International Journal of Research in Health and Allied Sciences

Journal home page: www.ijrhas.com

Official Publication of "Society for Scientific Research and Studies" [Regd.]

ISSN: 2455-7803

Original Research

Clinical profile of patients with AKI and liver cirrhosis attending tertiary care hospital

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ABSTRACT:

Background: Patients with cirrhosis are more susceptible to develop AKI than the noncirrhotic individuals. The present study was conducted to assess clinical profile of patients with AKI and liver cirrhosis. **Materials & Methods:** 104 patients who were diagnosed with cirrhosis of the liver with AKI of both genders were made part of the study. Baseline characteristics such as age, comorbid illnesses, AKI stage, and risk factors for cirrhosis was recorded and patients were classified into three groups: pre-renal azotemia, hepatorenal syndrome, and acute tubular necrosis. **Results:** There were 26 PRA, 64 ATN and 14 HRS patients. Male: Female ratio was 16:12, 34:30 and 6:8. Etiology for cirrhosis was alcohol in 15, 40 and 6, Hepatitis B in 6, 10 and 3, Hepatitis C in 4, 8 and 3, cryptogenic in 1, 6 and 2. AKI stage 1 was seen in 10, 14 and 3, stage 2 in 8, 20 and 6 and stage 3 in 8, 30 and 5 in PRA, ATN and HRS patients respectively. The mean MELD score was 28.1, 21.7 and 32.9, Child- Pugh score was 10.8, 10.9 and 11.5, creatinine at admission was 1.8 mg/dL, 3.2 mg/dL and 2.3 mg/dL. Max creatinine level was 3.6 mg/dL, 4.6 mg/dL and 4.1 mg/dL. eGFR at admission (mL/min/1.73 m2) was 39.2, 27.2 and 36.5. Hospital stay was 11 days, 10.4 days and 11 days in PRA, ATN and HRS patients respectively. The difference was non- significant (P>0.05). **Conclusion:** Acute kidney injuries in liver cirrhosis are associated with high in-hospital mortality.

Key words: Acute kidney injuries, Creatinine, Liver cirrhosis

Received: 22 June, 2021 Accepted: 27 July, 2021

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This article may be cited as: Jindal S, Mehra A, Modi D. Clinical profile of patients with AKI and liver cirrhosis attending tertiary care hospital. Int J Res Health Allied Sci 2021; 7(4): 172-175.

INTRODUCTION

Acute kidney injury (AKI) is common in hospitalized patients with cirrhosis of the liver and is an important risk factor for early in-hospital mortality and of utmost clinical and prognostic relevance. Patients with cirrhosis are more susceptible to develop AKI than the noncirrhotic individuals.[1] AKI has an estimated prevalence of approximately 20% to 50% among hospitalized patients with cirrhosis. Major causes of AKI in cirrhotic patients include pre-renal injury (PRI), acute tubular necrosis (ATN), and hepatorenal syndrome (HRS-AKI). Correct differentiation is imperative, as treatment differs substantially. Pre-renal AKI usually responds well to plasma volume expansion, whereas HRSAKI and ATN require different specific approaches and are associated with high in-hospital mortality.[2]

According to the Acute Kidney Injury Network (AKIN) criteria, AKI is defined as abrupt (within 48 hours) increase in serum creatinine of 0.3mg/dl or increase in serum creatinine by 1.5 times or oliguria of < 0.5 ml/kg/h for > 6 hours.[3] Risk Injury Failure Loss and End stage renal disease (RIFLE) criteria are defined based on increase in serum creatinine / GFR and decrease in urine output. The AKIN criteria differ from the RIFLE criteria in several ways. The RIFLE criteria are defined as changes within 7 days while AKIN criteria suggest using 48 hours. AKIN criteria avoid using glomerular filtration rate as a marker in AKI.[4]

It adapted the AKI to represent acute renal dysfunction in cirrhosis and defined it by an increase in SCr of 0.3 mg/dL (26.4 μmol/L) in < 48 h, or a 50% increase in SCr from a baseline within ≤ 3 months. The severity of AKI is described by three stages similar to acute kidney injury network (AKIN) criteria for defining AKI in noncirrhotic patients.[5] The present study was conducted to assess clinical profile of patients with AKI and liver cirrhosis.

MATERIALS & METHODS

The present study comprised of 104 patients who were diagnosed with cirrhosis of the liver with AKI of both genders. All were made part of the study with the written consent.

Demographic profile of each subject was recorded. Baseline characteristics such as age, comorbid illnesses, AKI stage, and risk factors for cirrhosis was recorded and patients were classified into three groups: pre-renal azotemia, hepatorenal syndrome, and acute tubular necrosis. For all patients, Child's and model for end stage liver disease (MELDs) scores were analyzed. The treatment was given and outcome was evaluated. Results were studied statistically.

RESULTS

Table I Baseline characteristics

Characteristics	PRA	ATN	HRS	P value
Number	26	64	14	0.01
M:F	16:12	34:30	6:8	0.05
Cirrhosis etiology				
Alcohol	15	40	6	0.03
Hepatitis B	6	10	3	
Hepatitis C	4	8	3	
Cryptogenic	1	6	2	
AKI stage				
Stage 1	10	14	3	0.05
Stage 2	8	20	6	
Stage 3	8	30	5	

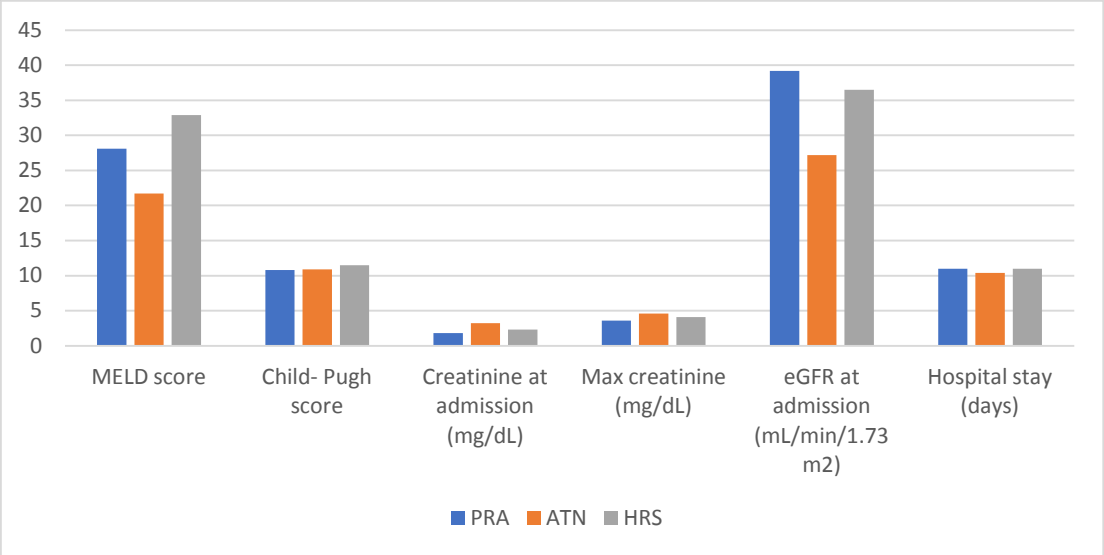
Table I shows that there were 26 PRA, 64 ATN and 14 HRS patients. Male: Female ratio was 16:12, 34:30 and 6:8. Etiology for cirrhosis was alcohol in 15, 40 and 6, Hepatitis B in 6, 10 and 3, Hepatitis C in 4 , 8 and 3, cryptogenic in 1, 6 and 2. AKI stage 1 was seen in 10, 14 and 3, stage 2 in 8, 20 and 6 and stage 3 in 8, 30 and 5 in PRA, ATN and HRS patients respectively. The difference was significant (P< 0.05).

Table II Comparison of parameters

Characteristics	PRA	ATN	HRS	P value
MELD score	28.1	21.7	32.9	0.12
Child- Pugh score	10.8	10.9	11.5	0.16
Creatinine at admission (mg/dL)	1.8	3.2	2.3	0.08
Max creatinine (mg/dL)	3.6	4.6	4.1	0.07
eGFR at admission (mL/min/1.73 m2)	39.2	27.2	36.5	0.35
Hospital stay (days)	11	10.4	11	0.91

Table II, graph I shows that mean MELD score was 28.1, 21.7 and 32.9, Child- Pugh score was 10.8, 10.9 and 11.5, creatinine at admission was 1.8 mg/dL, 3.2 mg/dL and 2.3 mg/dL. Max creatinine level was 3.6 mg/dL, 4.6 mg/dL and 4.1 mg/dL. eGFR at admission (mL/min/1.73 m2) was 39.2, 27.2 and 36.5. Hospital stay was 11 days, 10.4 days and 11 days in PRA, ATN and HRS patients 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] In pre renal causes of AKI underlying kidney function may be normal, but decreased renal perfusion associated with intravascular volume depletion (e.g., from vomiting or diarrhea) or decreased arterial pressure (e.g., from heart failure or sepsis) results in a reduced glomerular filtration rate. Autoregulatory mechanisms often can compensate for some degree of reduced renal perfusion in an attempt to maintain the glomerular filtration rate.[8] In patients with pre-existing chronic kidney disease, however, these mechanisms are impaired, and the susceptibility to develop acute-or-chronic renal failure is higher.[9] The present study was conducted to assess clinical profile of patients with AKI and liver cirrhosis.

In present study, there were 26 PRA, 64 ATN and 14 HRS patients. Male: Female ratio was 16:12, 34:30 and 6:8. Etiology for cirrhosis was alcohol in 15, 40 and 6, Hepatitis B in 6, 10 and 3, Hepatitis C in 4, 8 and 3, cryptogenic in 1, 6 and 2. AKI stage 1 was seen in 10, 14and 3, stage 2 in 8, 20 and 6 and stage 3 in 8, 30 and 5 in PRA, ATN and HRS patients respectively. Shetty et al[10] studied the clinical profile and predictors of in-hospital mortality in patients with cirrhosis of the liver with AKI. AKI staging was done based on the new 2015 Ascites Club Criteria. Patients were grouped into three types of AKI: pre-renal azotemia (PRA), hepatorenal syndrome (HRS), and acute tubular necrosis (ATN). Results Data of 123 patients with cirrhosis and AKI were analyzed. Most patients had AKI stage 3 (57.7%). ATN (42.3%) and HRS (43.9) were the predominant types of AKI followed by PRA (13.8%). The overall in-hospital mortality in our study was 44.7%. The mortality increased with increasing severity of AKI (p = 0.0001) and was the highest in AKI stage 3 (p = 0.001) and those who required hemodialysis (p = 0.001). There was a significant in-hospital mortality in patients with ATN and HRS in comparison to PRA (p = 0.001). On multivariate analysis, the factors predicting in-hospital mortality were AKI stage 3, and oliguria (p = 0.0001).

We found that mean MELD score was 28.1, 21.7 and 32.9, Child- Pugh score was 10.8, 10.9 and 11.5, creatinine at admission was 1.8 mg/dL, 3.2 mg/dL and 2.3 mg/dL. Max creatinine level was 3.6 mg/dL, 4.6 mg/dL and 4.1 mg/dL. eGFR at admission (mL/min/1.73 m2) was 39.2, 27.2 and 36.5. Hospital stay was 11 days, 10.4 days and 11 days in PRA, ATN and HRS patients respectively. Aroro et al[11] studied the clinical profile and predictors of acute kidney injury (AKI) in patients with decompensated cirrhosis on 175 consecutive patients with decompensated cirrhosis. The prevalence of AKI was 40.6%, with prerenal AKI 67.6%, hepatorenal syndrome (HRS) 23.8%, intrinsic renal AKI 7%, and postrenal AKI 1.4%. Mean arterial pressure (MAP), platelet count, and serum albumin were significantly lower and total leucocyte count (TLC), blood urea nitrogen, serum creatinine (SCr), total bilirubin, aspartate aminotransferase, international normalized ratio, Child-Turcotte-Pugh (CTP) score, and model for end-stage liver disease (MELD) score higher in cirrhosis patients with AKI than without AKI ($p < 0.05$ each). MAP, hemoglobin, TLC, and SCr were significantly different in various types of AKI ($p < 0.05$ each). AKI had a significant association with CTP score, alcohol, spontaneous bacterial peritonitis (SBP), sepsis, and shock. Type of AKI had significant association with SBP, sepsis, and shock. Mortality occurred in 33.8% patients with AKI with 64.7% mortality in patients with HRS. Outcome had

significant association with AKI, stage and type of AKI. Multivariate analysis showed SBP, sepsis, and shock as independent predictors of AKI.

CONCLUSION

Authors found that acute kidney injuries in liver cirrhosis are associated with high in-hospital mortality.

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