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A STUDY ON ASSOCIATION BETWEEN SEVERITY OF HEPATIC ENCEPHALOPATHY AND SERUM SODIUM IN PATIENTS WITH CHRONIC LIVER DISEASE

Medicine

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KEYWORDS

INTRODUCTION

Histologically, cirrhosis is a diffuse hepatic process characterized by fibrosis and conversion of normal liver architecture into structurally abnormal nodules. The natural history of cirrhosis can be divided into a preclinical and a subsequent clinical phase .The preclinical phase is usually prolonged over several years; once clinical events occur, such as ascites, encephalopathy or variceal bleeding, the remaining course of the disease is much shorter and usually fatal. The most commonly associated complications are: gastrointestinal hemorrhage, ascites, encephalopathy, spontaneous bacterial peritonitis, hepatorenal syndrome and hepatic failure.[1]

Hyponatremia is a common abnormal finding in approximately 57% of hospitalized patients with chronic liver disease and in 40% of outpatients with liver disease. This condition occurs as a result of high serum levels of renin/aldosterone owing to portal hypertension, a decreased vascular response to vasoactive drugs, and a reduced solute-free water clearance.[2] According to several studies, hyponatremia occurring as a result of a reduced solute-free water clearance was a key prognostic factor in patients with liver cirrhosis.[3]

The aim of this study is to analyse the association between serum sodium level and the severity of hepatic encephalopathy in chronic liver disease.

MATERIALS AND METHODOLOGY

The study was done on 100 patients of chronic liver disease admitted in Department of Medicine in Rajendra Institute of Medical Sciences, Ranchi between September, 2017 to April, 2018. Out of the 100 patients, 60 had complicated chronic liver disease and 40 patients had uncomplicated chronic liver disease. Complications included ascites, hepatorenal syndrome, spontaneous bacterial peritonitis, hepatic encephalopathy and oesophageal varices.

Hepatic encephalopathy was further graded based on severity. All patients of complicated and uncomplicated chronic liver disease were subjected to:

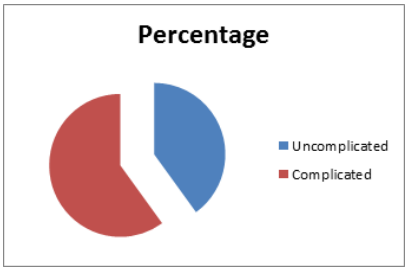
a) Complete history taken
b) Physical examination
c) Ultrasonography of the abdomen
d) Endoscopy of the upper gastrointestinal tract
e) Complete blood count and liver function test
f) Serum sodium and potassium

Observations

Out of 100 patients of chronic liver disease, 60 patients had complications (Table 1) like spontaneous bacterial peritonitis, hepatorenal syndrome, hepatic encephalopathy or ascites.

Table 1

	Frequency	Percent
Uncomplicated	40	40
Complicated	60	60
Total	100	100



Hepatic encephalopathy was present in 19 out of 60 complicated patients of chronic liver disease. In patients with hepatic encephalopathy, 70% patients with severe hepatic encephalopathy had severe hyponatremia as compared to only 30% patients with mild hepatic encephalopathy (Table 2). p value <0.05

Table 2

			Serum Sodium			Total
			>135	125-134	<125	
Encephalopathy	Absent	Count	27	14	0	41
		%	65.9%	34.1%	0%	100%
	Mild	Count	2	4	3	9
		%	22.2%	44.4%	33.4%	100%
	Severe	Count	0	3	7	10
		%	0%	30%	70%	100%
Total		Count	29	21	10	60
		%	48.3%	35%	16.7%	100%

DISCUSSION

Cirrhosis resulted in 1.2 million deaths in 2013, up from 0.8 million deaths in 1990.[4] Since chronic liver disease affects people in their most productive years of life, it has a significant impact on the economy as a result of premature death, illness, and disability. In developed countries, alcoholic liver disease, HCV and NASH are the most significant causes of cirrhosis.[5]

Normal serum sodium levels are between 135 and 145 mEq/L. Hyponatremia is defined as a serum level of less than 135 mEq/L and is considered severe when the serum level is below 125 mEq/L.[6] Hyponatremia is a frequent complication of advanced cirrhosis related to an impairment in the renal capacity to eliminate solute-free water that causes retention of water that is disproportionate to the retention of sodium, thus causing a reduction in serum sodium concentration and hypo-osmolality.[7]

There is evidence suggesting that hyponatremia may affect brain function and predispose to hepatic encephalopathy. Hyponatremia also represents a risk factor for liver transplantation as it is associated with increased frequency of complications and impaired short term survival after transplantation. Hence, hyponatremia in cirrhosis is associated with increased morbidity and mortality.[8]

In our study, we try to explore the prevalence of hyponatremia and its relation to hepatic encephalopathy in patients hospitalized with liver

cirrhosis. In the current study, the risk for hepatic encephalopathy was increased at low serum sodium concentrations. Hepatic encephalopathy has been shown in several studies to be worsened by the presence of hyponatraemia.

Treatment options for patients with hyponatremia and cirrhosis are limited. The first step in its management should be to identify and correct the underlying cause of hyponatraemia, which includes holding diuretics and addressing gastrointestinal losses. Free water restriction to less than 1.0–1.5 L/day has become standard practice in treating patients with hypervolemic hyponatraemia in cirrhosis and may have some anecdotal benefit in preventing a further drop in serum sodium. Severe hyponatraemia with serum sodium level <120 mEq/L is uncommon in the cirrhotic population, occurring in less than 1.2% of patients. In the setting of severe hyponatraemia with symptoms such as seizure, one must consider correction to a safe level, so as to prevent recurrence and neurological injury. This is the one situation where the administration of hypertonic saline is advised, with care taken to avoid overly rapid correction. The use of hypertonic saline in cirrhotic patients can lead to worsening ascites and oedema secondary to the sodium avid state that exists in the nephrons, and should be used only in acute situations.[9] Albumin infusion can be considered as a treatment for hyponatraemia in cirrhosis.

CONCLUSION

Out of 100 patients of chronic liver disease, 60 patients had complications like spontaneous bacterial peritonitis, hepatorenal syndrome, hepatic encephalopathy or ascites. There was a strong association between decrease in serum sodium level and the severity of encephalopathy (p=<0.05). We can therefore conclude that hyponatremia, especially serum levels <125 mmol/L, may indicate the existence of severe hepatic encephalopathy associated with chronic liver disease.

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