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Original Research

Hyponatremia in hepatic encephalopathy of chronic liver disease

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ABSTRACT

Background: To assess the correlation between hyponatremia in hepatic encephalopathy of chronic liver disease. **Materials & methods:**A total of 40 subjects with hepatic cirrhosis were enrolled. Patients with diagnosed hepatic cirrhosis of any etiology who were aged between 35-80 years were included. The patients were classified into different groups based on the serum sodium concentration as follows: level of <130 meq/l (significant/severe hyponatremia), between 131 and 135 meq/l (mild hyponatremia), and level of >135 meq/l (normal). All the results were evaluated using SPSS software. P- value of less than 0.001 was taken as significant. **Results:** In patients with hyponatremia, it ranged from 113 to 129 meq/L. Hyponatremia was present in 16 (40%) subjects. 2 (5%) patients had mild hyponatremia, 7 (17.5%) had moderate, and 5 (12.5 %) had severe hyponatremia. **Conclusion:** There exists a correlation between the hyponatremia in hepatic encephalopathy of chronic liver disease.

Keywords: Hepatic encephalopathy, Hyponatremia, liver disease.

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INTRODUCTION

Hyponatremia is the most common electrolyte abnormality observed in hospitalized patients.[1] Hyponatremia in cirrhosis is currently defined as a serum sodium level of less than 130 meq/L.[2] It has been suggested that the prevalence of a serum sodium concentration less than 135, 130 and 120 meq/L in patients with cirrhosis and ascites is 49.4%, 21.6% and 1.2%, respectively. [3]Hyponatremia in chronic liver disease is a condition characterised by increased renal retention of water relative to sodium because of impairment in the clearance of solute-free water, which is frequent in individuals with cirrhosis and portal hypertension. Hyponatremia can be caused by a variety of reasons among these patients, the most common of which is increased arginine vasopressin secretion (AVP; also known as antidiuretic hormone, or ADH). AVP is considered to be released in portal hypertension via baroreceptor-mediated nonosmotic stimulation induced by a decrease in effective circulation volume, which is generated by arterial splanchnic vasodilation. Decreased production of solute-free water along with reduction in the delivery of sodium to the distal tubule secondary to reduction in the glomerular filtration rate as well as an increase in the sodium resorption in the proximal tubule are other factors that could be responsible.[2]

Patients with cirrhosis frequently develop complications like ascites, variceal bleeding, and hepatic encephalopathy (HE). [5] The annual rate of growth of HE in cirrhotic patients is about 8%.[6] Various underlying pathologies like constipation, esophageal variceal bleed, and infections like spontaneous bacterial peritonitis can precipitate HE. [6]The clinical manifestations of HE range from the mildly altered level of sensorium to severely altered consciousness levels, difficulty in judgment, the day-night reversal of sleep, flapping tremor of hands, and irrelevant talking or speech. [7] Similarly, the patients with liver cirrhosis experience disturbance in the regulation of body fluid homeostasis. The kidneys start to retain the water excessively, which results in a significant derangement of sodium levels in the serum.[8] It is evident that the chances of developing HE and electroencephalographic abnormalities are high in the presence of hyponatremia.Hence, this study was conducted to assess the correlation between

hyponatremia in hepatic encephalopathy of chronic liver disease.

MATERIALS & METHODS

A total of 40 subjects with hepatic cirrhosis were enrolled. Patients with diagnosed hepatic cirrhosis of any etiology who were aged between 35-80 years were included. After taking relevant history and physical examination, the venous blood sample of each patient was drawn. Mild to moderate encephalopathy was classified under grades I-II, while severe encephalopathy was classified under grades III-IV. The severity of liver disease was enlisted according to the Child-Pugh score criteria. The patients were classified into different groups based on the serum sodium concentration as follows: level of <130 meq/l (significant/severe hyponatremia), between 131 and 135 meq/l (mild hyponatremia), and level of >135 meq/l (normal). All the results were evaluated using SPSS software. P- value of less than 0.001 was taken as significant.

RESULTS

A total of 40 subjecte were enrolled. 26 (65%) males and 14 (35%) females were included.The mean age of the patients was 54.8 years. In patients with hyponatremia, it ranged from 113 to 129 meq/L. Hyponatremia was present in 16 (40%) subjects. 2 (5%) patients had mild hyponatremia, 7 (17.5%) had moderate, and 5 (12.5 %) had severe hyponatremia. HE was present in 25 (62.5%) patients. HE grade I was present in 6 (15%), grade II in 8 (20%), grade III in 7 (17.5%), and grade IV in 4 (10%) patients.

Table 1: Hyponatremia and its correlation with hepatic encephalopathy

Hyponatremia	Hepatic encephalopathy		Total	P-value
	Yes	No		<0.001*
Yes	10	6	16	
No	15	9	24	
Total	25	15	40	

* : Significant

Table 2: Correlation of severity of hyponatremia with grades of hepatic encephalopathy

Severity of hyponatremia	Grades of hepatic encephalopathy					Total	P-value
	I	II	III	IV	None		<0.001*
Mild	1	1	0	0	0	2	
Moderate	3	1	0	1	2	7	
Severe	0	0	4	1	0	5	
None	2	6	3	2	13	26	
Total	6	8	7	4	15	40	

*** :**Significant

DISCUSSION

In patients without cirrhosis, hyponatremia depending on its severity may lead to a range of symptoms including mild cognitive dysfunction, falls, seizures, coma and very rarely death.[9] Hyponatremia in cirrhosis is a chronic process and this allows the brain to adapt to the hypo-osmolality of the extracellular fluid. The most important factor in determining the severity of neurologic symptoms in patients with hyponatremia is the acuity of fall of serum sodium rather than the absolute reduction of serum sodium. Hence patients with cirrhosis and hyponatremia are less likely to have severe neurologic symptoms.[10] However, hyponatremia may pose a second osmotic hit to cerebral edema and astrocyte swelling, in addition to the astrocyte dysfunction caused by increased intracellular glutamine concentration from ammonia metabolism, thereby precipitating hepatic encephalopathy. [10] Hence, this study was conducted to assess the correlation between hyponatremia in hepatic encephalopathy of chronic liver disease.

In the present study, a total of 40 subjects were enrolled. 26 (65%) males and 14 (35%) females were included. The mean age of the patients was 54.8 years. In patients with hyponatremia, it ranged from 113 to 129 meq/L. Hyponatremia was present in 16 (40%) subjects. 2 (5%) patients had mild hyponatremia. A study by Younas A et al, Overall, the serum sodium levels of the subjects ranged from 115 to 142 meq/L with a mean of 129.11 ±6.53 meq/L. In patients with hyponatremia, it ranged from 115 to 127 meq/L (mean 121.41 ±5.17 meq/L). Hyponatremia was present in 96 (36.9%) patients. Among these, 51 (53.12%) were male and 45 (46.8%) were female; 24 (9.2%) patients had mild hyponatremia, 56 (21.5%) had moderate, and 16 (6.2%) had severe hyponatremia. HE was present in 176 (67.7%) patients. HE grade I was present in 54 (20.8%), grade II in 62 (23.8%), grade III in 32 (12.3%), and grade IV in 28 (10.8%) patients. In 96 patients with hyponatremia, 84 were found to have HE (p-value: <0.001). Conclusion Based on our findings, cirrhotic patients with chronic hepatitis infections have a variable presence of low sodium levels. Sodium levels of <130 meq/L were associated with higher morbidity and mortality rate. Moreover, patients with lower levels of sodium had higher grades of HE.[11]

In the present study, 7 (17.5%) had moderate, and 5 (12.5 %) had severe hyponatremia. HE was present in 25 (62.5%) patients. HE grade I was present in 6 (15%), grade II in 8 (20%), grade III in 7 (17.5%), and grade IV in 4 (10%) patients. Another study by Qureshi MO et al, Out of 202 patients, 62 (30.7%) patients had serum sodium less than 130 meq/l. Out of 202, HE was present in 69 (34.15%) patients and out of these, 38 had grade III-IV HE and 31 had grade I - II HE. Out of 69 patients with HE 57 had sodium less than 135 ($p < 0.001$). Hyponatremia was a common feature in patients with cirrhosis and its severity increased with the severity of liver disease. The existence of serum sodium concentration < 135 mmol/L was associated with greater frequency of hepatic encephalopathy compared with patients with serum sodium concentration > 135 mmol/L.[12]The risk of developing ascites, variceal bleeding, HE, and other cirrhosis-related complications is directly proportional to the degree/severity of hyponatremia. Various studies have shown that severe hyponatremia is associated with increased severity of HE.[13,14] The association between HE and hyponatremia may be explained based on the higher severity of liver disease among patients with sodium levels of <130 meq/L and the hypothesis that there might be a pathophysiological link between these two events. In a study by Cordoba et al., it was concluded that hyponatremia causes mild cerebral edema, which results in increased osmotic pressure on astrocytes. Eventually, it leads to many neurological dysfunctions. [15]In a study conducted by Udagani et al., it was revealed that cirrhotic patients with hyponatremia had a greater risk of developing neurological disorders as compared to those who had normal sodium levels.[16]

CONCLUSION

There exists a correlation between the hyponatremia in hepatic encephalopathy of chronic liver disease.

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