



## Clinical Spectrum of Renal Dysfunction in Cirrhosis of Liver

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### Abstract

**Introduction:** Renal dysfunction is a common and serious problem in patients with advanced liver disease. Physicians involved in the care of patients of cirrhosis recognize that the development of renal dysfunction is associated with significant morbidity and mortality. Hence it is very important to diagnose condition early and recognize type of renal failure in order to decide further therapy.

**Methods-**This was a prospective single centered observational study was conducted on 100 patients with cirrhosis of liver admitted in a tertiary care hospital, according to inclusion and exclusion criteria during the study period Jan 2015 to Jul 2016. After collecting details of demographic data, subjects underwent complete general & systemic examination and later subjected to blood investigations, ultrasonography abdomen and kub, urine examination and upper gastrointestinal endoscopy. Child-pugh score and MELD score was calculated.

**Result-** In this study, Renal dysfunction was present in 40% of population which was later categorized according to the type of renal involvement. 21% of total study population was identified to develop acute kidney injury, which was most common presentation for renal involvement and 18% were found to have hepatorenal syndrome. Only one (1%) patient was diagnosed as chronic kidney disease. The most common etiology found for liver cirrhosis was alcoholic cirrhosis in both groups with or without renal dysfunction. In our study population total 16 patients were succumbed during hospitalization and all are belong to hepatorenal syndrome. Among the patients with fatal outcome maximum patients were male and had complications of spontaneous bacterial peritonitis, esophageal varices and were in CP class C with higher MELD score. Presence of spontaneous bacterial peritonitis and gastrointestinal bleed were major factors those were significantly associated with development of HRS. These factors alone or in combination were responsible for development of hepatorenal syndrome.

**Conclusion-**Renal dysfunction is a frequent and dreaded complication of cirrhosis of liver. An attempt should always be made to identify it early and categorize patients in different groups as treatment and prognosis differs in these subgroups. Patients usually have downhill course once hepato-renal syndrome develops while patients with AKI because of pre-renal azotemia can be successfully treated if detected early.

## Introduction

Renal dysfunction is a common and serious problem in patients with advanced liver disease. Physicians involved in the care of patients of cirrhosis recognize that the development of renal dysfunction is associated with significant morbidity and mortality. Methods for early and accurate diagnosis of renal failure may assist to initiate specific treatment at an earlier stage and improve the outcome. Patients with cirrhosis can commonly develop acute renal failure and may rarely suffer from underlying chronic kidney disease. Common types of renal dysfunction seen in cirrhosis of liver are hepatorenal syndrome; hypovolemia induced renal failure, renal failure because of intrinsic renal disease and drug-induced renal failure. To pinpoint the specific type of renal failure in cirrhosis though difficult is important for both prognostic and therapeutic point of view. Once renal dysfunction sets in prognosis of patient worsens leading to progressive downhill course, leading to significant morbidity and mortality. Hence it is very important to diagnose condition early and recognize type of renal failure in order to decide further therapy.<sup>1,2,3.</sup>

Hepatorenal syndrome (HRS) is a unique form of acute kidney injury seen in patient with cirrhosis in the absence of any other identifiable cause of renal failure. It usually results from renal vasoconstriction in the setting of systemic and splanchnic arterial vasodilatation in patients with advanced cirrhosis. HRS is typically subdivided into two types: type-1 in which there is a rapid deterioration of kidney function with the serum creatinine increasing by more than 100% from baseline to greater than 2.5 mg/dl within a two-week period, whereas type-2 HRS occurs in patients with refractory ascites with either a steady but moderate degree of functional renal failure ( $\geq 1.5$  mg/dl) or a deterioration in kidney function that does not fulfill the criteria for HRS type-1. In patients with advanced cirrhosis, HRS is reported to occur in 18% within one year of diagnosis and up to 40% at five years. Untreated, median survival is two weeks for patients with type-1

HRS and four to six months in patients with type-2 hepatorenal syndrome.<sup>4</sup>

Other types of renal dysfunction include 1. hypovolemia induced renal failure which occurs in setting of excessive fluid loss as occurs in vigorous diuretic use, Gastro-intestinal bleeding, vomiting or diarrhoea. It can be treated effectively by restoring lost volume. 2. Intrinsic renal disease can also occur in cirrhosis as in hepatitis C. It can be detected by proteinuria  $> 500$ mg/ day, or hematuria micro or macroscopic and by altered echo structure in USG. Distinction between acute tubular necrosis and the hepatorenal syndrome 1 remains a difficult issue; the presence of renal tubular epithelial cells in the urine sediment favors the diagnosis of acute tubular necrosis. Current or recent treatment with nonsteroidal anti-inflammatory drugs or aminoglycosides suggests drug-induced renal failure.<sup>5,6.</sup>

The term 'Hepatorenal Disorders' has been proposed to group all forms of kidney disease in patients with cirrhosis so as to describe their prognosis and to assist treatment decisions.<sup>7</sup>, in the majority of patients with advanced liver disease, HRS type-1 remains a terminal condition of requiring coordinated efforts for diagnosis and treatment.

Considering importance of renal dysfunction in natural history and prognostication, this project was undertaken to diagnose spectrum of renal dysfunction in cirrhosis and study risk factors for this dreaded complication. In our study, we are going to address the current knowledge on the diagnosis evaluation and risk assessment of renal dysfunction in patients with cirrhosis.

## Aims and Objectives

1. To study prevalence of renal dysfunction in cirrhosis of liver.
2. To categorise patients with renal dysfunction into different types.
3. To study risk factors for development of hepatorenal syndrome.
4. To correlate severity of cirrhosis with occurrence of hepatorenal syndrome.

## Materials and Methods

### Study Design

This was a prospective single centered observational study was conducted on patients with cirrhosis of liver admitted in general medicine wards or intensive care unit, in a tertiary care hospital. Institutional Ethics Committee approval was obtained prior to commencement of the study. The data was collected after obtaining written informed consent from the patient.

Sample -100 consecutive of cirrhotic patients admitted during the study period 19 months (Jan 2015 to Jul 2016) as per the following inclusion/exclusion criteria were included in study.

### Inclusion criteria:

- Patients above age 18 years
- Patients who were admitted in general medicine wards or intensive care unit with cirrhosis of liver and ascites.
- Patients willing to give written informed consent

### Exclusion Criteria:

- Shock
- Severe sepsis
- Multi-organ failure
- Patient on maintenance hemodialysis

**Shock** is a clinical syndrome that result from inadequate tissue perfusion. Irrespective of cause, the hypoperfusion induced imbalance between the delivery of and requirements for oxygen and substrate leads to cellular dysfunction. Clinical shock is usually accompanied by hypotension (mean arterial pressure < 60 mmHg in previously normotensive persons)

**Severe sepsis** is the harmful host response to infection systemic response to proven/suspected infection plus some degree of organ hypofunction arterial systolic blood pressure  $\leq 90$  mm of hg or mean arterial pressure  $\leq 70$  mm of hg that responds to administration of IV fluids. urine output 0.5 ml/kg per hour for 1hr despite adequate fluid resuscitation. Pao<sub>2</sub>/Fio<sub>2</sub>  $\leq 250$  or, if the lung is the only dysfunctional organ  $\leq 200$ . platlet count < 80000/ $\mu$ l or 50% decrease in platlet count from

highest value recorded over previous 3 days. A ph < 7.3 or a base deficit 5.0mEq/l and a plasma lactate level 1.5 times upper limit of normal for reporting lab.

**Multiorgan failure** is defined by simultaneous presence of physiologic dysfunction and/or failure of two or more organs. Typically, this syndrome occurs in the setting of severe sepsis, shock of any kind, severe inflammatory conditions.

### Data Collection

Purpose of study was explained to the patient and written informed consent was obtained.

Data was obtained from the medical history and clinical examination and relevent investigation were in Case Record Form (CRF).

The following parameters were noted:

- 1) Patients Demographic Data: Name, Age, and Gender.
- 2) Detailed medical history for following variables was evaluated: Abdominal distention, pain in abdomen, fever, reduced urinary output, haematemesis, melaena, vomiting, diarrhoea, swelling of feet yellow discolouration of eyes, altered level of conscious, smoking, details of alcohol intake, hypertension, diabetes mellitus, chronic obstructive pulmonary disease, and ischemic heart disease
- 3) Clinical examination: Temperature, pulse, blood pressure, presence of pallor, icterus, clubbing, pedal odema, spider naevi, gynaecomastia, breast atrophy, loss of axillary hair, flapping tremor, palmar erythema, drupatryons contracture, testiucular atrophy, splenomegaly, ascites, abdominal tenderness, crepts, consicousness, encephalopathy.
- 4) Laboratory Investigations:
  - All patients were subjected to lab investigations in the form of Complete blood count, coagulation profile, Liver function test and Kidney function test.
  - Appropriate cultures were sent in case of suspected infection.

- Urinary sodium and urine osmolality were also estimated.
  - Abdominal paracentesis was done and the ascitic fluid was subjected to biochemical and pathological examination in every patient
5. Radiological Investigations: All patient underwent Ultrasonography examination for evaluation of presence of ascites, examination of liver, spleen and kidneys.

Cirrhosis was diagnosed by clinical, biochemical and ultra-sonographic examination. Based on the patient details and investigations we also tried to find out etiology of cirrhosis and cryptogenic cirrhosis (unknown etiology) labelled after ruling out other common cause of cirrhosis of liver (like wilson disease, hepatitis B and C, autoimmune, drug induced). Patients were assessed for presence of renal dysfunction. Patients showing signs and findings related to renal dysfunction in presence of cirrhosis were identified, and a detailed evaluation was performed. Based on the clinical findings, biochemical parameters and radiological investigations, these patients were subcategorized as below categorizes.

#### 1. Based on acuity of presentation

- Acute kidney injury
- Chronic kidney disease
- Acute on chronic kidney disease

#### 2. Based on the cause of renal dysfunction

- Prerenal azotemia
- Hepatorenal syndrome
- Intrinsic renal disease: Acute tubular necrosis (ATN), glomerulopathies, acute interstitial nephritis (AIN), contrast-induced nephropathy and others
- Post-renal disease.

As follow up of patient were not done, patients could not be categorized between HRS1 and HRS2.

Patients with serum creatinine more than 1.5 mg/dl were given albumin 1gm/kg of body weight per day for 2 days with diuretic withdrawal. After 2 days if serum creatinine improve to less than or equal to 1.5mg/dl they were labelled as acute

kidney injury (AKI) because of prerenal azotemia and if no improvement was seen in serum creatinine they were labelled as hepatorenal syndrome<sup>72</sup>

Hepatorenal syndrome was defined as per the International Ascites Club (IAC) definition.

Prerenal azotemia was defined using clinical and laboratory parameters. Urinary markers necessary for diagnosis included a urine specific gravity greater than 1.020 as well as a fractional excretion of sodium less than 0.01 (1%) in oliguric patients not treated with diuretics. Diagnosis of Intrinsic renal disease required the presence of granular casts (epithelial cell casts) in urinary sediment, fractional excretion of sodium greater than 2%, or a urinary sodium concentration greater than 40 mmol/L, in the clinical setting of prolonged hypotension, tachycardia, sepsis, or bleeding.

All the patients include in study were divided into two groups, those with renal dysfunction or without renal dysfunction. clinical parameter and laboratory parameter and frequency of complications, severity of the disease (by MELD and CPT score) and mortality were compared between two groups and statistically analyzed.

Score Calculation:

For assessment of severity liver dysfunction CTP score and MELD scores were calculated in all patients.

- 1) CTP:(Child-Turcotte-Pugh) Score- shown in Table no. 5 and 6, Fig. no. 3 and 4)
- 2) MELD: (Model for End-Stage Liver Disease) Score-shown in Table no.6a, Fig.no. 5 and 6)

#### Statistical analysis of Data

The statistical analysis of the data was performed using statistical package for social sciences (SPSS) software version 15.0. Demographics, clinical and laboratory characteristics of the patients were summarized using descriptive statistics like mean, standard deviation, for continuous variables and frequency and percentage for categorical variables. Categorical variables were compared using Chi-square test. Independent

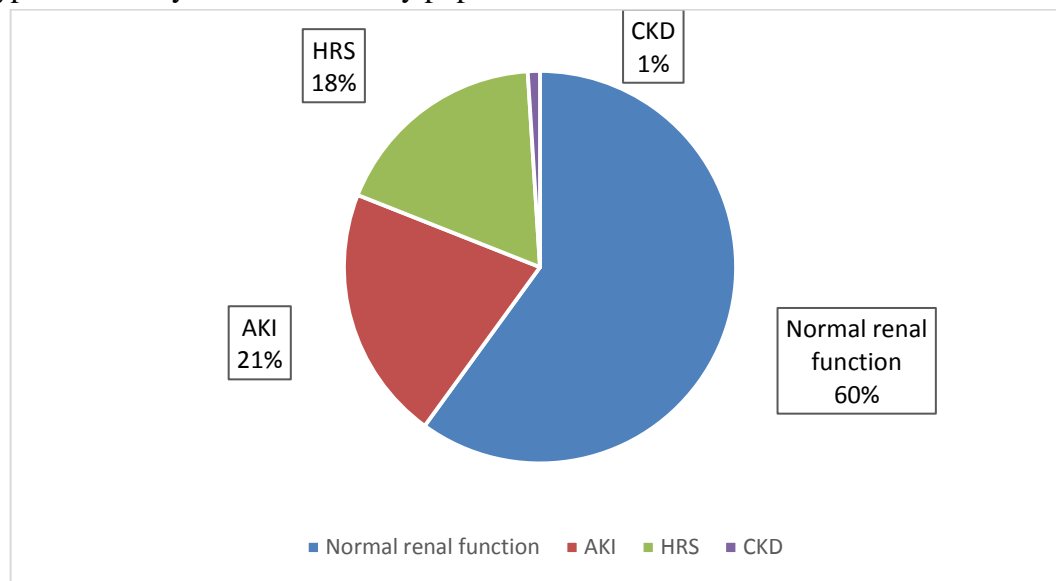
sample t test used to compare continuous variables in two groups. P value <0.05 was considered as significant.

## Results

**Table 10:** Frequency and type of renal dysfunction in study population

Renal Dysfunction	Frequency (%)
Present	40 (40.0)
Absent	60 (60.0)
Type of Renal Dysfunction	
Acute Kidney Injury	21 (21%)
Hepato-renal Syndrome	18 (18%)
Chronic kidney disease	1 (1%)

**Figure 8:** Type of renal dysfunction in study population



Renal dysfunction was present in 40% case in our study as shown in table 10/figure8. Amongst these patients, 21%, 18% and 1% had AKI, hepato-renal syndrome, and chronic kidney disease respectively.

**Table 24:** Child-Pugh classification of in both groups

Child-Pugh Class	Renal Dysfunction		P values
	Present (n=40)	Absent (n=60)	
B	14 (35%)	37 (61.7)	0.009
C	26 (65%)	23 (38.3)	

Significantly higher percentage of patients with renal dysfunction (65%) had more severe disease Child-Pugh class (C) compared to those without renal dysfunction (38.3%).

**Table 25:** Child-Pugh classification of in HRS cases

Child-Pugh Class	HRS-Present (n=18)	HRS-Absent (n=82)	P values
B	4 (22.2)	47 (57.3)	0.007
C	14 (77.8)	35 (42.7)	

From among patients with HRS, over 3/4th patients had hepatic disease of CP class C which was significantly higher than those without HRS (42.7%) (p=0.007) as shown in table 25.

**Table 26:** MELD score distribution in both groups

MELD Score	Renal Dysfunction		P values
	Present (n=40)	Absent (n=60)	
Mean	26.0±8.6	16.4±6.7	<0.0001
Categories according to the score			
40 or more	3 (7.5)	3 (5.0)	<0.0001
30–39	8 (20.0)	0	
20–29	19 (47.5)	3 (5.0)	
10–19	10 (25.0)	54 (90.0)	
<9	0	0	

MELD score distribution is depicted in table 26. Patients who had renal dysfunction were found to have significantly higher mean MELD score than those without renal dysfunction (p<0.0001). Also,

proportion of patients with higher MELD score was significantly greater in patients with renal dysfunction (p<0.0001)

**Table 27:** MELD score distribution in HRS population

MELD Score	HRS		P values
	Present (n=18)	Absent (n=82)	
Mean	33.1±7.7	17.4±6.2	<0.0001
Categories according to the score			
40 or more	3 (16.7)	3 (3.7)	<0.0001
30–39	8 (44.4)	0	
20–29	6 (33.3)	16 (19.5)	
10–19	1 (5.6)	63 (76.8)	
<9	0	0	

MELD score distribution is HRS cases depicted in table 27. Patients who had HRS were found to have significantly higher mean MELD score than those without HRS (p<0.0001). Also, proportion

of patients with higher MELD score was significantly greater in patients with HRS (p<0.0001).

**Table 29:** Distribution of important demographic, clinical and laboratory features in survivors and non-survivors

variables	Mortality		P values
	Yes (n=16)	No (n=84)	
Age	45.9±5.7	48.4±6.3	0.141
Male	13 (81.3)	81 (96.4)	0.019
Female	3 (18.7)	3 (3.6)	
History of Alcoholism	15 (93.8)	81 (96.4)	0.616
Hematemesis	9 (56.3)	15 (17.9)	0.001
Melaena	14 (87.5)	31 (36.9)	<0.0001
Spontaneous bacterial peritonitis	15 (93.8)	16 (19.0)	<0.0001
Esophageal varices	9 (56.3)	19 (22.6)	0.006
CP Grade – B	3 (18.8)	48 (57.1)	0.005
CP Grade - C	13 (81.3)	36 (42.9)	
MELD Score	34.7±6.6	17.5±6.1	<0.0001
Serum creatinine	2.9±0.56	1.5±0.71	<0.0001
Blood urea	72.7±20.2	53.1±14.3	<0.0001
Total bilirubin	16.6±9.3	4.4±7.0	<0.0001
SGOT	222.2±159.3	74.4±37.6	<0.0001
SGPT	139.6±116.4	43.5±15.9	<0.0001
Total protein	5.4±0.52	5.8±0.50	0.009
INR	2.2±0.6	1.3±0.3	<0.0001

Table 29 described the association of various population parameters with mortality outcome. Male gender ( $p=0.019$ ), hematemesis ( $p=0.001$ ), melaena ( $p<0.0001$ ), SBP ( $p<0.0001$ ), presence of oesophageal varices ( $p=0.006$ ), severe hepatic

disease CP class C ( $p=0.005$ ), higher MELD score, serum creatinine, blood urea, total bilirubin, SGPT/SGOT and INR ( $p<0.0001$  for all) and lower total protein levels ( $p=0.009$ ) were associated with mortality.

**Table 31:** Risk factor for development of hepato-renal syndrome

Risk factor	HRS		P value
	Present (n=18)	Absent (n=82)	
Sub-acute bacterial peritonitis (SBP)	16 (88.9)	15 (18.3)	<0.0001
GI bleed	9 (50.0)	19 (23.2)	0.022
Hyponatremia	13 (72.2)	55 (67.1)	0.672
Other - Infection (pneumonia)	1 (5.6)	1 (1.2)	0.444
SBP with GI bleed	8 (44.4)	6 (7.3)	-
GI bleed with hyponatremia	8 (44.4)	12 (14.6)	-
SBP with hyponatremia	11 (61.1)	8 (9.8)	-
SBP with GI bleed with hyponatremia	7 (38.9)	2 (2.4)	-

The risk factors associated with development of HRS are shown in table 31. Presence of SBP ( $p<0.0001$ ), GI bleed ( $p=0.022$ ) were major factors that were significantly associated with development of HRS. These factors alone or in combination were responsible for development of HRS as shown in table 32.

### Discussion

This prospective, single-center observational study was undertaken to determine the prevalence of renal dysfunction in patients with cirrhosis of liver to categorize these patients into different types, determine proportion of patients with cirrhosis developing hepatorenal syndrome and evaluate the risk factors involved in causation of Hepatorenal syndrome.

Data was collected from 100 indoor patients with cirrhosis of liver with concomitant ascites.

In present study, renal dysfunction was found to be present in 40% of the patient with cirrhosis of liver. patients were further categorized according to the type of renal involvement. It was seen that acute kidney injury was present in 21% of population which was the most common presenting renal involvement, followed by hepatorenal syndrome which was present in 18% of patients. only one patient(1%) was detected to have CKD in the entire population (Table10, Fig

8). The prevalence of 40% for renal dysfunction in our study was similar to another Indian study from North India<sup>8</sup> which showed that renal dysfunction were present in 44% patients with liver cirrhosis. Whereas, another Indian study from South India, showed that 22% patients with cirrhosis presented with renal dysfunction.<sup>9</sup> Another study by Howard et al, found that out of total 93 patients with a diagnosis of liver cirrhosis and normal renal function at admission, 23 patients developed renal dysfunction later within 15 days of hospitalization.<sup>10</sup>

MELD score distribution was calculated and assessed. Patients who had renal dysfunction were found to have significantly higher mean MELD score than those without renal dysfunction ( $p<0.0001$ ). Similar result was seen in those with HRS ( $p<0.0001$ ). Also, proportion of patients with higher MELD score was significantly greater in patients with renal dysfunction ( $p<0.0001$ ) and HRS ( $p<0.0001$ ). Maximum of the patients had the score of MELD between the range of 20-29. In our study, 16 patients had mortality as an outcome during hospitalization, out of which all were identified to be in the HRS and renal dysfunction group. No patients with acute kidney injury or chronic kidney disease showed a fatal outcome. In our study, 16 patients had mortality as an outcome during hospitalization, out of which

all were identified to be in the HRS and renal dysfunction group. No patients with acute kidney injury or chronic kidney disease showed a fatal outcome. Further we studied the distribution of important demographic, clinical and laboratory features in survivors and non-survivors. Our findings suggest that male population was more among the non-survivors. Among the patients with mortality outcome symptoms of hematemesis ( $p=0.001$ ), melaena ( $p<0.0001$ ) along with complications like sub-acute bacterial peritonitis ( $p<0.0001$ ), presence of oesophageal varices ( $p=0.006$ ), severe hepatic disease child Pugh class C ( $p=0.005$ ), higher MELD score, serum creatinine, blood urea, total bilirubin, SGPT/SGOT and INR ( $p<0.0001$  for all) and lower total protein levels ( $p=0.009$ ) were associated with mortality. Damien et al in his study, determined outcome and mortality risk related to acute renal failure (ARF) in critically ill patients with cirrhosis. It was observed that cirrhotic patients with ARF had higher MELD scores at baseline than those without ARF. They had more respiratory failure and cardiovascular failure during ICU stay, longer stay in ICU, and a greater crude hospital mortality rate (65% vs. 32%). In case-control studies both mild and severe ARF were independently associated with mortality (HR, 2.6, and 4.2, respectively). Cirrhotic patients with mild ARF patients had a higher risk of death than those without ARF (relative risk, RR, 2.0). Severe ARF was associated with an increase matched risk of death (RR 2.6), higher mortality of 51%, and higher risk-adjusted mortality rate (2.1 vs. 0.9).<sup>11</sup> Our study show similar findings on mortality suggesting a higher mortality in patients with renal dysfunction, depending on the severity of clinical presentation.

In our present study as well as in previous studies, patients with HRS generally had very high MELD scores of 20 or above.<sup>12</sup> Such high MELD scores are associated with 3-month mortality rates of 75% or more, irrespective of the presence of renal failure.<sup>13</sup> However, our data demonstrate that not

only severity of disease as assessed by the MELD score but also the presence of HRS has independent prognostic relevance for patients with cirrhosis and renal failure. This confirms the results of a recently published very important paper by Allesandria et al. which conceivably demonstrated that, in addition to the MELD score, the presence of HRS is an independent predictor of survival in cirrhotic patients with HRS. The probability of survival after 3 months was only 10% in that study, which was significantly less than in patients with the same MELD score, but without HRS. In our present study, the 3-month survival for HRS 1 patients was 33%. This slight discrepancy cannot be explained by different degrees of liver failure, as the MELD scores of HRS patients were almost similar (33.1 in our study vs 28 in the study described).

However nevertheless, serum creatinine which is used to analyze MELD score, should be interpreted with caution, since there is no universal standardized creatinine assay; there are interlaboratory variations, interactions with bilirubin and great influence by numerous non-renal factors such as body weight, race, age, and gender.<sup>14</sup>

Our study showed that esophageal varices (66.7%), use of diuretics (47.6%) and presence of diarrhoea and vomiting (23.8% each) were major cause of acute kidney injury alone or in combination. Whereas along with this the risk factors for development of hepatorenal syndrome was studied which showed that presence of sub-acute bacterial peritonitis (88 % vs 18.3%,  $p<0.0001$ ), GI bleed (50% vs 23.2%,  $p=0.022$ ) were major factors that were significantly associated with development of HRS. Hyponatremia was also a common risk factor in both the groups but the difference was not statistically significant (72.2% vs 67.1%,  $p=0.672$ ). Other risk factors encountered were infections.

Our findings go with those reported by others. Follo et al.<sup>15</sup> concluded that renal dysfunction is frequent in cirrhotic with spontaneous bacterial



peritonitis that occurs mainly in patients with kidney failure before infection and found that renal failure is an important predictor of survival especially in the presence of SBP. Also, the same conclusion was mentioned in the study of Ruiz-del Arbol et al.<sup>16</sup> as patients with SBP frequently develop a progressive severe renal and hepatic failure, and even death, despite of the resolution of infection and is usually associated with an extremely poor prognosis. These observations give an alarm for the emergency-pattern of combating coexisted infections in patients with ESLD to prevent these comorbidities and poor prognosis.

In our study we aimed to categorize the clinical presentation of renal dysfunction, the subtypes of renal involvement, hepatorenal syndrome along with assessment of risk factors and mortality. Martín-Llahí et al.<sup>(14)</sup> discussed this drawback by the absence of specific markers that may help in the identification of the cause of renal failure in cirrhosis. We also recommend the need to exert an effort to build-up a new battery of laboratory markers of renal failure that may be useful for the differential diagnosis. The main limitation in our study was the short duration of the study and the lack of incorporating new adjuvant markers that may help in better differentiation of the possible etiology of renal failure in cirrhotic patients. Therefore, we recommend to investigate new tools for early detection and good delineation of renal dysfunction among cirrhotic that will reflect on prognosis.

### Conclusion

Renal dysfunction is a frequent and dreaded complication of cirrhosis of liver. An attempt should always be made to identify it early and categorize patients in different groups as treatment and prognosis differs in these subgroups. Patients usually have downhill course once hepato-renal syndrome develops while patients with AKI because of pre-renal azotemia can be successfully treated if detected early. Patients with higher MELD score and Child C class patients are more

likely to develop renal dysfunction especially hepatorenal syndrome. Bleeding esophageal varices, SBP and hepatic encephalopathy are important risk factors for development of renal dysfunction.. Early diagnosis, prevention and appropriate treatment of renal dysfunction can reduce the morbidity and mortality.

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