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*Original Research Article*

# A study on clinical profile of patients with cirrhosis of liver with or without hepatic encephalopathy

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## Abstract

In patients suspected of having cirrhosis, abdominal imaging typically ultrasound is obtained to evaluate the liver parenchyma and to detect extrahepatic manifestations of cirrhosis. A liver biopsy is required to definitively confirm the diagnosis. However, it is generally not necessary if the clinical, laboratory, and radiologic data strongly suggest the presence of cirrhosis and the results would not alter the patient's management. Noninvasive serologic and radiographic methods for diagnosing cirrhosis are also being developed. We had selected 22 cases of liver cirrhosis without hepatic encephalopathy (Group A) and 22 cases of liver cirrhosis with hepatic encephalopathy (Group B). During the Study period approximately 200 to 250 cases satisfying the Inclusion and Exclusion criteria were expected in each group. So after selecting the first eligible case in each Group by simple random procedure, every 10th eligible case in that Group have been enrolled till the required sample size of 22 cases in each Group was achieved. In the present study mean serum bilirubin in group A was 2.06 ± 2.21 mg/dL and in group B was 4.16 ± 3.40 mg/dL. In the present study, mean serum albumin in group A was 3.21 ± 0.69 gm/dL and in group B 3.06 ± 0.55 gm/dL. In the present study, in group A 63.6% patients had ascites, 36.4% did not have ascites. In group B 59.1% patients had ascites, 40.9% did not have ascites.

**Keywords:** Clinical profile, cirrhosis of liver, hepatic encephalopathy

## Introduction

Cirrhosis represents a late stage of progressive hepatic fibrosis characterized by distortion of the hepatic architecture and the formation of regenerative nodules. It is generally considered to be irreversible in its advanced stages, at which point the only treatment option may be liver transplantation [1].

Patients with cirrhosis are susceptible to a variety of complications, and their life expectancy is markedly reduced.

There are numerous causes of liver disease that can result in cirrhosis, either by causing chronic hepatic inflammation or cholestasis. The most common causes of cirrhosis in the United States are hepatitis C, alcoholic liver disease and nonalcoholic liver disease, which

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together accounted for approximately 80 percent of patients on the liver transplantation waitlist between 2004 and 2013 [2].

Patients with compensated cirrhosis may be asymptomatic or they may report nonspecific symptoms, such as anorexia, weight loss, weakness, and fatigue. Patients with decompensated cirrhosis may present with jaundice, pruritus, signs of upper gastrointestinal bleeding (hematemesis, melena, hematochezia), abdominal distension from ascites, or confusion due to hepatic encephalopathy. Patients with cirrhosis may experience muscle cramps, which can be severe [3]. The cause of diarrhea in patients with cirrhosis may be multifactorial e.g., alterations in small bowel motility, small bowel bacterial overgrowth, changes in intestinal permeability and bile acid deficiency.

Several laboratory abnormalities may be seen in patients with cirrhosis. In addition, because it is common for panels of serum chemistries to be sent for screening or evaluation of specific complaints, laboratory abnormalities may be the first indication that a patient has cirrhosis. Common abnormalities include elevated serum bilirubin, abnormal aminotransferases, elevated alkaline phosphatase, gamma-glutamyl transpeptidase, a prolonged prothrombin time, elevated international normalized ratio (INR), hyponatremia, and thrombocytopenia [4].

In patients suspected of having cirrhosis, abdominal imaging typically ultrasound is obtained to evaluate the liver parenchyma and to detect extrahepatic manifestations of cirrhosis. A liver biopsy is required to definitively confirm the diagnosis. However, it is generally not necessary if the clinical, laboratory, and radiologic data strongly suggest the presence of cirrhosis and the results would not alter the patient's management. Noninvasive serologic and radiographic methods for diagnosing cirrhosis are also being developed [5].

HE is not a single clinical entity. It may reflect either a reversible metabolic encephalopathy, brain atrophy, brain edema, or any combination of these conditions. The mechanisms causing brain dysfunction in liver failure are still unknown. In advanced coma, the effects of brain swelling, impaired cerebral perfusion, and reversible impairment of neurotransmitter systems cannot be distinguished. Furthermore, these events overlap, at least in models of acute liver failure [6].

The metabolic factors contribute to the development of HE. Ammonia is clearly implicated; in addition, there may be a role for inhibitory neurotransmission through gamma-aminobutyric acid (GABA) receptors in the central nervous system and changes in central neurotransmitters and circulating amino acids. These hypotheses are not mutually exclusive, and multiple factors may be present at the same time. Therapies for hepatic encephalopathy are based upon these hypotheses.

## Methodology

**Study area:** Department of Medical Gastroenterology.
**Study design:** Prospective, observational study.
**Study population:** Cirrhotic patients who came to Department of Gastroenterology. The inclusion and exclusion criteria were as follows:

### Inclusion criteria

1. Age > 18 years.
2. Cirrhosis of liver with or without hepatic encephalopathy.

### Exclusion criteria

1. Patients with Suspected Inflammatory Bowel Disease.
2. Patients with Gastro Intestinal bleed.

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3. Patients on Proton Pump Inhibitors.
4. Patients on Non-steroidal anti-inflammatory drugs.
5. Patients with Diarrhea.

**Sampling procedure**

We had selected 22 cases of liver cirrhosis without hepatic encephalopathy (Group A) and 22 cases of liver cirrhosis with hepatic encephalopathy (Group B).
During the Study period approximately 200 to 250 cases satisfying the Inclusion and Exclusion criteria were expected in each group. So after selecting the first eligible case in each Group by simple random procedure, every 10th eligible case in that Group have been enrolled till the required sample size of 22 cases in each Group was achieved.

**Methods of measurement of outcome of interest and data collection methods**

A prospective observational study was conducted with 22 cirrhotic patients without hepatic encephalopathy and 22 cirrhotic patients with hepatic encephalopathy. Diagnosis of liver cirrhosis was based on clinical clues from the patient's medical history, physical examination, laboratory tests, abdominal ultrasonography and CT abdomen.
The degree of liver insufficiency was assessed by the Child-Pugh classification and Model of End Stage Liver Disease-Sodium (MELD Na). Hepatic encephalopathy staging was done according to West-Haven criteria.
Fecal calprotectin assay was done in patients of both the groups using Chemi Luminiscence Immuno Assay (CLIA). Calprotectin values <50 mcg/gm of feces was considered as normal and not indicative of inflammation in the gastrointestinal tract.
A "Patient information sheet" (Appendix-C) in English and also in local language was handed over to the patient prior to inclusion in the study. All patients gave written consent prior to inclusion in the study. The data collected was summarized using tables and charts.

**Statistical Methods:** The information collected regarding all the selected cases was recorded in a Master Chart in Excel sheet. Data analysis was done with the help of computer using SPSS statistical package-Version 17. Using this software, measures of central tendency, measures of dispersion, t-value, chi square and 'p' values was calculated. ANOVA and unpaired t-test was used to test the significance of difference between quantitative variables and Yate's and Fisher's chi square tests for qualitative variables. Post-hoc multiple comparison procedures with Bonferonni correction were performed to determine individual differences between the groups. A 'p' value less than 0.05 denoted significant relationship.

**Results**

**Table 1:** Mean age in group A and group B

| | **Mean Age in Years** |
|---|---|
| Group A | 58.32± 9.49 |
| Group B | 56.45± 9.86 |

Mean age in group A was 58.32 ± 9.49 years and group B was 56.45 ± 9.86 years.

**Table 2:** Sex distribution in group A and group B

| | **Male** | **Female** |
|---|---|---|
| Group A | 81.8% | 18.2% |
| Group B | 90.9% | 9.1% |

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Group A comprised 18(81.8%) male patients and 4(18.2%) female patients. Group B comprised 20(90.9%) male patients and 2(9.1%) female patients.

**Table 3:** Mean serum creatinine in group A and group B

| | **Mean serum Creatinine (mg/dL)** |
|---|---|
| Group A | 1.04± 0.78 |
| Group B | 1.26± 1.63 |

Mean serum creatinine in group A was 1.04 ± 0.78 mg/dL and in group B was 1.26 ± 1.63 mg/dL.

**Table 4:** Mean serum bilirubin in group A and group B

| | **Mean serum bilirubin (mg/dL)** |
|---|---|
| Group A | 2.06± 2.21 |
| Group B | 4.16± 3.40 |

Mean serum bilirubin in group A was 2.06 ± 2.21 mg/dL and in group B was 4.16 ± 3.40 mg/dL.

**Table 5:** Mean serum sodium in group A and group B

| | **Mean Serum Sodium (mEq/L)** |
|---|---|
| Group A | 136.36±4.03 |
| Group B | 133.64± 9.16 |

Mean serum sodium in group A was 136.36 ±4.03 mEq/L and in group B was 133.64 ± 9.16 mEq/L.

**Table 6:** Mean serum albumin in group A and group B

| | **Mean Serum Albumin (gm/dL)** |
|---|---|
| Group A | 3.21± 0.69 |
| Group B | 3.06± 0.55 |

Mean serum albumin in group A was 3.21 ± 0.69 gm/dL and in group B was 3.06 ± 0.55 gm/dL.

**Table 7:** Mean serum PT-INR in group A and group B

| | **Mean PT-INR** |
|---|---|
| Group A | 1.25± 0.19 |
| Group B | 1.51± 0.34 |

Mean PT-INR in group A was 1.25 ± 0.19 and in group B was 1.51± 0.34.

**Table 8:** Mean MELD Na score in group A and group B

| | **Mean Meld Na Score** |
|---|---|
| Group A | 14.55± 6.06 |
| Group B | 21.73± 10.70 |

Mean MELD Na in group A was 14.55 ± 6.06 and in group B was 21.73 ± 10.70.

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**Table 9:** Ascites in both groups

| | Ascites | |
|---|---|---|
| | **Present** | **Absent** |
| Group A | 63.6% | 36.4% |
| Group B | 59.1% | 40.9% |

In group A 14 (63.6%) patients had ascites, 8 (36.4%) did not have ascites. In group B 13 (59.1%) patients had ascites, 9 (40.9%) did not have ascites.

## Discussion

In the present study, mean age of patients in group A was 58.32±9.48 years and in group B was 56.45±9.86 years which is similar to the study done by Tamara Alempijevic, *et al*. [7] where mean age of patients was 55.2±15.1 years.

In the present study 86.4% were males and 13.6% were females. Similar sex distribution was also seen in a study by Tamara Alempijevic, *et al*. [7] where 80% were males and 20% were females.

In the present study baseline biochemical parameters of patients in Group A and Group B were similar to the study done by Inas Elkhedr Mohamed *et al*. [8].

In the present study, mean serum creatinine in group A was 1.036±0.78 mg/dL and in group B was 1.264±1.63 mg/dL which was similar to study by Inas Elkhedr Mohamed *et al*. [8] where mean serum creatinine in uncomplicated cirrhosis group was 0.86±0.2 mg/dL and in HE group was 1.15±0.6 mg/dL. In both the studies serum creatinine was higher in patients with hepatic encephalopathy.

In the present study mean serum bilirubin in group A was 2.06 ± 2.21 mg/dL and in group B was 4.16 ± 3.40 mg/dL. Whereas in study by Inas Elkhedr Mohamed *et al*. [8], serum bilirubin was 1.5±0.3 mg/dL in uncomplicated cirrhosis and 2.9±0.3 mg/dL in HE group. In both the studies serum bilirubin was higher in patients with hepatic encephalopathy.

In the present study mean serum sodium in group A was 136.36 ±4.03 mEq/L and in group B was 133.64 ± 9.16 mEq/L which was similar to a study by Inas Elkhedr Mohamed *et al*[8] where serum sodium was 137±1.1 mEq/L in uncomplicated cirrhosis and 126± 2 mEq/L in HE group. Both the studies show that serum sodium was lower in patients with hepatic encephalopathy.

In the present study, mean serum albumin in group A was 3.21 ± 0.69 gm/dL and in group B 3.06 ± 0.55 gm/dL which was similar to a study by Amany Lashin *et al*. [9] where mean serum albumin in uncomplicated cirrhosis was 2.5±0.54 gm/dL and in hepatic encephalopathy was 2.16±0.58 gm/dL. In both the studies serum albumin was lower in hepatic encephalopathy group.

In present study mean PT-INR in group A was 1.25 ± 0.19 and in group B was 1.51± 0.34 which was comparable to a study by Amany Lashin *et al*. [9] where it was 1.39±0.24 in uncomplicated cirrhosis and 1.51±0.3 in hepatic encephalopathy group. In both studies PT-INR was higher in hepatic encephalopathy group.

In the present study mean MELD Na in group A was 14.55 ± 6.06 and in group B was 21.73 ± 10.70.

In the present study, in group A 63.6% patients had ascites, 36.4% did not have ascites. In group B 59.1% patients had ascites, 40.9% did not have ascites. In the study Gundling *et al*. [5]. 62.3% patients had ascites.

In the present study, in group A, 40.9% of patients belonged to CTP class A, 45.5% belonged to class B and 13.6% belonged to class C. In group B 9.1% belonged to CTP class A, 31.8% belonged to class B and 59.1% belonged to class C. In a study by Tamara Alempijevic *et al*.

[7], 33.3% of patients belonged to CTP class A, 46.7% belonged to class B and 20% belonged class C [10].

## Conclusion

- Mean serum creatinine in group A was 1.036±0.78 mg/dL and in group B was 1.264±1.63 mg/dL.
- Mean serum bilirubin in group A was 2.06 ± 2.21 mg/dL and in group B was 4.16 ± 3.40 mg/dL.
- Mean serum sodium in group A was 136.36 ±4.03 mEq/L and in group B was 133.64 ± 9.16 mEq/L.
- Mean serum albumin in group A was 3.21 ± 0.69 gm/dL and in group B 3.06 ± 0.55 gm/dL.
- Mean PT-INR in group A was 1.25 ± 0.19 and in group B was 1.51± 0.34.
- Mean MELD Na in group A was 14.55 ± 6.06 and in group B was 21.73 ± 10.70.
- In group A, 40.9% of patients belonged to CTP class A, 45.5% belonged to class B and 13.6% belonged to class C. In group B 9.1% belonged to CTP class A, 31.8% belonged to class B and 59.1% belonged to class C.

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