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Study of Clinical Profile and Outcome in Patients of Alcohol Induced Chronic Liver Disease with Hepato Renal Syndrome

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Abstract

Background: Decompensated Chronic liver disease and cirrhosis are frequently complicated with renal dysfunction and this combination, called Hepato-Renal Syndrome (HRS) leads to significant morbidity and mortality. Acute renal dysfunction occurs in 15% to 25% of hospitalized patients with cirrhosis. The annual frequency of Hepato-renal syndrome(HRS) in cirrhotic patients with ascites is roughly 8% and, in some reports, as high as 40%.The observation that morbidity and mortality remain high once the syndrome is established has led to a focus on the prevention and early therapy of renal dysfunction in patients with cirrhosis. Hepato-renal syndrome (HRS) is a serious complication of liver cirrhosis with critically poor prognosis. Rapid diagnosis and management are important, since recent treatment modalities including vasoconstrictor therapy can improve short-term outcome and buy time for liver transplantation, which can result in complete recovery. Recognizing the trait about chronic alcoholism in a patient, counselling by doctor about avoidance of alcohol, & early recognition of progression to CLD by investigations may be helpful in preventing this complication. **Aims and Objectives:** To study clinical profile, assess the course and severity and outcome of patients admitted with alcohol induced chronic liver disease with hepato-renal syndrome. **Methodology:** A prospective (observational), hospital based study was carried out in 57 patients who met our inclusion criteria. **Results:** The study population's most common age group was between 36 and 45 years (33.3%), 46 to 55 years (24.6%), and more than 55 years (22.8%) with the mean age of 43 + 7.8 years amongst study population with a male predominanace (87.7%), Icterus and ascitis (100%) followed by Flapping tremors (98.25%), Oliguria (82.5%), Edema Feet (73.68%), Altered sensorium (66.7%) and Abdominal Distension (59.65%) were the commonest clinical features. History of alcohol intake for more than 10 years (61.4%) followed by less than 10 years (38.6%) and most of them were heavy drinkers (>4 drinks per day) (44.2%) while moderate and light drinkers (2-4 drinks/day and 1-2 drinks/day) were 32.7% and 23.1% respectively. Previous history of admissions due to similar or related illness was observed in 78.9% of study population. Most of our patients had Type I HRS (75.4%) followed by Type II (24.53%). In our study, death was occurred in all patients with worsened hepatic and renal dysfunction (100%) and the difference was statistically significant. **Conclusion:** Hepato Renal Syndrome is a major decompensation in advanced alcohol induced liver cirrhosis with a high short-term mortality rate.

Keywords: Decompensated Chronic Liver Disease and Cirrhosis, Type 1 and Type 2 Hepato-renal Syndromes

1. Introduction

Approximately 10,000 to 24,000 deaths from cirrhosis may be attributable to alcohol consumption each year, according to the National Institutes of Health. Approximately 10 to 35% of heavy drinkers develop alcoholic hepatitis, and 10 to 20% develop cirrhosis. Cirrhosis- Is a type of liver damage where healthy cells are replaced by scar tissue. Renal dysfunction - Is a common complication of liver cirrhosis and of utmost clinical and prognostic relevance.

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Patients with cirrhosis are more prone to developing Acute Kidney Injury (AKI) than the non-cirrhotic population. Hepatorenal Syndrome (HRS) is common, with a reported incidence of 10% among hospitalized patients with cirrhosis and ascites. In decompensated cirrhotics, the probability of developing HRS with ascites ranges between 8-20% per year and increases to 40% at 5 years

Common causes of AKI in cirrhosis of liver

- Pre-renal AKI.
- Hepatorenal syndrome type of AKI (HRS-AKI, formerly known as 'type 1').
- Acute Tubular Necrosis.

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. Renal dysfunction usually results from renal vasoconstriction in the setting of systemic and splanchnic arterial vasodilatation in patients with advanced cirrhosis[1].

Types of HRS

- Type-1 HRS - 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[2].
- Type-2 HRS - Occurs in patients with refractory ascites with either a steady but moderate degree of functional renal failure (≥1.5 mg/dl) or a deterioration in kidney function that does not fulfil the criteria for HRS type-1[2].

Several studies suggest that HRS is associated with the highest mortality of all types of AKI[3]. Hepatic-renal syndrome is thought to be due to splanchnic vasodilation causing hormonal imbalances that ultimately result in renal vasoconstriction and impaired renal function[4,5]. In patients with cirrhosis, acute renal failure is mainly due to prerenal failure (caused by renal hypo-perfusion) and tubular necrosis.

HRS is a major clinical event during the course of decompensated cirrhosis. Although the most characteristic feature of the syndrome is a functional renal failure caused by an intense renal vasoconstriction, it is a more generalized process affecting the heart, brain, and the splanchnic organs[6]. The Hepatorenal Syndrome is a relatively frequent complication in cirrhotic patients with ascites that is associated with an extremely short survival. Liver size, plasma renin activity, and serum sodium concentration are predictors of hepatorenal syndrome occurrence in these patients[7]. The prognostic value of the different causes of renal failure in cirrhosis is not well established[8].

The definitive treatment for HRS1 is liver transplantation because this eliminates liver dysfunction and portal hypertension, the main pathophysiological factors that lead to the development of splanchnic and systemic vasodilatation[9]. Due to increasing critical cases along with major treatment- avoidance of alcohol, counseling by doctor, recognizing the trait about chronic alcoholism, recognizing progression to CLD, this topic is taken for the further observation.

2. Aims and Objectives

- To study clinical profile of patients admitted with alcohol induced chronic liver disease with hepato-renal syndrome.
- To study course and severity of these patients admitted with hepato-renal syndrome.
- To study outcome of these patients admitted with hepatorenal syndrome in tertiary care centre.

3. Material and Methods

3.1 Study Design

It was a prospective (observational), hospital based study.

- Patients fulfilling the inclusion criteria were included in the study and examined clinically according to the study proforma. Relevant blood and radiological investigations were carried out for them. And the data was analyzed.

3.2 Study Period

Study was carriedout from August 2018 to December 2020

3.3 Study Setting

Study was conducted at the Department of Medicine at a Tertiary Care Hospital.

The minimum estimated sample size for the aforesaid study was 57 patients

3.4 Study Population

- Sample size = 57
- P=14% L=9 % (margin of error)

- Z=1.96 q = 1-p
- Formula = $z^2 * p * q / l^2$

$$\frac{(1.96)^2 [14 (1-14)]}{(9)^2} = 57$$

3.5 Inclusion and Exclusion Criteria

3.5.1 Inclusion Criteria

- Patients (male & female) of more than or equal to 18 yrs of age, admitted with alcohol induced chronic liver disease with deranged renal parameters.
- Patients willing to give written informed consent.

3.5.2 Exclusion Criteria

- Patients with known underlying renal parenchymal disease or chronic kidney disease.
- Patients who were non-alcoholic and were having other aetiology for chronic liver disease.

Purpose of study was explained to the patient and written informed consent was obtained. Data was obtained from the study proforma which included detailed medical history, clinical examination and all relevant blood investigations and imaging studies.

The data thus obtained was statistically analysed, interpreted and tabulated and final inferences were drawn.

4. Observation & Results

Table 1. Age group and Sex distribution amongst study population

Age group	Frequency	Percent
18 to 25 years	0	0
26 to 35 years	11	19.3
36 to 45 years	19	33.3
46 to 55 years	14	24.6
more than 55 years	13	22.8
Total	57	100.0
Sex		
Female	7	12.3
Male	50	87.7
Total	57	100.0

As seen in the above (table 1), the most common age group amongst study population was 36 to 45 years (33.3%) followed 46 to 55 years (24.6%) and more than 55 years (22.8%) with a male predominance (87.7%) .

Table 2. Clinical features amongst study population

Clinical features	Frequency	Percent
Abdominal Distension	34	59.65
Icterus	57	100.00
Ascites	57	100
Flapping tremors	56	98.25
Edema Feet	42	73.68
Vomiting	24	42.11
Weakness	32	56.14
Hematemesis	28	49.12
Fever	14	24.56
Oliguria	47	82.5
Altered sensorium	38	66.7

As seen in the above (table 2), the most common clinical features amongst study population was **Icterus** and **Ascites** (100%) followed by Flapping tremors

Table 3. Duration, Quantity, Frequency of alcohol consumption and type of alchohol consumed amongst study population

Duration of alcohol consumption	Frequency	Percent
Less than 10 years	20	38.6
more than 10 years	32	61.4
Total	52	100.0
Quantity of alcohol consumption		
Light drinking (1-2 drinks/day)	12	23.1
Moderate drinking (2-4 drinks/day)	17	32.7
Heavy drinking (>4 drinks per day)	23	44.2
Total	52	100.0
Frequency of alcohol consumption		
Daily	24	45.6
Once a week	19	36.8
Monthly	5	10.5
Social drinking	4	7.02
Total	52	100.0
Type of alcohol consumed		
Whiskey	16	30.8
Rum	9	17.3
Beer	1	1.9
Country liquor	26	50.0
Total	52	100.0

(98.25%), Oliguria (82.5%), Edema Feet (73.68%), Altered sensorium (66.7%) and Abdominal Distension (59.65%)

Table 4. Type of Hepato-Renal Syndrome (HRS) amongst study population

Type of HRS	Frequency	Percent
Type I *	43	75.4
Type II**	14	24.53
Total	57	100.0

As seen in the above (table 3), most of study population had history of alcohol intake for more than 10 years (61.4%) followed by less than 10 years (38.6%). Heavy drinking (>4 drinks per day) (44.2%) followed by Moderate drinking (2-4 drinks/day) (32.7%), and Light drinking (1-2 drinks/day) (23.1%) were observed in the study population.History of daily alcohol intake noted was (45.6%) followed by Once a week (36.8%), Monthly (10.5%) and Social drinkers (7.02%).As seen in the above table, most of study population had history of country liquor (50%) followed by Whiskey (30.8%), Rum (17.3%) and Beer (1.9%)

Table 5. Various laboratory parameters observed in our study population

Hemoglobin	**Frequency**	**Percent**
less than 7	8	14.0
7.1 to 10	30	52.6
more than 10	19	33.3
Total	57	100.0
TLC		
Decreased	3	5.3
Increased	31	54.4
Normal	23	40.4
Total	57	100.0
Platelet count		
Decreased	51	89.5
Normal	6	10.5
Total	57	100.0
Total Bilirubin (Units)		
less than 3	10	17.5
3 to 8	20	35.1
8.1 to 12	6	10.5
more than 12	21	36.8
Total	57	100.0
SGOT		
Normal	8	14.0
Raised	49	86.0
Total	57	100.0
SGPT		
Normal	34	59.6
Raised	23	40.4
Total	57	100.0
Sodium		
Hyponatremia	45	78.9
Normal	12	21.1
Total	57	100.0
Potassium		
Hyperkalemia	5	8.8
Hypokalemia	20	35.1
Normal	32	56.1
Total	57	100.0
Sr.Albumin		
Decreased	52	91.2
Normal	5	8.8
Total	57	100.0
Sr. Creatinine		
less than 3	11	19.3
3 to 6	29	50.9
more than 6	17	29.8
Total	57	100.0
BUN		
less than 50	6	10.5
50 to 100	29	50.9
more than 100	22	38.6
Total	57	100.0
INR		
Normal	4	7.0
Raised	53	93.0
Total	57	100.0

Previous H/O admissions for similar or related illness was observed in 78.9% of study population.

As seen in the above (table 4), most of study population had Type I HRS (75.4%) followed by Type II (24.53%).

*Type 1 HRS: Associated with rapid and progressive impairment of renal function

**Type 2 HRS : Associated with gradual & slow impairment of renal function

Oesophageal Varices was observed in 19.3% of study population.**Worsened Renal dysfunction** after treatment was observed in 68.4% of study population while improvement was observed in 31.6%. **Worsened hepatic dysfunction** after treatment was observed in 68.4% of study population while improvement was observed in 31.6%. **Hemodialysis** requirement was observed in 22.8% of study population.

Table 6. Number of cycles of Hemodialysis amongst study population

HD	Frequency	Percent
1 cycle	4	30.8
2 cycle	7	53.8
3 cycle	1	7.7
4 cycle	1	7.7
Total	13	100.0

As seen in the above (table 6), most of study population took 2 cycle of Hemodialysis (53.8%) followed by 1 cycle (30.8%) and 3 cycle (7.7%).

At the end of the study we observed that 68.4 % of study population succumbed while 31.6 % responded to treatment and were discharged.

Table 7. Varices v/s Final outcome amongst study population

Death Discharged			Final outcome		Total
Varices	No	Count	28	18	46
		%	71.8%	100.0%	80.7%
	Yes	Count	11	0	11
		%	28.2%	0.0%	19.3%
Total		Count	39	18	57
%		100.0%	100.0%	100.0%	

As seen in the above (table 7), death was commonly occurred in study population with varices (28.2%) and the difference was statistically significant.

As seen in the above (table 8), death was commonly occurred in study population with Worsened renal dysfunction (100%) and the difference was statistically significant.

As seen in the above (table 9), death was commonly occurred in study population with Worsened hepatic dysfunction (100%) and the difference was statistically significant.

Table 8. Renal dysfunction vs Final outcome amongst study population

Death Discharged			Final outcome		Total
renal dysfunction	Worsened	Count	39	0	39
		%	100.0%	0.0%	68.4%
	Improved	Count	0	18	18
		%	0.0%	100.0%	31.6%
Total		Count	39	18	57
%		100.0%	100.0%	100.0%	

Table 9. Hepatic dysfunction vs Final outcome amongst study population

Death Discharged			Final outcome		Total
hepatic dysfunction	Worsened	Count	39	0	39
		%	100.0%	0.0%	68.4%
	Improved	Count	0	18	18
		%	0.0%	100.0%	31.6%
Total		Count	39	18	57
%		100.0%	100.0%	100.0%	

5. Discussion

Liver disease related to alcohol consumption fits into 1 of 3 categories: fatty liver, alcoholic hepatitis, or cirrhosis. Fatty liver, which occurs after acute alcohol ingestion, is generally reversible with abstinence and is not believed to predispose to any chronic form of liver disease if abstinence or moderation is maintained. Alcoholic hepatitis is an acute form of alcohol-induced liver injury that occurs with the consumption of a large quantity of alcohol over a prolonged period of time; it encompasses a spectrum of severity ranging from asymptomatic derangement of biochemistries to fulminant liver failure and death. Cirrhosis involves replacement of the normal hepatic parenchyma with extensive thick bands of fibrous tissue and regenerative nodules, which results in the clinical manifestations of portal hypertension and liver failure.

The liver and, to a lesser extent, the gastrointestinal tract, are the main sites of alcohol metabolism.

Within the liver there are 2 main pathways of alcohol metabolism: Alcohol dehydrogenase and cytochrome P-450 (CYP) 2E1. Alcohol dehydrogenase is a hepatocyte cytosolic enzyme that converts alcohol to acetaldehyde. Acetaldehyde subsequently is metabolized to acetate via the mitochondrial enzyme acetaldehyde dehydrogenase. CYP 2E1 also converts alcohol to acetaldehyde[10].

Liver damage occurs through several interrelated pathways. Alcohol dehydrogenase and acetaldehyde dehydrogenase cause the reduction of Nicotinamide Adenine Dinucleotide (NAD) to NADH (reduced form of NAD). The altered ratio of NAD/NADH promotes fatty liver through the inhibition of gluconeogenesis and fatty acid oxidation. CYP 2E1, which is upregulated in chronic alcohol use, generates free radicals through the oxidation of Nicotinamide Adenine Dinucleotide Phosphate (NADPH) to NADP. Chronic alcohol exposure also activates hepatic macrophages, which then produce tumor necrosis factor-alpha (TNF-alpha)[11]. TNF-alpha induces mitochondria to increase the production of reactive oxygen species. This oxidative stress promotes hepatocyte necrosis and apoptosis, which is exaggerated in the alcoholic who is deficient in antioxidants such as glutathione and vitamin E. Free radicals initiate lipid peroxidation, which causes inflammation and fibrosis. Inflammation is also incited by acetaldehyde that, when bound covalently to cellular proteins, forms adducts that are antigenic[12].

In the present study, the study population's most common age group was between 36 and 45 years (33.3%), 46 to 55 years (24.6%), and more than 55 years (22.8%) with the mean age of 43 + 7.8 years amongst study population. This was in agreement with the study conducted by Suthar et al.,[108] (41 yrs), Sarin et al (43±8.7 yrs). This result emphasizes the fact that younger and middle aged individuals are under the influence of alcoholism which is alarming, taking into consideration that this is the most productive age group of society.

In the present study, there was male predominance (87.7%) as compared to female (12.3%). Similarly in the study conducted by Suthar et al., it was observed that only 8 of the patients was female comparable to the study by where all the cases were male. This is due to the social norms and cultural influences in our country, where alcohol consumption rate in females is much less compared to the western population.

Alcoholic hepatitis is a syndrome with a spectrum of severity, and therefore manifesting symptoms vary. Symptoms may be nonspecific and mild and include anorexia and weight loss, abdominal pain and distention, or nausea and vomiting. Alternatively, more severe and specific symptoms can include encephalopathy and hepatic failure. Physical findings include hepatomegaly, jaundice, ascites, spider angiomas, fever, and encephalopathy.

Established alcoholic cirrhosis can manifest with decompensation without a preceding history of fatty liver or alcoholic hepatitis. Alternatively, alcoholic cirrhosis may be diagnosed concurrently with acute alcoholic hepatitis. The symptoms and signs of alcoholic cirrhosis do not help to differentiate it from other causes of cirrhosis. Patients may present with jaundice, pruritus, abnormal laboratory findings (eg, thrombocytopenia, hypoalbuminemia, coagulopathy), or complications of portal hypertension, such as variceal bleeding, ascites, or hepatic encephalopathy.

In the present study, Icterus and ascitis (100%) followed by Flapping tremors (98.25%), Oliguria (82.5%), Edema Feet (73.68%), Altered sensorium (66.7%) and Abdominal Distension (59.65%) were the commonest clinical features. This is agreement with the various studies conducted by Suthar et al (60%) in ascites was common finding. Similar findings was observed by Pathak et al.[13], (57.5%), Mendenhall et al.(50.9%). Our findings are comparable to the study conducted by Singh et al. in which all patients had jaundice, ascites and esopheageal varices and 36(85.7%) patients presented with oliguria, 30(71.4%) with abdominal distension, 26(61.9%) with altered sensorium, 17(40.9%) with fever, 11(26.2%) with pain abdomen, 10(23.8%) with malena and 4(9.5%) with haematemesis. Salerno F et al.,[14] demonstrated that all the patients of HRS presented with ascites, jaundice, hepatic encephalopathy and in renal failure. Watt K et al[15], observed that most of the patients with HRS present with oliguria, high coloured urine, ascites change in mental status, nausea, vomiting and GI bleed.

In the present study, most of study population had history of alcohol intake for more than 10 years (61.4%) followed by less than 10 years (38.6%) and most of them were heavy drinkers (>4 drinks per day) (44.2%) while moderate and light drinkers (2-4 drinks/day and 1-2 drinks/day) were 32.7% and 23.1% respectively. Intake of 40 to 80 grams ethanol/day by males and of 20 to 40 grams/day by females for 10 to 12 years is a general predictor of more severe cases of ALD, including alcoholic steatohepatitis, fibrosis, and cirrhosis (Becker et al. 1996)

In our study, 45.6% had daily alcohol drinkers followed by once a week (36.8%), Monthly (10.5%) and Social drinkers (7.02%). Regular drinking with intermittent bingeing was the most common drinking pattern. Risk being more in patients who were social drinkers but with frequent socializing and binge drinking. Regular drinking with intermittent bingeing was seen significantly higher in the alcoholic cirrhosis group in comparison to the ADS group (65% vs. 37.5%). This observation is also seen in animal experiments, where after an alcoholic binge, ethanol metabolism causes oxidative stress and hepatic mitochondrial DNA degradation in mice. Their progression also depends on the pattern of alcohol intake – drinking alcohol at meal times results in a lower risk of liver disease than consumption at other times; intermittent drinking is more sparing for the liver than a continuous supply of alcohol.

In our study, history of country liquor (50%) followed by Whiskey (30.8%), Rum (17.3%) and Beer (1.9%). In India vast majority of people consume illicit country liquor. The exact percentage of alcohol depends on the method of brewing which is often below standard and unsupervised. Since it is affordable to the lower income groups it is widely prevalent and as such we found that it was the most commonly used alcoholic beverage in our study group.

In our study, previous history of admissions due to similar or related illness was observed in 78.9% of study population. Most of our patients had Type I HRS (75.4%) followed by Type II (24.53%).

In the present study, most of study population had hemoglobin between 7.1 to 10 (52.4%) followed by more than 10 (33.3%), and less than 7 (14%). Increased TLC was observed in 54.4% of study population. DecreasedPlatelet count was observed in 54.4% of study population. This is agreement with the findings of Suthar et al., (10.1g%). Similar findings was observed by Sarin et al.,[16] (10.2g%) and Pathak et al., (11.85g%) respectively. This may be due to the low socioeconomic and poor nutritional status of most of the cases. The mean total leucocyte count was 8904 ± 2679.2. Platelet count was72460 ± 12159.4 (< 1.5 lakh) while in study by Pathak et al., it was seen in 57.9% cases.

Mechanisms for AKI due to underlying cirrhosis AH and cirrhosis are associated with systemic arterial vasodilation because of increased endogenous vasodilators, especially nitric oxide and 3', 5' cyclic guanosine monophosphate. Systemic arterial vasodilation causes a decrease in systemic vascular resistance (SVR) leading to high cardiac output and hyperdynamic circulation. Increase in cardiac output may be insufficient to keep up with a drop in SVR leading to hypotension. Further insult in the form of sepsis or decreased cardiac output may overcome renal blood flow autoregulation, rendering patients prone to pre-renal AKI and Acute Tubular Necrosis (ATN).

In the present study, most of study population had total bilirubin between more than 12 (36.8%) followed by 3 to 8 (35.1%) and less than 3 (17.5%). Raised SGOT was observed in 86% of study population. Raised SGPT was observed in 40.4% of study population. Hepatic encephalopathy, derangement in renal function, hyperbilirubinemia, and prolonged prothrombin time are seen more often in patients who succumb to the illness than in those who survive. Both the discriminant function and the Model for End-stage Liver Disease (MELD) score can be used to predict short-term mortality in patients with alcoholic hepatitis

Hyponatremia and Hypokalemia was observed in 78.9% and 35.1% of study population respectively in our study. In the present study, Raised INR was observed in 93% of study population. There were 4 patients having INR value between 3.0-3.5. All 4(100%) patients died, none survived. This finding suggests that, as patient's INR value increases above 2.5, there is increased in mortality of the patient. This finding is statistically significant as 'p' value is 0.008636. Mean INR of survived patients was 1.45 & in non survived patients were 2.16 in our study. This suggest that the mean INR level is more in non survived patients than in survived patients

In the present study, Raised FBS level was observed in 61.4% of study population.

In the present study, improvement in renal and hepatic function after treatment was observed in 31.6% while in 68.4% worsening of function was observed leading to death. Our findings are comparable to the study conducted by Singh et al., in which 17 patients survived and 25 succumbed to hepatorenal syndrome.

In the present study, hemodialysis requirement was observed in 22.8% of study population with most of study population took 2 cycle of hemodialysis (53.8%) followed by 1 cycle (30.8%) and 3 cycle (7.7%)

In our study, death was observed in 2 out 7 women patients this is because women are more susceptible to alcohol-related liver damage than men. It was observed that higher blood alcohol concentrations in women than

in men who ingest the same amount of alcohol, resulting from a lower proportion of body water in females compared with males of equal weight (Mumenthaler et al. 1999)[17]. There also are reports that women possess a lower capacity than men to oxidize ethanol in the gut, a process called first-pass metabolism (Frezza et al. 1990). This deficit in women allows greater quantities of ethanol into the portal circulation, thereby exposing their livers to higher ethanol concentrations.

Most of the patients who died due to hepatorenal syndrome had oliguria (82.1%), bilirubin more than 12 (48.7%), raised SGOT (87.2%), raised SGPT (46.2%), hypokalemia (28.2%), decreased albumin levels (94.9%), Serum creatinine levels>3 to 6 (87.2%), BUN > 100 (48.7%) and varices (28.2%). In the similar study by Aher Sangeeta et al., there were 3 patients having Bilirubin value between 40-50 mg/ dl. All 3 patients (100%) died, none survived. This finding suggest that, as patient's Bilirubin value increases above 20 mg/dl, there is increased in mortality of the patient. Our findings are comparable to the study conducted by Singh et al., in which Oliguria (96%) and hepatic encephalopathy (84%) was more predominant in the non survival group. Serum creatinine values were found to be insignificantly higher in non survival group. Serum bilirubin levels were found to be significantly higher in non survival group as compared to survival group. Hypoalbuminea, hyponatremia and Coagulopathy were more pronounced in non survival group. They also reported that in 13 patients having Serum Albumin value between 2.5-3.0 g/dl. Out of these 8(61.53%) were discharged and 5(38.47%) patients died. This indicates that, as patients serum Albumin decreases there is increase in mortality of the patient.

Our findings are comparable with the study conducted by Aher Sangeeta et al., in which 7 patients having creatinine value between more than 5 mg/dl. All 7 patients (100%) died, none survived. This finding suggests that, there is increased mortality as patient's creatinine increases above 3 mg/dl. Mean creatinine of survived patients was 2.12 mg/dl and in non survived patients was 4.6 mg/dl in our study. In their study there were 13 patients having creatinine >4mg/dl, out of which 12 patients (92.3%) died. So all these findings suggest that as patient's Creatinine value increases, there is increased in mortality of the patient. In our study, death was occurred in all patients with worsened hepatic and renal dysfunction (100%) and the difference was statistically significant. Our findings are similar to other studies. [3,6,7,8,10]. Our findings are comparable to the study conducted by Singh et al., in which the poor prognostic factors in non survival group were found to be presence of ascites, severe jaundice, hepatic encephalopathy, alcohol abuse, hypoalbuminemia, progressive renal failure.

Thus hepatorenal syndrome in decompensated cirrhosis is not that uncommon and judicious treatment helps in saving a significant number of patients. Once considered fatal with mortality of greater than 90% in hepatorenal syndrome, there is improved prognosis of the entity with novel therapies including terlipressin, dopamine and albumin infusion. This study observed 31% survival in patients of hepatorenal syndrome. However to decrease the incidence of this fatal disease, the emphasis should be to educate the society about abstinence from alcohol and prevention of hepatotropic viral infection. The main aspect of hepatorenal syndrome management is preventing its recurrence. Lastly, the preventive factors are avoidance of alcohol, counseling by doctor, recognizing the trait about chronic alcoholism, recognizing progression to CLD. Many treatment options for hepatorenal syndrome patients are now promising but the only definitive treatment for both Type 1 and Type 2 HRS is liver transplantation. The most suitable bridge treatment or treatment for patients who are not eligible for transplantation is a combination of terlipressin and albumin.

6. Conclusion

Hepato Renal Syndrome is a major decompensation in advanced alcohol induced liver cirrhosis. It entails a high short-term mortality rate. In our study the prevalence of Type I HRS was more compared to Type II. It more commonly prevailed in patients who were daily drinkers. The most suitable bridge treatment for patients who were not eligible for transplantation is treatment with combination of terlipressin and albumin. Finally, Liver Transplant is the only curative treatment for patients with end stage liver disease with complications. Lastly, the preventive factors are avoidance of alcohol, counselling by doctor, recognizing the trait about chronic alcoholism, recognizing progression to CLD and its complications.

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