ORIGINAL RESEARCH

Clinical presentation of patients with AKI and liver cirrhosis

Dr. Mansi Bansal

Assistant Professor, Department of General Medicine, Noida International Institute of Medical Sciences, India

Corresponding Author
Dr. Mansi Bansal
Assistant Professor, Department of General Medicine, Noida International Institute of Medical Sciences, India

Received: 19 May, 2022 Accepted: 25 June, 2022

ABSTRACT

Background: Hospitalized patients with cirrhosis of the liver frequently experience acute kidney damage (AKI), which is highly significant from a clinical and prognosis standpoint and a risk factor for early in-hospital mortality. The present study was conducted to assess clinical features of patients with AKI and liver cirrhosis. **Materials & Methods:**82 patients with cirrhosis of the liver and AKI of both genderswere divided into three groups- group I was pre-renal azotemia, group II was hepatorenal syndrome, and group III was acute tubular necrosis. For all patients, Child- Pugh score scores, etiology and creatinine level etc. was recorded. **Results:** Group I had 14 males and 10 females, group II had 27 males and 13 females and group III had 11 males and 9 females. The etiology for cirrhosis was alcohol in 11, 23 and 8, Hepatitis B in 6, 10 and 5, Hepatitis C in 4, 3 and 4, and cryptogenic in 3, 4 and 3 patients. AKI stage1 was seen in 12, 24 and 11, stage 2 in 8, 10 and 6 and stage 3 in 4, 6 and 3 patients in group I, II and III respectively. The difference was significant ($P< 0.05$). The mean Child- Pugh score was 10.3, 10.5 and 11.6. The Max creatinine level was 3.7 mg/dL, 4.6mg/dL and 4.2 mg/dL. eGFR at admission (mL/min/1.73 m2) was 38.2,26.2 and 35.2. Hospital stay was 12.3 days, 10.6 days and 11.1 days in group I, II and III respectively. The difference was non- significant ($P>0.05$). **Conclusion:** Acute renal damage in patients with liver cirrhosis is linked to a high death rate while hospitalized. Child-Pugh scores may be considered as indicators of AKI.

Key words: Acute renal damage, cirrhosis, creatinine

INTRODUCTION

Hospitalized patients with cirrhosis of the liver frequently experience acute kidney damage (AKI), which is highly significant from a clinical and prognosis standpoint and a risk factor for early in-hospital mortality. Individuals who do not have cirrhosis are less likely than those who do to get AKI.[1] The estimated prevalence of AKI in hospitalized cirrhosis patients ranges from 20% to 50%. Pre-renal injury (PRI), acute tubular necrosis (ATN), and hepatorenal syndrome (HRS-AKI) are the main causes of AKI in cirrhotic patients.[2] Accurate classification is essential because treatment varies greatly. Pre-renal AKI typically reacts favorably to an increase in plasma volume, while HRSAKI and ATN need distinct strategies and are linked to increased in-hospital mortality.[3]

The AKIN criteria recommend utilizing 48 hours, whereas the RIFLE criteria indicate changes within 7 days. Glomerular filtration rate is not used as a marker in AKI by the AKIN criteria.[4]Acute renal impairment in cirrhosis was represented by an AKI that was modified to measure a rise in SCr of 0.3 mg/dL (26.4 μmol/L) in less than 48 hours or a 50% increase from a baseline in less than three months. Similar to the acute kidney injury network (AKIN) criteria for identifying AKI in noncirrhotic patients, the severity of AKI is characterized by three stages.[5]The present study was conducted to assess clinical features of patients with AKI and liver cirrhosis.

MATERIALS & METHODS

The present study comprised of 82 patients with cirrhosis of the liver and AKI of both genders. All gave their written consent to participate in the study.

Data such as name, age, gender, etc. was recorded. Parameters such as comorbid illnesses, AKI stage, and risk factors for cirrhosis was recorded. Patients were divided into three groups- group I was pre-renal azotemia, group II was hepatorenal syndrome, and group III was acute tubular necrosis. For all patients, Child- Pugh score scores were recorded. In all cases, outcome was evaluated. Results were statisticallyanalyzed. P value less than 0.05 was considered significant.

RESULTS

Table I Baseline parameters

Parameters	Variables	Group I (24)	Group II (40)	Group III (20)	P value
Gender	Male	14	27	11	0.05
	Female	10	13	9	
Cirrhosis etiology	Alcohol	11	23	8	0.05
	Hepatitis B	6	10	5	
	Hepatitis C	4	3	4	
	Cryptogenic	3	4	3	
AKI stage	Stage 1	12	24	11	0.04
	Stage 2	8	10	6	
	Stage 3	4	6	3	

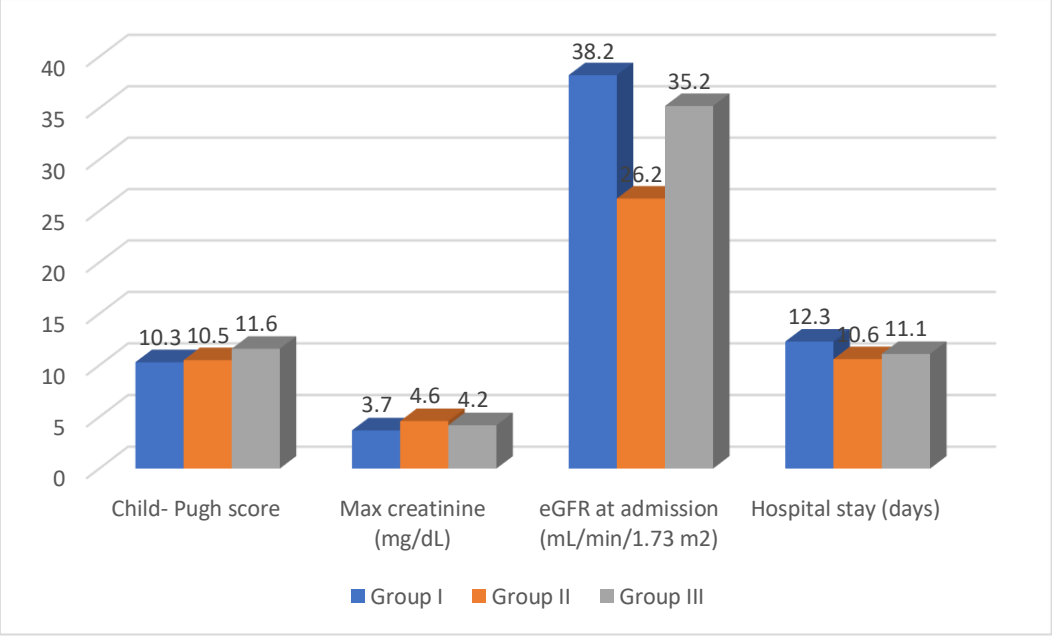
Table I shows that group I had 14 males and 10 females, group II had 27 males and 13 females and group III had 11 males and 9 females. The etiology for cirrhosis was alcohol in 11, 23 and 8, Hepatitis B in 6, 10 and 5, Hepatitis C in 4, 3 and 4, and cryptogenicin 3, 4 and 3 patients. AKI stage1 was seen in 12, 24and 11, stage 2 in 8, 10and 6 and stage 3 in 4, 6 and 3 patients in group I, II and III respectively. The difference was significant (P< 0.05).

Table II Comparison of parameters

Parameters	Group I	Group II	Group III	P value
Child- Pugh score	10.3	10.5	11.6	0.32
Max creatinine (mg/dL)	3.7	4.6	4.2	0.09
eGFR at admission (mL/min/1.73 m2)	38.2	26.2	35.2	0.53
Hospital stay (days)	12.3	10.6	11.1	0.94

Table II, graph I shows that mean Child- Pugh score was 10.3, 10.5 and 11.6. The Max creatinine level was 3.7mg/dL, 4.6mg/dL and 4.2mg/dL. eGFR at admission (mL/min/1.73 m2) was 38.2, 26.2 and 35.2. Hospital stay was 12.3 days, 10.6 days and 11.1 days in group I, II and III respectively. The difference was non-significant (P>0.05).

Graph I Comparison of parameters



DISCUSSION

Acute kidney injury (AKI) is defined by the impairment of kidney filtration and excretory function over days to weeks, resulting in the retention of nitrogenous and other waste products normally cleared by the kidneys.[6] The causes of AKI have traditionally been divided into three broad categories: prerenal azotemia, intrinsic renal parenchymal disease, and post renal obstruction.[7]The Acute Kidney Injury Network (AKIN) guidelines defines AKI as oliguria of less than 0.5 ml/kg/h for more than 6 hours or an abrupt (within 48 hours) increase in serum creatinine of 0.3 mg/dl or increase in serum creatinine by 1.5 times.[8] The definition of the Risk Injury Failure Loss and End Stage Renal Disease (RIFLE) criteria is based on a decrease in urine output and an increase in

serum creatinine/GFR. There are various ways in which the RIFLE and AKIN requirements are not the same.[9,10]The present study was conducted to assess clinical profile of patients with AKI and liver cirrhosis.

We found that group I had 14 males and 10 females, group II had 27 males and 13 females and group III had 11 males and 9 females. The etiology for cirrhosis was alcohol in 11, 23 and 8, Hepatitis B in 6, 10 and 5, Hepatitis C in 4, 3 and 4, and cryptogenic in 3, 4 and 3 patients. AKI stage1 was seen in 12, 24 and 11, stage 2 in 8, 10 and 6 and stage 3 in 4, 6 and 3 patients in group I, II and III respectively. Sharma et al[11]evaluated epidemiological data on the etiological profile of cirrhosis of the liver in adults in a tertiary care hospital. In total, 178 patients who were diagnosed with cirrhosis on the basis of history, physical examination, biochemistry and radiology, and of age >18 years were included in the study. Alcohol was the leading cause of cirrhosis in our study (62.9%), hepatitis B was the second(10.1%), Non-Alcoholic Steatohepatitis (NASH) was the third (7.9%), and autoimmune the fourth (3.9%) most common cause for cirrhosis. Hepatitis C was present in 2.8% of patients as a cause of cirrhosis. Wilson disease and cardiac cirrhosis were present in one patient each. In 9.6% the etiology was cryptogenic.

We observed that the mean Child- Pugh score was 10.3, 10.5 and 11.6. The Max creatinine level was 3.7 mg/dL, 4.6mg/dL and 4.2 mg/dL. eGFR at admission (mL/min/1.73 m2) was 38.2,26.2 and 35.2. Hospital stay was 12.3 days, 10.6 days and 11.1 days in group I, II and III respectively. Prakash et al[12]studied the clinical spectrum of renal disorders in patients with cirrhosis of liver and their prognostic impact.Four hundred and four patients were included in this study and renal diseases were present in 44% (n = 178) patients. The spectrum of renal diseases were acute kidney injury (AKI; 24.5%), chronic kidney disease (CKD; 15.6%), acute on chronic renal failure (1.5%), nephritic syndrome (1.5%), and nephrotic syndrome (1%). The types of AKI were acute tubular necrosis (ATN; 44.4%), prerenal failure (36.4%), and hepatorenal syndrome (19.2%). The incidence of renal diseases was 15.7% in class A, 50% in class B, and 54.8% in class C cirrhosis. There was significant increase in mortality in patients with class C cirrhosis versus without renal disease (78.1% vs. 53.2%; $p < 0.001$).

CONCLUSION

Authors found thatacute renal damage in patients with liver cirrhosis is linked to a high death rate while hospitalized. Child-Pugh scores may be considered as indicators of AKI.

REFERENCES

1. Mokdad AA, Lopez AD, Shahraz S, Lozano R, Mokdad AH, Stanaway J, et al. Liver cirrhosis mortality in 187 countries between 1980 and 2010: a systematic analysis. BMC Med. 2014;12:145.
2. Parke CY, Martin P, Bunnapradist S. Renal dysfunction in cirrhosis: Renal Dysfunction in Cirrhosis. Clin Liver Dis. 2015;5:150–3.
3. Angeli P, Gatta A, Caregaro L, et al. Tubular site of renal sodium retention in ascitic liver cirrhosis evaluated by lithium clearance. Eur J Clin Invest. 1990;20:111–7.
4. Kumar T. Incidence of cirrhosis caused by hepatitis B virus in different sex and age groups in Bihar. J MGIMS. 2006;11:52–4.
5. Shetty S, Nagaraju SP, Shenoy S, Attur RP, Rangaswamy D, Rao IR, Mateti UV, Parthasarathy R. Acute kidney injury in patients with cirrhosis of liver: Clinical profile and predictors of outcome. Indian journal of Gastroenterology. 2018 May;37(3):248-54.
6. Götzberger M, Kaiser C, Landauer N, Dieterle C, Heldwein W, Schiemann U. Intrarenal resistance index for the assessment of early renal function impairment in patients with liver cirrhosis. Eur J Med Res. 2008 Aug 18;13(8):383-7.
7. Francoz C, Prié D, AbdelRazek W, Moreau R, Mandot A, Belghiti J, Valla D, Durand F. Inaccuracies of creatinine and creatinine-based equations in candidates for liver transplantation with low creatinine: impact on the model for end-stage liver disease score. Liver transplantation. 2010 Oct;16(10):1169-77.
8. Bal C, Longkumer T, Patel C, Gupta SD, Acharya SK. Renal function and structure in subacute hepatic failure. Journal of gastroenterology and hepatology. 2000 Nov;15(11):1318-24.
9. Keshav R, Kumar B. Spectrum of renal dysfunctionin cirrhotic patients of Jharkhand. Indian. J Appl Res.2018; 8:48-50.
10. Sarin SK, Guptan RC, Banerjee K, Khandekar P. Low prevalence of hepatitis C viral infection in patients with non-alcoholic chronic liver disease in India. J Assoc Physicians India. 1996;44:243–5.
11. Sharma B, Marwah R, Raina S, Sharma N, Kaushik M, Kaushal SS. A study on the etiology of cirrhosis of liver in adults living in the Hills of Himachal Pradesh, India. Tropical Gastroenterology. 2017 Apr 13;37(1):37-41.
12. Prakash J, Mahapatra AK, Ghosh B, Arora P, Jain AK. Clinical Spectrum of Renal Disorders in Patients with Cirrhosis of Liver. Ren Fail. 2011;33:40–6.