ORIGINAL RESEARCH

Analysis of serum sodium levels in liver cirrhosis patients: A Biochemical study

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ABSTRACT

Background: To assess serum sodium levels in liver cirrhosis patients. **Materials & methods:** A total of 100 patients were enrolled. Only those confirmed cases of cirrhosis of liver were enrolled. Complete demographic and clinical details were recorded. Grading of all the patients according to Child-Pugh Score grade was done as follows- Class A, Class B and Class C.serum levels were obtained and were the biochemistry lab where auto-analyser was used for evaluation of serum sodium levels. **Results:** Mean serum sodium levels were found to be 128.4 mmol/L. Hyponatremia was found to be present in 58 percent of the patients. While correlating the occurrence of hyponatremia with Child-Pugh score, significant results were obtained. **Conclusion:** Hyponatremia might indicate the existence of severe complications associated with liver cirrhosis.

Key words: Sodium, Hyponatremia, Cirrhosis

INTRODUCTION

Hyponatremia is common in patients with cirrhosis and portal hypertension and is characterized by excessive renal retention of water relative to sodium as a result of reduced solute-free water clearance. Hyponatremia may result from several factors related to cirrhosis and portal hypertension, the most prominent of which is increased release of arginine vasopressin (AVP; also known as antidiuretic hormone, or ADH).[1-3]

AVP release in portal hypertension is thought to occur via baroreceptor-mediated nonosmotic stimulation caused by a reduction in effective circulating volume, due in turn to arterial splanchnic vasodilation.[4]

According to several recent studies, hyponatremia occurring as a result of a reduced solute-free water clearance was a key prognostic factor in patients with liver cirrhosis when hyponatremia was incorporated into the MELD score. Hyponatremia is a common abnormal finding in approximately 57% of hospitalized patients with chronic liver disease and in 40% of outpatients with liver disease.[5]

even though patients with cirrhosis and serum sodium concentration between 130 and the lower normal limit of 135 mmol/L could not be considered as hyponatremic according to this definition, they present pathogenic and clinical features similar to those with serum sodium lower than 130 mmol/L. With the cutoff of 135 mmol/L, the prevalence of hyponatremia rises to almost 50%. Instead, the occurrence of severe hyponatremia, that is serum sodium concentration lower than 126 mmol/L, is rare and its prevalence is 6%.[6, 7]Hence; the present study was conducted for analysing serum sodium levels in liver cirrhosis patients.

MATERIALS & METHODS

The present study was conducted analysing serum sodium levels in liver cirrhosis patients. A total of 100 patients were enrolled. Only those confirmed cases of cirrhosis of liver were enrolled. Complete demographic and clinical details were recorded. Grading of all the patients according to Child-Pugh Score grade was done as follows- Class A, Class B and Class C.serum levels were obtained and were the biochemistry lab where auto-analyser was used for evaluation of serum sodium levels. All the results were recorded in Microsoft excel sheet followed by subjected to statistical analysis using SPSS software.

RESULTS

Mean age of the patients was 51.3 years. Significant proportion of patients were males. Alcohol was the major etiologic agent responsible for cirrhosis of liver

found to be present in 51 percent of the patients. Mean serum sodium levels were found to be 128.4 mmol/L. Hyponatremia was found to be present in 58 percent of the patients. While correlating the occurrence of hyponatremia with Child-Pugh score, significant results were obtained.

Table 1: Sodium levels

Sodium levels	Value (mmol/L)
Mean	128.4
SD	12.6

*: Significant

Table 2: Prevalence of hyponatremia

Hyponatremia	Number	Percentage
Present	58	58
Absent	42	42

Table 3: Correlation of hyponatremia with severity grading of child-Pugh score

Child Pugh class	Hyponatremia present	Hyponatremia absent	Total	p- value
Class A	11	12	23	0.001*
Class B	19	15	34	
Class C	28	15	43	
Total	58	42	100	

*: Significant

DISCUSSION

Hyponatremia is a common finding in patients with decompensated cirrhosis due to an abnormal regulation of body fluid homeostasis. Although hyponatremia in cirrhosis was described more than 50 years ago, its importance in the clinical assessment of patients with cirrhosis was overlooked for many years. Interest in hyponatremia was fostered by studies in the late 1970s and 1980s indicating that hyponatremia is an important prognostic indicator in cirrhosis. Recent studies extended these observations and showed that hyponatremia is an important marker of prognosis in both the pretransplant and posttransplant settings. Moreover, hyponatremia has also gained attention because of the discovery of vaptans, drugs that improve solute-free water excretion by antagonizing the effects of arginine vasopressin (AVP) in the renal tubules, which are currently being evaluated for the management of hyponatremia associated with cardiac failure, the syndrome of inappropriate antidiuretic hormone secretion, and cirrhosis.[8- 10]

In cirrhosis, total body water stores are increased, yet effective arterial volume is decreased. The decrease in effective arterial volume is a product of splanchnic arterial vasodilatation that is mediated by excessive production of nitric oxide and other vasodilator compounds, such as endotoxin, substance P, and endogenous cannabinoids, in the setting of increased intrahepatic resistance. This process leads to sodium avidity in the proximal portion of the nephron, by activation of the renin–angiotensin–aldosterone axis and excess ADH-mediated free water re-absorption in the collecting tubule. Arterial side baroreceptors, found in areas such as the left ventricle and the carotid sinus, have been shown to be a potent regulator of ADH secretion that can overcome the suppressive effects of hypo-osmolality. In the cirrhotic patient with ascites, the non-osmotic release of ADH from the posterior pituitary becomes the dominant force and the end result is impaired free water excretion and subsequent dilutional hyponatraemia.[10- 12]

In the present study, mean serum sodium levels were found to be 128.4 mmol/L. Hyponatremia was found to be present in 58 percent of the patients. While correlating the occurrence of hyponatremia with Child-Pugh score, significant results were obtained.Mohamed Zameer et al evaluated the association between serum sodium levels and presence and severity of cirrhotic complications. Data of 250 inpatients with cirrhotic complications were collected retrospectively. Based on the serum sodium concentration measured at the time of admission, patients were divided into three groups: serum sodium ≤130 mmol/L, serum sodium between 131 and 135 mmol/L, and serum sodium ≥136 mmol/L. The prevalence of dilutionalhyponatremia classified as serum sodium concentrations of ≤135 mmol/ was 56%. The serum sodium level was strongly associated with the severity of liver function impairment as assessed by Child-Pugh and MELD scores (P<0.0001). Sodium levels less than 130 mmol/L was associated with the occurence of massive ascites (P =0.003), grade III or higher hepatic encephalopathy (P =0.02), spontaneous bacterial peritonitis (P =0.001), and hepatic hydrothorax (P=0.004). Hyponatremia, especially serum levels <or=130 mmol/L, is associated with the existence of severe complications associated with liver cirrhosis.[12] Although hyponatremia can be found in patients with early or moderately advanced cirrhosis belonging to classes A and B of Child-Pugh classification, in most cases it occurs in an advanced disease (Child-Pugh class C). The relationship between hyponatremia and severity of cirrhosis is further evidenced by its close association with the occurrence of complications:

indeed, the prevalence of hepatic encephalopathy, hepatorenal syndrome and spontaneous bacterial peritonitis is substantially higher in patients with serum sodium concentration ≤130 mmol/L than in those with higher levels. Moreover, among patients with ascites, those with hyponatremia have a lower response to diuretics, a higher incidence of refractory ascites, and more often need therapeutic paracentesis at shorter intervals (Angeli, P et al, Ginès, P et al, Pugh, R.N et al).[13- 15] Kim JH et al evaluated the association between the serum sodium level and the severity of complications in liver cirrhosis.The prevalence of dilutional hyponatremia, classified as serum sodium concentrations of ≤135 mmol/L, ≤130 mmol/L, and ≤125 mmol/L, were 20.8%, 14.9%, and 12.2%, respectively. The serum sodium level was strongly associated with the severity of liver function impairment as assessed by Child-Pugh and MELD scores ($p<0.0001$). Even a mild hyponatremia with a serum sodium concentration of 131-135 mmol/L was associated with severe complications. Sodium levels less than 130 mmol/L indicated the existence of massive ascites, grade III or higher hepatic encephalopathy spontaneous bacterial peritonitis and hepatic hydrothorax.[16]

CONCLUSION

Hyponatremia might indicate the existence of severe complications associated with liver cirrhosis.

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