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ORIGINAL ARTICLE

Frequency of Complications by Serum Sodium Level Among Patients with Liver Cirrhosis

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ABSTRACT

Objective: Aim of current study is to determine the prevalence of complication among patients with liver cirrhosis by serum sodium level.

Study Design: Case-Control study

Place and Duration: DHQ Hospital Faisalabad.June 2020-Dec 2020

Methods: There were one hundred and sixty patients of both sexes had liver disease cirrhosis with ages 18-75 were included. Age, gender, height and weight, BMI, and other medical conditions were collected after informed written permission was obtained from each participant. The blood sodium levels and severity of problems of 160 inpatients were evaluated. Child Pugh class was implemented. SPSS 24.0 version was used to analyze complete date.

Results: In our study most of the cases 102 (63.8%) were males and 58 (36.2%) females. Age of the patients was 53.6±11.28 years and mean BMI was 25.4±8.31 kg/m^2.HBV was the most common cause of liver disease found in 95 (59.8%) cases followed alcohol in 40 (25%) cases. Mean MELD score was 13.9±6.44. Most of the cases had child-pugh class C 80 (50%). We found that majority of the cases 79 (49.4%) had serum sodium level (>135meq/l) followed by hyponatraemia (<130meq/l)) in 42 (26.3%) and 39 (24.4%) patients had serum sodium level (131-135meq/l). We found ascites was the most common complication found in 132 (82.5%) patients with, followed by esophageal varix and hepatic encephalopathy. Severity of complications was significantly found in cases of hyponatraemia.

Conclusion: As a result, we came to the conclusion that hyponatremia, particularly serum levels less than 130 mmol/L, might suggest the presence of serious problems associated with liver cirrhosis.

INTRODUCTION

In patients with advanced cirrhosis, intractable ascites, severe hyponatremia, and reduced arterial pressure are all common clinical findings.. Portal hypertension, impaired vascular responsiveness to vasoactive medications, and decreased solute-free water clearance are all contributing factors in the development of these disorders [1,2]. Patients with liver cirrhosis who had hyponatremia due to impaired clearance of solute-free water were more likely to die than those with normal clearance of water [3,4]. The prevalence of hyponatremia in patients with chronic liver disease is around 57% in the hospitalised and 40% in outpatients [5]. The frequency of hyponatremia in Korean hospitalised patients with liver cirrhosis has not been studied to far, however. Because of this lack of research, it is impossible to say whether or not serum sodium levels are linked to liver cirrhosis consequences.

When extracellular fluid volume is increased, hyponatremia is more likely to develop. Diuretic usage or gastrointestinal losses may lead to hypovolemic hyponatraemia in certain patients, although this is not the norm. This should be the only or a key contributing factor when assessing a cirrhotic patient with low sodium serum levels.

Cirrhosis causes a reduction in effective arterial volume despite an increase in total body water storage [6]. Excessive nitric oxide generation, as well as other potent vasodilators such endotoxin and substance P, as well as endogenous cannabinoids, are all involved in the reduction in splanchnic arterial blood volume [7, 8]. Due to stimulation of the renal angiotensin–aldosterone axis (RAA) and excess ADH-mediated absorption of free water by ADH in the collecting tubule (CTU), this process leads to increased sodium-avidity in the proximal region of the kidney, which results in increased sodium excretion in the CTU. Baroreceptors present in the left ventricle and carotid sinus were discovered to be a powerful regulator of ADH production that may override hypo-osmolality-induced suppression [6]. It is the non-osmotic anterior pituitary release of ADH that becomes the dominating factor in the cirrhotic patient with ascites. As a consequence, there is impaired free water excretion and dilutional hyponatremia.

Hepatic encephalopathy may be triggered by hyponatremia, however the exact process is still a mystery. Osmotic gradients between fluid compartments in extracellular space have been hypothesised to cause astrocyte enlargement [9] When a person has hyponatremia, they are eight times more likely to get hepatitis E. [11] The degree of hyponatremia is strongly connected to the worsening of HE grades. In individuals with chronic liver disease, recent investigations have shown that hyponatremia is a significant prognostic factor.[12]

Our study of liver cirrhosis-related complications and the prevalence of hyponatremia included measurements of serum sodium levels to see whether they were linked to cirrhotic problems or their severity, as well as survival rates for patients hospitalized with these problems.

MATERIAL AND METHODS

This Case-control study was conducted at DHQ Hospital Faisalabad and comprised of 160 patients. Age, gender, height and weight, BMI, and other medical conditions were collected after informed written permission was obtained from each participant. Exclusion criteria included having hepatocellular cancer at the time of admission, taking diuretics during the previous month, or throughout the course of the study.

A diagnosis of liver cirrhosis was only made in situations when the relevant diagnostic criteria were satisfied. Hepatitis B surface antigen and anti-HCV antibodies were used to identify individuals with chronic hepatitis B as causal factors in the development of cirrhosis of the liver. In the absence of additional causal variables, such as the use of medications or the presence of a viral infection, patients who had consumed an average of 80 g of alcohol per day for more than ten years were considered to have alcoholic liver disease. Cases that didn't fit into any of the other categories were labelled as such. Patients were separated into three categories based on their blood sodium content at the time of admission: sodium 130 mmol/L, sodium between 131 and 135 mmol/L, and sodium 136 mmol/L. Some of the most common side effects were simple ascites and persistent ascites, as well as symptoms such hepatic encephalopathy and intractable ascites, as well as bacterial peritonitis, hepatic hydrothorax, and hepatic infection. Ascites, hepatic encephalopathies, and esophageal varices were categorized depending on severity. Ascites was categorised into three grades: grade I, where ascites was detected on imaging but its presence was unclear on physical examination;

grade II, where ascites was easily recognized on gross examination and palpation; and grade III, where ascites was present on gross examination and large-volume paracentesis was performed for therapeutic purposes. The whole data set was analyzed using SPSS 24.0.

RESULTS

In our study most of the cases 102 (63.8%) were males and 58 (36.2%) females. Age of the patients was 53.6±11.28 years and mean BMI was 25.4±8.31 kg/m^2.HBV was the most common cause of liver disease found in 95 (59.8%) cases followed alcohol in 40 (25%) cases. Mean MELD score was 13.9±6.44. Most of the cases had child-pugh class C 80 (50%).(table 1)

Table 1: Characteristics details of enrolled cases

Variables	Frequency	Percentage
Gender		
Male	102	63.8
Female	58	36.2
Mean age (years)	53.6±11.28	
Mean BMI (kg/m^2)	25.4±8.31	
Causes		
HBV	95	59.8
HCV	25	15.6
Alcohol	40	25
Child Pugh Class		
A	20	12.5
B	60	37.5
C	80	50
Mean MELD	13.9±6.44	

We found that majority of the cases 79 (49.4%) had serum sodium level (>135meq/l) followed by hyponatraemia (<130meq/l)) in 42 (26.3%) and 39 (24.4%) patients had serum sodium level (131-135meq/l).(fig 1)

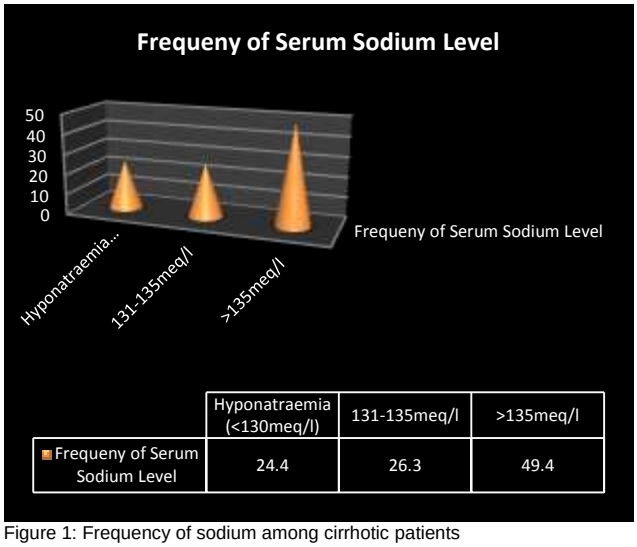

Figure 1: Frequency of sodium among cirrhotic patients

We found ascites was the most common complication found in 132 (82.5%) patients with, followed by esophageal varix and hepatic encephalopathy.(table 2)

Table 2: Association of complication among enrolled cases

Complications	Frequency	Percentage
Ascites		
Yes	132	82.5
No	28	17.5
Esophageal varix		
Yes	120	75
No	40	25
Hepatic encephalopathy		
Yes	50	31.3
No	110	68.7
Variceal Bleeding		
Yes	45	28.1
No	135	71.9
Gastric Varix		
Yes	40	25
No	120	75
Bacterial Peritonitis		
Yes	35	21.9
No	125	78.1
Hepatorenal Syndrome		
Yes	12	7.5
No	148	92.5

Severity of complications was significantly found in cases of hyponatraemia.(table 3)

Table 3: Severity of complication with according to serum sodium level

Complications	<130meq/l	131-135meq/l	>135meq/l
Ascites			
Severe	25	22	33
Non	17	19	46
Hepatic encephalopathy			
Severe	20	15	15
Non	20	22	46
Esophageal varix			
Severe	30	28	62
Non	12	11	17

DISCUSSION

Patient hyponatremia is a frequent complication of hospitalization. The most common cause of dilutional hyponatremia is impaired clearance of free water due to a deterioration in solute concentrations. [13] Hypertension of the portal vein is often seen in patients with hyponatremia caused by impaired excretion of solute-free water. Having hyponatremia as a potential prognostic factor for liver cirrhosis has gotten a lot of attention in the last several years.[14] To yet, no Korean research have examined its prevalence or the concomitant existence of its problems. Hyponatremia and associated consequences in Korean patients with liver cirrhosis were the focus of this investigation.

In our study 160 patients of both genders with ages 18-75 years were included. 102 (63.8%) were males and 58 (36.2%) females. Age of the patients was 53.6±11.28 years and mean BMI was 25.4±8.31 kg/m^2.HBV was the most common cause of liver disease found in 95 (59.8%) cases followed alcohol in 40 (25%) cases. Mean MELD score was 13.9±6.44. Most of the cases had child-pugh class C 80 (50%).These results were comparable to the previous studies.[15,16] We found that majority of the cases 79 (49.4%) had serum sodium level (>135meq/l) followed by hyponatraemia (<130meq/l)) in 42 (26.3%) and 39 (24.4%) patients had serum sodium level (131-135meq/l). Hyponatraemia 130 was detected in 30 percent of patients by Arroyo et al. [17] There was also a 24.9 percent decrease (between 132-135 meq/l) in the prevalence of low blood sodium levels in our research, which is a higher percentage than previously reported. Even though it is well accepted that ascites is more difficult to cure if the blood sodium concentration is below 130 meq/l, few research have examined the association between serum sodium levels and ascites responsiveness to diuretic treatment. It has been shown that individuals with blood sodium levels under 130 meq/l had worse glomerular filtration rates and solute-free clearance, as well as a poorer response to diuretics than those whose sodium levels are higher. It was later discovered[18] that people who do not react to diuretics had lower salt levels in their blood than those who do. Patients with a salt content in their blood below 130 milligrammes per litre (mg/dL) had a higher incidence of refractory ascites, a lesser response to weight loss therapy, a greater need for large-

volume paracentesis, and a shorter time between paracentesis procedures.

We found ascites was the most common complication found in 132 (82.5%) patients with, followed by esophageal varix and hepatic encephalopathy. There is a clear correlation between hyponatremia severity and the likelihood of developing ascites, variceal haemorrhage, HE, and other cirrhosis-related consequences. Severe hyponatremia has been linked to a worsening of HE in many investigations [19,20]. It is possible that the relationship between HE and hyponatremia may be explained by the greater severity of liver disease in individuals who have a low amount of salt in their blood. Hyponatremia induces minor cerebral edoema, according to a research by Cordoba et al., which leads to increased osmotic pressure on astrocytes. It eventually causes a wide range of neurological issues. [21] Severity of complications was significantly found in cases of hyponatraemia. Other research have reached similar conclusions, and this one does as well. According to a research by Angeli et al., 38% of patients with severe hyponatremia developed HE, but only 24% of those with moderate hyponatremia did.[22]

CONCLUSION

As a result, we came to the conclusion that hyponatremia, particularly serum levels less than 130 mmol/L, might suggest the presence of serious problems associated with liver cirrhosis.

REFERENCES

1. Angeli P, Wong F, Watson H, Gines P. Hyponatremia in cirrhosis: results of a patient population survey. Hepatology. 2006;44:1535–1542
2. Fernandez-Esparrach G, Sanchez-Fueyo A, Gines P, et al. A prognostic model for predicting survival in cirrhosis with ascites. J Hepatol. 2001;34:46–52
3. Cosby RL, Yee B, Schrier RW. New classification with prognostic value in cirrhotic patients. Miner Electrolyte Metab. 1989;15:261–266
4. Fernandez-Esparrach G, Sanchez-Fueyo A, Gines P, et al. A prognostic model for predicting survival in cirrhosis with ascites. J Hepatol. 2001;34:46–52
5. Gines P, Berl T, Bernardi M, et al. Hyponatremia in cirrhosis: from pathogenesis to treatment. Hepatology. 1998;28:851–864.
6. Schrier RW. Water and sodium retention in edematous disorders: Role of vasopressin and aldosterone, Am J Med, 2006, vol. 119 7 Suppl 1(pg. S47-53)
7. Ginès P, Cárdenas A. The management of ascites and hyponatremia in cirrhosis, Semin Liver Dis, 2008, vol. 28 (pg. 43-58)
8. Iwakiri Y. The molecules: Mechanisms of arterial vasodilatation observed in the splanchnic and systemic circulation in portal hypertension, J Clin Gastroenterol, 2007, vol. 41 Suppl 3(pg. S288-94)
9. Tivers MS, Handel I, Gow AG, Lipscomb VJ, Jalan R, Mellanby RJ: Hyperammonemia and systemic inflammatory response syndrome predicts presence of hepatic encephalopathy in dogs with congenital portosystemic shunts. PloS One. 2014, 9:e82303.
10. Yun BC, Kim WR: Hyponatremia in hepatic encephalopathy: an accomplice or innocent bystander? . Am J Gastroenterol. 2009, 104:1390-1391.
11. Angeli P, Wong F, Watson H, Ginès P; CAPPS Investigators: Hyponatremia in cirrhosis: results of a patient population survey. Hepatology. 2006, 44:1535-1542. 1
12. Shaikh S, Mal G, Khalid S, Baloch GH, Akbar Y: Frequency of hyponatraemia and its influence on liver cirrhosis-related complications. J Pak Med Assoc. 2010, 60:116-120.
13. Adrogué HJ, Madias NE. Hyponatremia. N Engl J Med. 2000;342:1581–1589
14. Gines P, Berl T, Bernardi M, et al. Hyponatremia in cirrhosis: from pathogenesis to treatment. Hepatology. 1998;28:851–864
15. Kim JH, Lee JS, Lee SH, et al. The association between the serum sodium level and the severity of complications in liver cirrhosis. Korean J Intern Med. 2009;24(2):106-112.
16. Younas A, Riaz J, Chughtai T, et al. (February 06, 2021) Hyponatremia and Its Correlation With Hepatic Encephalopathy and Severity of Liver Disease. Cureus 13(2): e13175.
17. Arroyo V. Hecker R, Sherlock S. Electrolyte and circulatory changes in terminal liver failure (Lancet 1956;2:1221-1225). J Hepatol 2002; 36: 315-20.
18. Bernardi M, Laffi G, Salvagnini M, Azzena G, Bonato S, Marra F, et al.Efficacy and safety of the stepped care medical treatment of ascites in live rcirrhosis: a randomized controlled clinical trial comparing two diets withdifferent sodium content. Liver 1993; 13: 156-62
19. Barakat AA, Metwaly AA, Nasr FM, El-Ghannam M, El-Talkawy MD, Taleb HA: Impact of hyponatremia on frequency of complications in patients with decompensated liver cirrhosis. Electron Physician. 2015, 7:1349-1358
20. Guevara M, Baccaro ME, Ríos J, et al.: Risk factors for hepatic encephalopathy in patients with cirrhosis and refractory ascites: relevance of serum sodium concentration. Liver Int. 2010, 30:1137-1142.
21. Cordoba J: Hepatic encephalopathy: from the pathogenesis to the new treatments . ISRN Hepatol. 2014, 2014:236268.
22. 8. Angeli P, Wong F, Watson H, Ginès P; CAPPS Investigators: Hyponatremia in cirrhosis: results of a patient population survey. Hepatology. 2006, 44:1535-1542