Sleep disturbance | 19 |
| Insomnia | 10 |
| Ataxia | 09 |
| Headache | 07 |
| Seizures | 07 |
| Tremors | 05 |
| Total | 100 |

**Table 3: Etiologic profile of hepatic encephalopathy**

| Aetiology | Number of subjects |
|---|---|
| Neurological | 49 |
| Hyponatremia | 27 |
| Hypoglycaemia | 14 |
| Pneumonia | 10 |
| Total | 100 |

**Discussion**

Hepatic encephalopathy (HE) is a reversible syndrome observed in patients with advanced liver dysfunction. The syndrome is characterized by a spectrum of neuropsychiatric abnormalities resulting from the accumulation of neurotoxic substances in the bloodstream (and ultimately in the brain). Symptoms typically include confusion, personality changes, disorientation, and a depressed level of consciousness. The earliest stage is often characterized by an inverted sleep-wake pattern wherein patients are found to be sleeping during the day and awake throughout the night. Throughout the intermediate stages, patients tend to experience worsening levels of confusion, lethargy, and personality changes. In the advanced stages, hepatic encephalopathy may eventually lead to coma (e.g., hepatic coma or coma hepaticum) and ultimately to death.[15]

The clinical features and presentation of HE vary based on its severity. While patients with subclinical or minimal HE (MHE) have disturbances detected only on neuropsychiatric and psychomotor testing, patients with overt HE may present with coma. Disturbance in the diurnal sleep pattern is a common early manifestation of HE and is related to altered melatonin secretion. More advanced neurologic features of HE include bradykinesia, asterixis (flapping motions of outstretched, dorsiflexed hands), hyperreflexia, and transient decerebrate posturing. Rarely, HE may be associated with development of transient focal neurologic deficits, the most common of which is hemiplegia. Although asterixis is commonly seen in patients with HE it is not specific to this disease and it can also be observed in patients with other forms of metabolic encephalopathies such

as in uremia, respiratory failure, and barbiturate toxicity.[16]

Hence, this study was conducted to assess the etiologic and clinical profile of subjects suffering from hepatic encephalopathy.

In this study, out of 100 subjects, 70 subjects were males and 30 were females. Cognitive impairment was seen in 43/100 subjects. Sleep disturbances, insomnia, ataxia and headache were observed in 19,10,9 as well as 7 out of 100 subjects. Seizures as well as tremors were observed in 5 as well as 4 subjects. Neurological aetiology was seen in 49 (49%) patients. Hyponatremia was present in 27(27%) patients and hypoglycaemia was seen in 14 (14%) patients. Pneumonia was the commonest infection present in 10(10%) patients.

Devrajani et al[17] determined the precipitating factors of hepatic encephalopathy (HE) in patients with liver cirrhosis at Liaquat University Hospital Hyderabad/Jamshoro. This hospital based descriptive study was conducted from April 2007 to September 2007. All the patients who were more than 12 years of age and were diagnosed as hepatic encephalopathy were studied. During this period, 87 patients of hepatic encephalopathy were admitted. All patients were carefully examined, relevant investigations were performed and data was collected through pre-designed proforma. Male patients were 65 (75%), above 40 years of age 58 (67%), belonging to interior/periphery of Sindh 54 (62%), in grade IV of hepatic encephalopathy 70 (80%) and Anti-HCV positive were 52 (60%). The most common precipitating factors detected were infection 58 (67%), constipation 43 (49%) and gastrointestinal bleeding 39 (45%). Out of 87 patients, 68 had increased total leucocytes count, 09 patients had hypokalaemia, 24 patients, hyponatraemia, 64 hypoalbuminaemia and 54 patients had a disturbed coagulation profile. Fifty nine patients recovered and were discharged while 20 patients expired. Majority of expired patients had Child-Pugh score 10-15 and were in grade IV of hepatic encephalopathy. The study concluded that there were different factors which play a key role in hepatic encephalopathy. In these factors, infection was the most common.

Sharma et al [18] characterized the clinical, laboratory and radiologic findings in patients with HE. Retrospective analysis of clinical features and diagnostic test data. Clinical features, laboratory, radiologic, electroencephalography (EEG) findings associated with HE and therapeutic outcome. Thirteen consecutive patients were identified as having HE. The median age at onset was 48.5 years (range, 19–62 years). There was a female preponderance (76.9%). Clinical manifestations were cognitive impairment and behavioral changes in 10 (76.9%), sleep disturbance in 9 (69.2%), seizures in 6 (46.1%), headache in 4 (30.8%), psychosis or paranoia in 5 (38.5%), transient symptoms in 6 (46.1%), myoclonus in 4 (30.8%), ataxia or gait disorder in 4 (30.8%). The most frequent laboratory abnormalities were increased TPO (n = 13) in all cases, increased thyroid stimulating hormone levels (n = 6), and increased erythrocyte sedimentation rate (n = 5). The cerebrospinal fluid protein level was elevated in 8 of 9 patients (88.8%). Magnetic resonance imaging abnormalities were present in 2 patients (15.4%). EEG changes were seen in 7 patients (53.8%). All but two patients showed significant therapeutic benefit with steroids. It was concluded that HE had a wide range of clinical, laboratory, and radiologic findings. All patients with an unexplained encephalopathy should be screened for this condition as treatment response is excellent. It is important to screen each patient with hepatic encephalopathy. Physicians, endocrinologists, and neurophysicians may benefit from earlier diagnosis and treatment with positive clinical outcomes if they are more aware of this disorder.

## Conclusion

It is important to screen each patient with hepatic encephalopathy. Physicians, endocrinologists, and neurophysicians may benefit from earlier diagnosis and treatment with positive clinical outcomes if they are more aware of this clinical entity.

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