e-ISSN: 0975-1556, p-ISSN:2820-2643

Available online on www.ijpcr.com

International Journal of Pharmaceutical and Clinical Research 2024; 16(5); 1681-1682

Original Research Article

The Significance of Hyponatremia as Poor Prognostic Markers in Patients with Chronic Liver Disease

Rajeev Kumar¹, Janmeshwar Prasad², Shiv Kumar³

¹Senior Resident, Department of General Medicine, AIIMS, Patna ²Assistant Professor, Department of General Medicine, AIIMS, Patna ³Senior resident, Department of General Medicine, AIIMS, Patna

Received: 11-02-2024 / Revised: 14-03-2024 / Accepted: 10-04-2024

Corresponding Author: Dr. Janmeshwar Prasad

Conflict of interest: Nil

Abstract:

Background and Objectives: Hyponatremia in advanced cirrhosis results from the hemodynamic complications associated withworsening portal hypertension, primarilyintravascular hypovolemia and renal hypoperfusion in the setting of total body volumeoverload. To study the hyponatermia and hypoalbuminemia as a poor prognostic marker in chronic liver disease patients.

Methods: Patients with chronic liver disease were recruited from medical wards after obtaining ethical clearance and written informed consent from the patient. Department of General medicine, AIIMS Patna.

Result: Serum albumins were lower among patients who died than survivors. Serum sodium was lower among patients who died than survivors. Serum potassium was higher among patients who died than survivors.

Conclusion: We concluded that hyponatermia and hypoalbuminemia was a poor prognostic marker in chronic liver disease patients

Keywords: K, Na, Albumin.

This is an Open Access article that uses a funding model which does not charge readers or their institutions for access and distributed under the terms of the Creative Commons Attribution License (http://creativecommons.org/licenses/by/4.0) and the Budapest Open Access Initiative (http://www.budapestopenaccessinitiative.org/read), which permit unrestricted use, distribution, and reproduction in any medium, provided original work is properly credited.

Introduction

Chronic liver disease (CLD) is a progressive deterioration of liver functions for more than sixmonths. which includes synthesis of clotting factors, other proteins, detoxification of harmful products of metabolism, and excretion of bile. CLD is a continuous process ofinflammation, destruction, and regeneration of liver parenchyma, which leads to fibrosis and cirrhosis. The spectrum of etiologies is broad forchronic liver disease, which includes toxins, alcohol abuse for a prolonged time, infection, autoimmune diseases, genetic and metabolic disorders. Cirrhosis is a final stage of chronic liver disease that results in disruption of liver architecture, the formation of widespreadnodules, vascular reorganization, neo- angiogenesis, and deposition of an extracellular matrix. The underlying mechanism of fibrosis and cirrhosis at a cellular level is the recruitmentof stellate cells and fibroblasts. resulting in fibrosis, while parenchymal regeneration relieson hepatic stem cells. Chronic liver disease is anextremely common clinical condition, and thefocus is done on the common etiologies, clinical manifestations, and management. [1-2]

Hyponatremia in advanced cirrhosis results from the hemodynamic complications associated withworsening portal hypertension, primarily intravascular hypovolemia and renal hypoperfusion in the setting of total body volume overload. Furthermore, the hepatic synthetic dysfunction associated with cirrhosis leads to abnormally low serum levels of albumin, a negatively charged protein that helps maintain adequate plasma oncotic pressure. A therapy for intravascular volume expansion in cirrhosis was introduced as early as the 1950s, and has been shown in small studies to be superior to normal saline or fluid restriction for correcting serum sodium in cirrhotics. [3]

Material and Methods

Observational longitudinal follow up study. Department of General Medicine at All India Institute of Medical Sciences, Patna. Patients with chronic liver disease were recruited from medical wards after obtaining ethical clearance and written informed consent from the patient.

Inclusion Criteria

- Patients of more than 18 years of age.
- Adult patients diagnosed with chronic liver disease

Exclusion Criteria

- Patients with ascites due to tuberculosis or malignancy.
- Patients with malignancies other thanhepatocellular carcinoma.

Data analysis

Interpretation and analysis with comparison of obtained results was carried out and data thus collected were subjected to descriptive statistical analysis of patients with chronic liver disease using SPSS 19, Student's T-test was used for comparison of continuous data. p- value <0.05 was considered significant. All repoted P values are two sided.

Results

Age range of patients was from 20 to 80 years. Mean age of patients was 48.94 ± 12.63 years. Male preponderance was seen in our series. Maximum number of patients of chronic liver disease were in the age group of 51 to 60 years.

e-ISSN: 0975-1556, p-ISSN: 2820-2643

Table 1: Mortality among CLD patients

| Outcome | No. of Patients | Percentage |
|---------|-----------------|------------|
| Alive | 170 | 85.00 |
| Expired | 30 | 15.00 |

Out of total 200 patients 15.00% patients expired while 85.00% were survivors.

Table 2: Comparison of baseline laboratory parameters and scores between survivors and thepatients who died within 3 months

| | Total patients | Survivors | Patients who died | p -value |
|------------------------|----------------|-------------|-------------------|----------|
| Serum Albumin (g/dL) | 2.30±0.65 | 2.41±0.62 | 1.75±0.52 | 0.001 |
| Serum Sodium (mmol) | 131.58±4.68 | 133.05±4.19 | 130.028±6.28 | 0.002 |
| Serum Potassium (mmol) | 4.18±1.12 | 4.18±1.15 | 4.29±1.08 | 0.511 |

Unpaired 't' Test

Serum albumins were lower among patients who died than survivors. Serum sodium was lower amongpatients who died than survivors. Serum potassium was higher among patients who died than survivors.

Discussion

As the liver cirrhosis progresses, there is fall in serum sodium concentration. It is a negative prognostic factor associated with increased short-term mortality. ⁷During cirrhosis, hyponatremia results from solute free water retention. Volume expansion with resuscitative fluid infusion is often required for hospitalized cirrhotic patients. Evidence supports humanserum albumin infusion in the treatment of certain specific complications of cirrhosis including HRS and prevention of both acute kidney injury in SBP and post-paracentesis circulatory dysfunction following large volume paracentesis. However, given the significantlyhigher cost associated with albumin compared tocrystalloid, limiting administration to appropriateclinical indications is essential. [4]

Albumin infusion is postulated to confer many benefits to cirrhotic patients beyond volume expansion, including immunomodulation, antioxidant effects, endothelial stabilization, andhemostatic effects, any and all of which might contribute to increased survival. [5]

Conclusion

We concluded that hyponatermia and hypoalbuminemia was a poor prognostic markerin chronic liver disease patients

References

- 1. D'Amico G, Garcia-Tsao G, Pagliaro
- 2. L. Natural history and prognostic indicators of survival in cirrhosis: a systematic review of 118 studies. *J Hepatol.* 2006; 44:217–231.
- 3. Kim WR, Biggins SW, Kremers WK, Wiesner RH, Kamath PS, Benson JT, Edwards E, Therneau TM. Hyponatremia and mortality among patients on the liver- transplant waiting list. N Engl J Med. 2008; 359: 1018-26.
- 4. Sola E, Watson H, Graupera I, Turon F, Barreto R, Rodriguez E, Pavesi M, Arroyo V, Guevara M, Gines P. Factors related to quality of life in patients with cirrhosis and ascites: relevance of serum sodium concentration and leg edema. J Hepatol. 2012;57: 1199-206.
- 5. John S, Thuluvath PJ. Hyponatremia in cirrhosis: pathophysiology and management. World J Gastroenterol. 2015; 21: 3197-205.
- Garcia-Martinez R, Caraceni P, Bernardi M, Gines P, Arroyo V, Jalan R. Albumin: pathophysiologic basis of its role in the treatment of cirrhosis and its complications. Hepatology. 2013; 58:1836-46.