Frequency of Risk Factors for Hepatic Encephalopathy in Patients of Chronic Liver Disease

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To study the frequency of risk factors for hepatic encephalopathy in patients of chronic liver disease. This is a retrospective, non interventional hospital based observational case study between April 2002 to April 2003 carried out in North Medical Ward, Mayo Hospital, Lahore. Two hundred consecutive patients of chronic liver disease presenting with hepatic encephalopathy were studied. The frequency of risk factors was 47%, due to infections, 30% due to GIT bleeding, 19% due to constipation, 4% due to miscellaneous factors. Early recogtnition of the precipitating factors improves the prognosis and final outcome.
Key words: Frequency, chronic liver disease, risk factors

Hepatic enceophalopathy is a well recognized clinical complication of chronic liver disease[12,15,16]. About 30% of patients with cirrhosis die in hepatic coma. Hepatic encephalopathy can be acute, subacute or chronic. It can be clinical or subclinical. It is usually as a result of substantial damage to the liver so that this organ cannot maintain its metabolic function. In cases of chronic liver disease already much damage is there so any further insult will lead on to hepatic encephalopathy. There is usually an alteration in the balance of neurotransmitters in brain. The pathogenesis is usually multifactorial[1,5]. Blood ammonia levels are raised[6], there is presence of false neurotransmitters like branched chain amino-acids, accumulation of neuroinhibitory substances and manganese, different monoamines[14] and endogenous opiates.

Liver disease may be known or the patient is presenting for the first time. The signs of chronic[18,19] liver disease may or may not be present. Neurological symptoms fluctuate. Flapping tremors or positive babinski's sign may be seen. There are changes in mental state, consciousness, behaviour and personality. There can be forgetfulness, confusion, disorientation, delirium, dementia, mood changes, decreased alertness and inverted sleep pattern. The patient loses self care ability. There can be loss of small hand movements and deterioration of hand writing, speech impairment, muscle stiffness, agitation, seizures, progressive stupor leading on to coma is seen. The factors leading on to hepatic encephalopathy are multiple.

The presence of blood in the gastrointestinal tract results in increased ammonia and nitrogen absorption. Bleeding may predispose to kidney hypoperfusion and azotemia. Blood transfusion may result in mild hemolysis with resulting elevated blood ammonia levels.

Infections may predispose to impaired renal function and to increased tissue catabolism, both of which increase blood ammonia levels.

Constipation increases intestinal production and absorption of ammonia. Drugs that act upon the central nervous system, such as opiates, benzodiazepines, antidepressants and antipsychotic agents may precipitate or worsen hepatic encephalopathy. Diuretic therapy decreases serum potassium levels and alkalosis facilitates the conversion of Nh_4^+ to NH_3.

Dietary protein overload is an infrequent cause of hepatic encephalopathy hypoxia, hypercarbia and electrolytes imbalance can lead to hepatic encephalopathy.

The cases of chronic liver diseases are on the increase. There is no previous study in Pakistan to see the frequency of these factors leading on to hepatic encephalopathy.

Patients and methods

Two hundred consecutive patients admitted in North Medical Ward, Mayo Hospital, Lahore during April 2002 and April 2003 were included in the study. Detailed history and physical examination was noted down. Other acute and chronic causes of altered mental status were kept in mind like alcohol intoxication, sedative overdosage, alcohol withdrawal, subdural hematoma, meningitis and hypoglycemia. In the history it was specifically noted that the patient was using the already prescribed medicine or not. History of fever, cough, expectoration, H/O constipation, sedative drug intake, self medication etc., were noted down. Usually the diagnosis was obvious. A decision to perform additional neurological studies was based on the presence of focal neurological findings or the patient's unresponsiveness to an empirical trial with cathartics.

Results

Out of two hundred patients, 58% were male and 42% were female patients.

Table 1 Age distribution

Age in years	Male	Female
20-30	12(6%)	2(1%)
31-40	52(26%)	44(22%)
41-50	36(18%)	28(14%)
51 and above	16(8%)	10(5%)

One hundred and ninety patients were known cases of chronic liver disease while ten patients presented for the first time with hepatic encephalopathy.

Table 2. Patients receiving different drugs for CLD

Diuretics	n=	%age
Laxatives	156	82
Beta blockers	150	79
Antibiotics	23	12
Nitrates	68	36

Table 3. Drug compliance of the patients (n=190)

Compliance	n=	%age
Good	140	74
Poor	40	21
Very poor	10	5

Table 4. Number of days after the last stool passed (n=38).

Number of days	n=
01-03 days	8
04-06 days	29
More than 6 days	01

Table 5. Frequency of risk factors for hepatic encephalopathy (n=200).

Risk factors	n=	%age
Sedatives	04	02
Constipation	38	19
Self medication	23	12
G.I.T. bleed	59	29.5
Respiratory tract infection	15	7.5
Gastroenteritis	10	05
High protein diet	01	01
Fever	70	35

Discussion

Hepatic encephalopathy is a medical emergency and hospitalization is required. It is extremely important to identify the precipitating factor and treat accordingly. Failure to recognize the precipitating factor may leasd to a fatal outcome[8,9,10,11].

There is much evidence that if blood ammonia[2,3,4,7] levels are lowered the encephalopathy can improve. Dietary modification can help the patient limiting the amount of protein especially the amount of animal proteins especially red meat is necessary. Vegetable proteins appear to be better tolerated. This may be due to increased content of dietary fibre – a natural cathartic and decreased levels of aromatic amino acids which worsen hepatic encephalopathy. Normal skeletal muscle aids in the metabolism of ammonia in the conversion of glulamate to glutamine. The muscle wasting due to prolong protein restriction may potentiate hyperammonemia. So substituting animal proteins with vegetable proteins is better than total protein restriction[13,17]. Identify infections because these may not be associated with typical response of body to infection. The patient bleeding should present at the earliest.. Proper guidance and explanation of risk factors of hepatic encephalopathy will help reduce its incidence and its consequences.

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