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Original Research Article.

Biochemical Evaluation of Serum Sodium Levels in Patients with Cirrhosis of Liver: An Institutional Based Study

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ABSTRACT

Background: To evaluate serum sodium levels in subjects with cirrhosis of liver.

Materials & Methods: A total of 100 subjects were enrolled. The subjects were divided into 2 groups cirrhosis group and control group as 50 in each. The results were analysed using SPSS software. The p- value less than 0.005 was considered significant.

Results: The mean age in subjects with serum sodium levels ≤130 mmol/L was 55.3 years and with 131-135 mmol/L concentration was 50.4 years. The mean serum sodium levels in control group were 141.7mmol/L and in cirrhosis group were 132.4mmol/L.

Conclusion: Lower serum sodium levels were associated with cirrhosis of liver.

Keywords: Liver Cirrhosis, Sodium Levels.

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INTRODUCTION

Reduced serum sodium concentration is a common finding in patients with cirrhosis[1,2], being the most common electrolyte disorder in this setting. Indeed, about 20% of patients have values lower than 130 mmol/L, which is the current definition of hyponatremia in cirrhosis.[3] However, 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%.[2]

Although hyponatremia can be found in patients with early or moderately advanced cirrhosis belonging to classes A and B of Child-Pugh classification, [4] 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.[2] Hyponatremia is the most common electrolyte abnormality observed in hospitalized patients.[5] Hyponatremia in cirrhosis is currently defined as a serum sodium level of less than 130 meq/L.[6] 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.[7] Hence, this study was conducted to evaluate serum sodium levels in subjects with cirrhosis of liver.

MATERIALS & METHODS

A total of 100 subjects were enrolled for the present study conducted in Department of Biochemistry, Krishna Mohan Medical College & Hospital, Mathura, Uttar Pradesh, India. The subjects were divided into 2 groups cirrhosis group and control group as 50 in each. The age group included was 45 to 60 years. Complete history was taken. Laboratory investigations were done. The data was collected. The results were analysed using SPSS software. The p- value less than 0.005 was considered significant.

RESULTS

A total of 100 subjects were enrolled. The mean age in subjects with serum sodium levels ≤130 mmol/L was 55.3 years and with 131-135 mmol/L concentration was 50.4 years. The p- value was 0.12. The control group had a serum sodium concentration as

≥136 mmol/L whereas, the subjects with cirrhosis had concentration ≤130 mmol/L and 131-135 mmol/L. The mean serum sodium levels in the control group were 141.7mmol/L and in cirrhosis group were 132.4mmol/L.

Table 1: Characteristics of patients according to serum sodium concentration

Characteristics	≤130 mmol/L	131-135 mmol/L	≥136 mmol/L	P- value
Gender				
Male	12	22	25	0.5
Female	6	10	25	
Age (years)	55.3	50.4	54.1	0.12

Table 2: Serum sodium levels

Serum sodium levels	Control group	Cirrhotic group	P – value
≤130 mmol/L	0	18	0.001
131-135 mmol/L	0	32	
≥136 mmol/L	50	0	

Table 3: Mean serum sodium concentration

Groups	Mean concentration (mmol/L)
Control group	141.7
Cirrhosis group	132.4

DISCUSSION

Hyponatremia is an electrolyte imbalance that commonly occurs in hospitalized patients. Most cases are dilutional hyponatremia caused by the impairment of solute-free water clearance. [8] Hyponatremia resulting from the impairment of solute-free water excretion is commonly accompanied by portal hypertension. [9] Hyponatremia is associated with a broad variety of neurological manifestations, whose intensity is related not only to the extent of serum sodium reduction, but also, and mainly, to the rate of fall. In fact, patients with acute hyponatremia have a much higher incidence of neurological symptoms than patients with chronic hyponatremia. [3] In patients without liver disease, the clinical effects of hyponatremia are related to brain edema, such as headache, disorientation, confusion, focal neurological deficits, seizures, and, in some cases, death due to cerebral herniation. [8] Moreover, hyponatremia leads to substantial changes in the brain intracellular environment to limit intracellular hyperhydration. These defense mechanisms consist of a rapid release of intracellular electrolytes, particularly potassium, which occurs within 24 h; subsequently, low-molecular-weight organic compounds, particularly myoinositol, are also discharged/released. [8] Hence, this study was conducted to evaluate serum sodium levels in subjects with cirrhosis of liver.

In the present study, a total of 100 subjects were enrolled. The mean age in subjects with serum sodium levels ≤130 mmol/L was 55.3 years and with 131-135 mmol/L concentration was 50.4 years. The p- value was 0.12. The control group had a serum sodium concentration as ≥136 mmol/L whereas, the subjects with cirrhosis had concentration ≤130 mmol/L and 131-135 mmol/L. A study by Kim JH et al, showed the prevalence of dilutional hyponatremia, classified as serum sodium concentrations of <or=135 mmol/L, <or=130 mmol/L, and <or=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 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 (OR, 2.685; CI, 1.316-5.477; p=0.007), grade III or higher hepatic encephalopathy (OR, 5.891; CI, 1.490-23.300; p=0.011), spontaneous bacterial peritonitis (OR, 2.562; CI, 1.162-5.653; p=0.020), and hepatic hydrothorax (OR, 5.723; CI, 1.889-17.336; p=0.002). Hyponatremia, especially serum levels <or=130 mmol/L, may indicate the existence of severe complications associated with liver cirrhosis.[10]

In the present study, the mean serum sodium levels in control group were 141.7mmol/L and in cirrhosis group were 132.4mmol/L. Another study by Bernardi M et al, showed patients with cirrhosis usually develop slowly progressing hyponatremia. In different clinical contexts, it is associated with neurological manifestations due to increased brain water content, where the intensity is often magnified by concomitant hyperammonemia leading to hepatic encephalopathy. Severe hyponatremia requiring hypertonic saline infusion is rare in cirrhosis. The management of asymptomatic or mildly symptomatic hyponatremia mainly rely on the identification and treatment of precipitating factors. However, sustained resolution of hyponatremia is often difficult to achieve. V2 receptor blockade by Vaptans is certainly effective, but their long-term safety, especially when associated to diuretics given to control ascites, has not been established as yet. As in other conditions, a rapid correction of long-standing hyponatremia can

lead to irreversible brain damage. The liver transplant setting represents a condition at high risk for the occurrence of such complications. [11] Another by John S et al, depicted that hyponatremia is frequently seen in patients with ascites secondary to advanced cirrhosis and portal hypertension. The development of ascites in patients with cirrhosis is multi-factorial. Portal hypertension and the associated systemic vasodilation led to activation of the sodium-retaining neurohumoral mechanisms which include the renin-angiotensin-aldosterone system, sympathetic nervous system and antidiuretic hormone (ADH). The net effect is the avid retention of sodium and water to compensate for the low effective circulatory volume resulting in the development of ascites. Although not apparent in the early stages of cirrhosis, the progression of cirrhosis and ascites leads to impairment of the kidneys to eliminate solute- free water. This leads to additional compensatory mechanisms including non-osmotic secretion of ADH, also known as arginine vasopressin, further worsening excess water retention and thereby hyponatremia. Hyponatremia is associated with increased morbidity and mortality in patients with cirrhosis and is an important prognostic marker both before and after liver transplant. The management of hyponatremia in this setting is a challenge as conventional therapy for hyponatremia including fluid restriction and loop diuretics are frequently inefficacious. [12]

CONCLUSION

Lower serum sodium levels were associated with cirrhosis of liver.

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