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A STUDY OF CLINICAL AND LABORATORY PROFILE IN PATIENTS WITH HEPATIC ENCEPHALOPATHY

General Medicine

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ABSTRACT

Background: Hepatic encephalopathy is a syndrome that is characterized by abrupt deterioration in patients with CLD and has high short-term mortality. It is of utmost important to diagnose cause of CLD , precipitating factors for hepatic encephalopathy for better management of patients. **Objectives:** To study clinical and laboratory profile in patients with hepatic encephalopathy. **Method:** prospective study was an analytical study conducted at tertiary care centre, on 50 patients. **Conclusion:** hepatic encephalopathy is the commonest complication of cirrhosis . It has detrimental effect on health related quality of life and survival . ammonia plays key role in pathogenesis of HE via induction of astrocyte sweeling and cerebral edema . There is no diagnostic test , so combination of clinical examination , Different psychometric tests and EEG helps in diagnosis . nevertheless , condition is often remains undiagnosed ,so remains untreated .

KEYWORDS

INTRODUCTION :

Hepatic Encephalopathy (HE) is a complex, potentially reversible neuropsychiatric condition that occurs as a consequence of acute or chronic liver disease. It is a well recognized clinical complication of cirrhosis of liver and the presence and prompt identification of well defined precipitating factors is extremely important in diagnosis and treatment of this fatal condition. Cirrhosis of liver is more common in our day to day clinical practice as in our sub-continent there is high prevalence of Hepatitis B as well as Hepatitis C and alcoholism. In the management of patients with HE in cirrhosis of liver it is important to stage the encephalopathy into four clinical stages and then try to identify and treat the precipitating factors. In the presence of the precipitating factors the neurological deficits are usually completely reversible upon their correction and the prognosis is better if the precipitant can be treated. This study aims at studying the clinical profile and the spectrum of precipitating factors of HE in cirrhosis of liver, hence identifying them and initiating theappropriate treatment can bring down the morbidity and mortality.

Hepatic encephalopathy (HE) is a hallmark of liver failure and affects up to 40% of patients with liver cirrhosis. It is defined as a multifactorial neuropsychiatric disorder presenting with a broad spectrum of cognitive impairment and neuromuscular dysfunction. HE is a significant contributor to repeated hospitalizations for patients with liver cirrhosis and severely impacts on the quality of life of both patients and caregivers. It is a marker of poor prognosis in cirrhotic patients, with reported rates of survival of only 36% at 1 year from its first presentation. Chronic liver disease is the fifth most common cause of death in the UK, with the mortality expected to rise due to increase in cirrhosis caused by alcohol-related liver disease, chronic hepatitis B & C and nonalcoholic fatty liver disease. Patients commonly present to primary and secondary care services with complications such as HE with or without a prior diagnosis of chronic liver disease. A milder form of the disorder, covert hepatic encephalopathy (CHE) or covert encephalopathy with subtle alterations of cognitive function, also exists. Although less severe, patients with CHE are at significant risk of interference with their quality of life, including increased falls, hospitalizations and progression to overt HE.

The aims of this review are to provide a comprehensive, “state of the art”, account of the pathophysiology, clinical manifestations (classification, symptoms, signs and investigations), current treatments and future targets for the management of patients with HE. The review is targeted to physicians in primary or secondary care and also to health care professionals who are likely to encounter patients with liver disease in their professional roles.

METHOD: This is a Prospective Observational type of study conducted among indoor patients admitted in medicine department at B.J. Medical College and Civil Hospital, Asarwa; Ahmedabad during the study period , enrolment of 50 cases.

Inclusion Criteria:

Patients diagnosed as hepatic encephalopathy, after excluding other causes of encephalopathy.

Exclusion Criteria: Patients with other causes of encephalopathy like, hypoxic, hypoglycemic, uremic, metabolic, cerebrovascular accident etc. have been excluded.

OBSERVATION:

1. Mean age of presentation is 42 years, with maximum number of patients were in age group of 41-50 years.
2. There is preponderance of male with hepatic encephalopathy as compared to female with ratio of 7.33:1.
3. Commonest symptom observed was jaundice(80%), vomit/constipation (34%), altered level of consciousness (66%), hematemesis & malena(32%), fever(20%), convulsion(10%).
4. Commonest sign observed was icterus(82%), pedal edema(62%), spider naevi(30%), loss of body hairs(10%), gynacomastia(24%), testicular atrophy(18%), petechiae(24%) ecchymosis(22%), foetor hepaticus(20%), palmar erythema(10%), dupuytren contracture(2%).
5. Commonest abdominal finding was splenomegaly (82%) . others are ascites (48%), distended abdominal veins (30%), and hepatomegaly (58%)
6. Commonest neurological abnormalities seen were abonormal behaviour (84%) . others are , altered sleep pattern(44%), constructional apraxia & number connection test(42%), flapping tremor (72%), Extensor plantar(20%).
7. Cirrhosis was most important causative factor followed by alcoholic hepatitis. In acute variety , viral hepatitis E> viral hepatitis A .
8. out of 50 cases 19 were acute HE , 14 chronic HE, 15 subacute HE and 2 were hyperacute HE.
9. commonest alteration in haemogram isthrombocytopenia , mainly in chronic variety followed by microcytic hypochromic anaemia .
10. Serum billirubin was elevated in 47 patients out of 50 , maximum rise in hyperacute variant . liver enzymes (SGPT ,SGOT , ALP) Elevated in average 29 patients with maximum rise in acute variant . serum protiens and serum albumin decreased in 42 patients with maximum decreased in chronic variant .
11. In hyperactue mean INR was 1.67, in acute its 1.71, subacute it is 1,71 and in chronic its 1.57 . It is an indication of hepatocellular variant
12. serum ammonia mean value is 100 , 117 , 127 , 107 in acute , chronic , hyperacute and subacute HE respectively .
13. Serum sodium levels: Mean sodium levels in acute was 136.47, hyperacute was 137.50, subacute was 134.53 and chronic was

135.41 . Serum potassium levels:mean levels in acute was 3.79 mmol/l, hyperacutewas 3.06 mmol /1, subacute was 3.81mmol/1 and chronic was 3.57 mmol/l. Serum creatinine levels: Mean levels in acute was 1.15 mg%, hyperacute was1.50 mg%, subacute was 2.91 mg% and chronic was 2.11 mg.

14. MELD SCORE is <09 in 2patients , 10-19 in 10patients , 20-29 in 20 patients , 30-39 in 4 patients and >40 in 1patient of liver parenchymal disease.
15. CPS CLASS is A in 12 patients , B in 22 patients and C is in 5 patients of liver parenchymal disease.
16. EEG study done on 15 patients with grade 1 HE , out of which 12 patients showed major alpha activity and some theta activity. (not possible in grade 2 , 3 and 4of hepatic encephalopathy
17. Maximum mortality was seen in hyperacute encephalopathy (100%) followed by subacute encephalopathy (53 %), chronic encephalopathy (42.8%), acute encephalopathy (31.5%).

CONCLUSIONS:

Hepatic encephalopathy is the commonest complication of cirrhosis . It has detrimental effect on health related quality of life and survival . ammonia plays key role in pathogenesis of HE via induction of astrocyte sweeling and cerebral edema. There is no diagnostic test , so combination of clinical examination , Different psychometric tests and EEG helps in diagnosis. nevertheless, condition is often remains undiagnosed ,so remains untreated. Understanding inter-organ metabolism of ammonia and pathophysiological basis of HE are most likely to lead to development of new therapeutic approaches . At present , Therapy is directed at reducing circulating ammonia by use of non-abosrbable diasaccharides and nonabosrbable antibiotics , with focus on identifying and treating precipitating factor . Considering higher mortality and significant morbidity in such patients , we are still lack of investigation for specific diagnosis and molecule for specific treatment . Extracorporeal liver assist devices like MARS : molecular adsorbent recirculating system and SPAD : single pass albumin dialysis , are presumed to be future of management of HE .

REFERENCES:

1. Bruce R. Bacon ; cirrhosis and its complications; Harrisons internal medicine; Fauci,Braunwald,Kasper,Hauser,Longo,Jame son,Loscalzo; Mac Graw Hill ;2008,volume-2, 20th edition.
2. Adam RD,Foleys JM;hepatic encephalopathy;Adams and victor's principles of neurology ; Allan H. Ropper, Martin A, Samules; Mac Graw Hill; 2009;9th edition ;
3. N.D.C. Finlayson ,P.C.Hayes, K. J. Simpson; hepatic encephalopathy; Davidson's Principal and Practice Of medicine; John A.A. Hunter, Christopher Haslett,Edwin R. Chivers,Nicholas A.Boon;Churchill Livingstone2007 ;20th edition
4. Sheila Sherlock & James Dooley;hepatic encephalopathy;Diseases of the liver and biliary system; Sheila Sherlock & James Dooley ; Blackwell ;2003,11th edition ;