COMPARISON OF SERUM SODIUM LEVELS IN COMPENSATED AND DECOMPENSATED CIRRHOSIS SECONDARY TO CHRONIC VIRAL HEPATITIS C

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ABSTRACT

Objective: To compare the serum sodium levels in compensated (Child Pugh A) and decompensated cirrhosis (Child Pugh C) secondary to chronic viral hepatitis C.

Study Design: Comparative descriptive study.

Place and Duration of Study: The study was carried out in Department of Medicine Military Hospital Rawalpindi over six months from July to Dec 2012.

Material and Methods: A Total of 100 consecutive patients of cirrhosis secondary to viral hepatitis C infection fulfilling the inclusion criterion were taken from wards and emergency department of hospital. Two groups were made depending on examination and laboratory investigation. Group A comprising of patients with decompensated cirrhosis in Child Pugh class C and group B of patients with compensated cirrhosis in Child Pugh class A. Venous blood sample of each patient was collected for serum sodium levels and was transported to Armed Forces Institute of Pathology immediately for analysis.

Results: Serum sodium level in group A comprising of 60 patients with decompensated cirrhosis ranged from 120 to 145 mmol/with a mean value of 133.03 ± 6.70 whereas serum sodium level in group B comprising of 40 patients with compensated cirrhosis ranged from 132 to 145 mmol/l with a mean sodium level of 140.30 ± 3.20mmol/L. Difference was statistically significant (p=0.010).

Conclusion: Mean serum sodium levels were lower in patients with decompensated cirrhosis as compared to compensated cirrhosis.

Keywords: Compensated cirrhosis, Chronic viral hepatitis C, Decompensated cirrhosis, Serum sodium level.

INTRODUCTION

Hepatitis C viral infection is rapidly increasing worldwide. It has been called "The Viral Time Bomb" by the World Health Organization[1]. It is the most important chronic viral infection in Pakistan which is now the second largest country in terms of positive seroprevalance of hepatitis C. The reason of its rapid spread being the lack of awareness of its transmission factors and inadequate preventive measures[2].

HCV infection has a high morbidity because it becomes chronic in 50 to 70% of cases out of which 20 to 30% develop cirrhosis[3]. This cirrhosis which is initially compensated can become decompensated over time and patients can develop complications like ascites, variceal haemorrhage, hepatic encephalopathy, spontaneous bacterial peritonitis and hepatocellular carcinoma[4]. Patients with decompensated cirrhosis have a higher Child Pugh class and have repeated hospital visits and admissions which have considerable social and economic consequences.

In these decompensated cirrhotic patients, hyponatremia is a frequent electrolyte imbalance and a marker of poor prognosis. It is associated in a graded fashion with severe ascites, increased rate of spontaneous bacterial peritonitis, hepatic encephalopathy, and hepatorenal syndrome[5]. This association of hyponatremia with liver failure complications has been shown by several international studies and because of this reason it is now thought important to monitor the serum

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Received: 17 Jul 2014; revised received: 26 Mar 2015; accepted: 20 Apr 2015

sodium concentrations in patients with cirrhosis in order to prevent the rapid development of cirrhosis related complications. Studies are being carried out internationally to see the effect of treating hyponatremia by vasopressin receptor blockers in patients of cirrhosis with complications[6,7]. Also there is an ongoing debate to add sodium levels as a variable in Model for End-Stage Liver Disease (MELD) scoring system as it is a strong marker to predict and prioritize the need for liver transplantation in patients with decompensated cirrhosis[9]. This study is aimed to compare the serum sodium levels in compensated and decompensated cirrhosis secondary to chronic viral hepatitis C.

MATERIAL AND METHODS

This comparative descriptive study was carried out at Department of Medicine, Military Hospital Rawalpindi from July to Dec 2012. Administrative permission from concerned authorities and ethical committee was taken. Cirrhotic patients, aged between 30 to 70 years regardless of gender, ethnicity, religion and socioeconomic status with evidence of hepatitis C infection by positive PCR test, evidence of cirrhosis liver on ultrasound and of Child Pugh class A or C were included in study. Patients with Child Pugh class B cirrhosis, chronic kidney disease, congestive cardiac failure, neuropsychiatric disorder, and hepatocellular carcinoma were excluded from study. One hundred patients were included in the study through nonprobability consecutive sampling. Informed consent was taken from the patients except for cases in hepatic encephalopathy where it was taken from the attendant of the patient. Patients were placed in two groups depending upon clinical examination and laboratory investigations. Group A comprising of patients with decompensated cirrhosis in Child Pugh class C and group B comprising of patients with compensated cirrhosis in Child Pugh class A.

Venous blood samples of the selected patients were collected for serum sodium levels using full aseptic measures and were immediately transported to Armed Forces Institute of Pathology for analysis. Serum sodium level were estimated done by automated analyzer using ion selective electrodes. Clinical information proforma was filled against each patient pertaining to the relevant clinical details. It included the patient's demographic and diagnostic profile. Each patient's Child Pugh class and serum sodium levels were entered in it.

SPSS 17 was used for data analysis. Mean and standard deviation was calculated for age and serum sodium levels. Independent sample t-test was used to compare quantitative variables. Frequency and percentages were calculated for qualitative variables. *P*-value of <0.05 was considered as significant.

RESULTS

A total of 100 cirrhotic patients with chronic viral hepatitis C were included in the study. Out of 100 patients 67% (67) were male and 33% (33) were females. The age of patients ranged from 30 to 70 years. Mean age in group A was 50.9 ± 11.95 and in group B it was 48.32 ± 11.067. Both the groups were comparable with respect to age (p=0.2788) and gender (p=0.3902) (table-I). Range of serum sodium levels in group A was 120 to 145 mmol/l with a mean value of 133.03 ± 6.70mmol/L whereas in group B serum sodium levels ranged from 132 to 145 mmol/l with mean sodium level of 140.30 ± 3.20mmol/L. Serum sodium levels were significantly lower in group A as compared to group B (p=0.010) (table-II).

DISCUSSION

Hyponatremia is the most frequent electrolyte imbalance seen in patients with major organ failures especially in patients who are hospitalized. In cirrhotic patients with decompensated liver hyponatremia is a common finding. It is negatively associated with complications like ascites, Spontaneous Bacterial Peritonitis (SBP), encephalopathy and hepatorenal syndrome. The lower the serum sodium levels the more severe the complications which are difficult to treat. It acts as a prognostic marker and guides towards patients worsening

condition[10]. Due to the association of hyponatremia with adverse prognosis in cirrhosis it is being considered to add levels of serum sodium. Maintaining the serum sodium levels may prevent the development of the above

came from all parts of the country and from all social strata.

There is a growing interest in clinicians about role of hyponatremia in cirrhosis and studies are being carried out worldwide. Several

Table-I: Comparison of demographic characteristics of patients in both groups.

Parameters		Group A (n=60)	Group B (n=40)	*p*-value
Gender	Male	38	29	p=0.3902
	Female	22	11	
Mean Age		50.9 ± 11.95	48.32 ± 11.067	*p*=0.2788

Table-II: Comparison of mean serum sodium levels in both groups.

Parameter	Group A (n=60)	Group B (n=40)	*p*-value
Mean Sodium levels	133.03 ± 6.70	140.30 ± 3.20	*p*=0.010

mentioned complications of liver cirrhosis. Treatment of hyponatremia is warranted when levels are too low or the patient is having overt symptoms. Studies are being carried out internationally to see the role of VAPTANS, vasopressin receptor antagonists in the treatment of hyponatremia and their effect on prevention of liver cirrhosis related complications[8]. Sodium levels as a variable to MELD scoring system as it can better predict the need for liver transplantation and to prioritize patients on waiting list which is evident by several international studies[11,12].

This study was done to emphasize upon the presence of low sodium levels in decompensated cirrhosis, however it has few limitations. It lacks the assessment of correlation of hyponatremia with complications of decompensated cirrhosis like refractory ascites, spontaneous bacterial peritonitis, encephalopathy, variceal heamorrage and hepatorenal syndrome. It also does not throw light on the effect of sodium replacement in the management of these complications including their prevention. Moreover it is emphasized that the data cannot be easily applied to whole of population of cirrhotic patients as we did the study in a highly selected group of patients with cirrhosis secondary to viral hepatitis C. However, the study population is a representative sample of the general population of patients with cirrhosis in Pakistan and the tested population

studies have shown negative impact of hyponatremia on prognosis of cirrhosis. Angeli P et al carried out a large study on analyzing hyponatremia in cirrhosis with prospective data on 997 patients. Results showed high frequency of hyponatremia in refractory as cites, hepatic encephalopathy, Spontaneous Bacterial Peritonitis (SBP) and hepatorenal syndrome (HRS)[13]. When compared to that study our study lacks the assessment of correlation of hyponatremia with each of the above mentioned complications.

A case control study carried out at Liaqat University Hospital Hyderabad on 217 cirrhotic patients also showed increased frequency of hyponatremia in patients with decompensated cirrhosis as compared to those having normal sodium levels[14]. Similarly, a study published in Korean Journal of Internal Medicine, carried out on 188 cirrhotic patients reported higher frequency of complications of cirrhosis with lower sodium levels[15]. In all of the above mentioned studies patients were divided into three groups based on sodium levels and results showed that patients with normal sodium levels had no or less complications as compared to those with lower sodium levels.

Alam et al studied all factors which precipitate hepatic encephalopathy and hyponatremia was a significant factor[16]. In a study carried out at dept of Med Isra University

and Liaquat University of Medical & Health Sciences (LUMHS) Jamshoro/Hyderabad on 100 patients with cirrhosis, hyponatremia was present in 35%. The study concluded that decreased sodium level is a major risk factor for mortality while normal sodium or mild hyponatremia had no effect[17].

Different studies indicate relationship of hyponatremia with cirrhosis related complications and its role as a prognostic marker. Studies should be done to assess the effect of treatment or prevention of hyponatremia on these complications. It is important to see whether preventing hyponatremia can delay the onset or progression of these complications.

CONCLUSION

Mean serum sodium levels were substantially lower in decompensated as compared to compensated cirrhosis.

CONFLICT OF INTEREST

This study has no conflict of interest to declare by any author.

REFERENCES

1. Waheed Y,Shafi T,Safi SZ,Qadri I.Hepatitis C virus in Pakistan: A systematic review of prevalence, genotypes and risk factors.World J Gastroenterol. 2009; 7; 15(45): 5647-5653.
2. Umar M, Bushra HT,Ahmed M,Khurram M, Usman S, Arif M et al. Hepatitis C in Pakistan: A Review of the Available Data. Hepat Mon. 2010 Summer; 10(3): 205-214.
3. Amico GD, Garua-Tsao G, Pagliavo L.Natural history and prognostic indicators of survival in cirrhosis: A systematic review of 118 studies. Journal of Hepatology 2006; 44: 217-231.
4. Al-Busafi SA, McNabb-Balter J, Farag A, Hilzenrat N. Clinical Manifestations of Portal hypertension. International Journal of Hepatology 2012; 12: 56-66.
5. Gines P, Guevara M. Hyponatremia in cirrhosis: pathogenesis, clinical significance and management.Hepatology 2008; 48: 1002-1010.
6. Cardenas A, Gines P, Marotta P, Czerwiec F, Oyuang J, Guevara M, Afdhal N. The use of vaptans in hyponatremic patients with liver cirrhosis. The Korean Journal of Hepatology 2011; 17: 335-337.
7. Boyder TD, Tolvaptan and hyponatremia in a patient with cirrhosis. Hepatology 2010; 51: 699-702.
8. Gines P. The vaptans:a promising therapy in the management of advanced cirrhosis . J Hepatol 2007; 46: 1150-1152.
9. Ruf AE, Kremers WK, Chavez LL, Descalzi VI, Podesta LG, Villamil FG. Addition of serum sodium into the MELD score predicts waiting list mortality better than MELD alone. Liver Transpl 2005; 11: 33.
10. Jenq CC, Tsai MH, Tian YC,Chang MY, Lin CY, Lien JM et al. Serum sodium predicts prognosis in critically ill cirrhotic patients. J Clin Gastroenterol. 2010; 44: 220-6
11. Luca A, Angermayr B, Bertolini G , Koeing F, Vizzini G, Ploner M et al. An integrated MELD Model including serum sodium and age improves the prediction of early mortality in patients with cirrhosis. Liver transpl 2007; 13: 1174-1180.
12. Wong VW, Chim AM, Wong GL, Sung JJ, Chan HL. Performance of the new MELD-Na score in predicting 3-month and 1-year mortality in Chinese patients in chronic hepatitis B. Liver Transpl 2007; 13: 1228-1235.
13. Angeli P, Wong F, Watson H, Gines P. Hyponatremia in cirrhosis:results of a patient population survey. Hepatology 2006; 44: 1535-42.
14. Shaikh S, Mal G, Khalid S, Baloch GH, Akbar Y. Frequency of hyponatremia and its influence on liver cirrhosis related complications. J Pak Med Assoc. 2010; 60: 116-120.
15. Kim JH, Lee JS, Lee SH, Bae WK, Kim NH, Kim KA et al. The association between the serum sodium level and the severity of complications in liver cirrhosis.Korean J Intern Med, 2009; 24: 106-112.
16. Alam I, Razaullah, Haider I, Hamayun M. Taqueem A, Nisar M. Spectrum of precipitating factors of hepatic encephalopathy in liver cirrhosis. Pak J Med Res 2005; 44(2): 96-100.
17. Siddiqui AA, Ujjan I,Shaikh MA. Impact of hyponatremia on the complications of liver cirrhosis, MC 2012; 19(2): 76-80.