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Original Research Paper

# Clinical profile of Autoimmune hepatitis related decompensated Cirrhosis and their treatment outcomes: A study from Western India

**Authors**

**[1]Dr.Vikramaditya L. Rawat, [2]Dr.Shamshersingh Chauhan, [3]Dr.Vikas Pandey, [4]Dr Mit Shah,**

**[5]Dr.Kiran B, [6]Dr. Meghraj Ingle**

*[1,4,5]Resident doctor, Department of Medical Gastroenterology, Lokmanya Tilak Municipal General Hospital and Lokmanya Tilak Municipal Medical College, Sion ,Mumbai,India*
*[2]Assistant Professor, Department of Medical Gastroenterology, Lokmanya Tilak Municipal General Hospital and Lokmanya Tilak Municipal Medical College, Sion ,Mumbai,India*
*[3]Associate Professor, Department of Medical Gastroenterology, Lokmanya Tilak Municipal General Hospital and Lokmanya Tilak Municipal Medical College, Sion ,Mumbai,India*
*[6]Professor and Head of Department, Department of Medical Gastroenterology, Lokmanya Tilak Municipal General Hospital and Lokmanya Tilak Municipal Medical College, Sion , Mumbai, India*

**Corresponding Author:** Dr. Meghraj Ingle, Professor and Head of Department, Department of Medical Gastroenterology, Lokmanya Tilak Municipal General Hospital and Lokmanya Tilak Municipal Medical College, Sion , Mumbai, India

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ABSTRACT

**Background:** Our study was undertaken to analyse the clinical profile and management strategies of patients having AIH related decompensated cirrhosis in a non-transplant resource limited setting from western India. The parameters analysed were the demographic profile, 1 year transplant-free survival, occurrence of infections and changes in CTP and MELD scores at the end of one year in patients with AIH related decompensated cirrhosis . **Methods:** In the retrospective data collected from January 2017 to December 2020, patients with AIH related decompensated cirrhosis were treated with steroids with/without immunomodulators using a predetermined treatment protocol. The demographic data, types of presentation, treatment imparted, response, changes in CTP, MELD and survival at the end of one year was studied. **Results:** Thirty-five patients with AIH related decompensated cirrhosis were studied. The mean age was 45.4 ± 14.6 years and female to male ratio was 1.2:1. Ascites (91.4%) was the most common form of decompensating event at presentation. Twenty-eight patients received treatment with immunosuppressants. The overall survival rate was 85.7% at the end of one year. The most common cause of death was sepsis with spontaneous bacterial peritonitis (SBP) being the most common form of infection .Encephalopathy on presentation was a significant factor leading to mortality in our study (p=0.0001). There was no significant difference in the change in MELD, survival rates or rates of infections based on the treatment received by the patient of AIH. **Conclusion:** In a non-transplant resource limited setting, immunosuppressive therapy did not improve survival in patients with AIH related decompensated cirrhosis.

***Keywords: Decompensated liver disease, Autoimmune hepatitis, Immunosuppressive therapy, Transplant free survival***

## INTRODUCTION:

Autoimmune hepatitis (AIH) is characterised by circulating autoantibodies, interface hepatitis on histology and elevated immunoglobulin levels [1].Diagnosis is made by excluding the common causes of chronic liver disease and supported by various scoring systems and liver biopsy [2,3]. The prevalence is around 5% of all patients with chronic liver disease in India [4-10]. Approximately one third of adult patients and one half of paediatric patients present with cirrhosis at diagnosis [11-16]. Liver transplantation in AIH related decompensated liver disease has excellent five-year survival rates varying from 83-92% in western countries [17]. However, in India, treatment with immunosuppressive medicines may be administered to these cohort of patients as liver transplantation is still a farfetched dream for most of the patients in India. Very few studies have addressed the response to corticosteroids in AIH related decompensated cirrhosis in our country [18,19]. This

study was undertaken to analyse the clinical profile and management strategies of patients having AIH related decompensated cirrhosis in a non-transplant resource limited setting from western India.

## MATERIAL AND METHODS:

**Study design:**

The retrospective analysis of prospectively maintained data of patients attending the liver clinic or admitted at a tertiary care centre in western India was collected from January 2017 to December 2020. Patients with AIH related decompensated cirrhosis were included in the study and their demographic data, types of presentation, treatment imparted, response and survival at the end of one year were looked at. Patients previously treated with immunosuppression, overlap with primary biliary cholangitis (PBC) and/or primary sclerosing cholangitis (PSC), presenting with acute-on-chronic liver failure (ACLF) were excluded from the study [20]. Ethical approval was obtained by the hospital's Institutional review board. All procedures performed in the studies were in accordance with the ethical standards of the institutional review board and are in accordance with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

**Definitions:**

Cirrhosis was diagnosed based on the clinical, radiological/ endoscopic evidence of portal hypertension and presence of >F4 fibrosis using the Ishak staging system on liver biopsy [21,22]. Decompensation was defined by the presence of clinical ascites, variceal bleed, hepatic encephalopathy or jaundice [23]. AIH was diagnosed in accordance with the 1999 revised International Autoimmune Hepatitis Group (IAIHG) diagnostic criteria [3]. Patients with a pre-treatment score of 10 points or more were included.

**Data collection:**

The prospectively maintained data of patients with AIH related decompensated cirrhosis was assessed for detailed clinical history, type of decompensation (past and current), family history, history of other autoimmune diseases, comorbidities, clinical examination and biochemical tests which included complete blood count, liver function test, renal function test, serum electrolytes, prothrombin time, international normalised ratio (INR) and blood sugar levels. Autoimmune markers like anti-nuclear antibodies (ANA) performed by indirect immunofluorescence, anti-smooth muscle antibodies (ASMA) performed by indirect immunofluorescence and anti-liver kidney microsomal types I (anti LKM1) antibodies performed by solid phase ELISA using recombinant CYP2D6 were recorded. The titres were considered positive if values were more than 1:40. Serum total immunoglobulin G level was done by immunoturbidimetric method and was found to be elevated when levels were found to be > 1500 mg/dl. Other causes of liver diseases were excluded by blood and other tests as follows: Hepatitis B surface antigen by ELISA, anti HCV antibodies by ELISA, Anti HBc total antibody by ELISA and HIV by ELISA were performed in all the patients. Serum Ceruloplasmin levels by copper oxidase method, 24-hour urinary copper measurement and slit lamp examination to look for KF ring were done to rule out Wilson's disease. History of significant alcohol intake (< 30 g/day in men and < 20 g/day in women) was assessed to rule out alcohol associated liver injury and drug induced liver injury was excluded by detailed history taking. Ultrasonography (USG), computerised tomography (CT), and/or magnetic resonance imaging (MRI) were performed to exclude hepatocellular carcinoma, vascular disorders and bile duct disorders. The diagnosis of AIH related decompensated liver disease was confirmed by liver biopsy in all the patients included in the study. Liver biopsy was done by either a percutaneous USG guided or transjugular approach, depending upon the clinical scenario.

**Statistical methods:**

Data recording was done in MS Excel. Continuous variables are reported as Mean $\pm$ Standard Deviation (SD), Median {Interquartile range (IQR)} and Range. Discrete variables are summarized in terms of frequencies and percentages. Shapiro-Wilk test was used to determine whether data sets differed from a normal distribution. We assessed the differences in categorical variables with the chi square test and fisher's exact test. We assessed the difference continuous variable between two group by Mann-Whitney U test and for two or more group Kruskal-Wallis test. All statistical analysis was performed using "R Studio version 1.4.1103". A two-tailed p value of $<0.05$ was considered to be statistically significant.

**Treatment protocol:**

All patients were counselled about the benefits and adverse effects of steroids/other immunosuppression before starting the therapy. Prior to the institution of treatment, screening for diabetes, blood pressure and osteoporosis was conducted, and they were managed according to standard protocols if present. All patients were prescribed elemental calcium of 1000 mg/day and a weekly dose of vitamin D of 60000 units along with steroids. Corticosteroids were only started after confirming the absence of active infection, acute kidney injury or variceal bleed. The presence of infection was ruled out by aspirating ascitic fluid to look for spontaneous bacterial peritonitis (SBP), in addition chest X-ray and blood/urine cultures were also obtained. Corticosteroids (Prednisolone) was started at a dose of 0.5 mg/kg [max 30 mg/day] to begin with. Steroids were continued in the same dose for 4 weeks followed by taper off by 5 mg every 2 weeks until a dose of 5–10 mg was attained, which was then continued for a prolonged period of time.

Azathioprine (1-2 mg/kg/day) or Mycophenolate mofetil (20 mg/kg/day, max- 2 gm/day) were added if total bilirubin was less than 3mg/dL along with steroids. All patients received standard of care management for ascites, variceal bleed, spontaneous bacterial peritonitis (SBP), hepatorenal syndrome, and hepatic encephalopathy according the accepted guidelines [24].

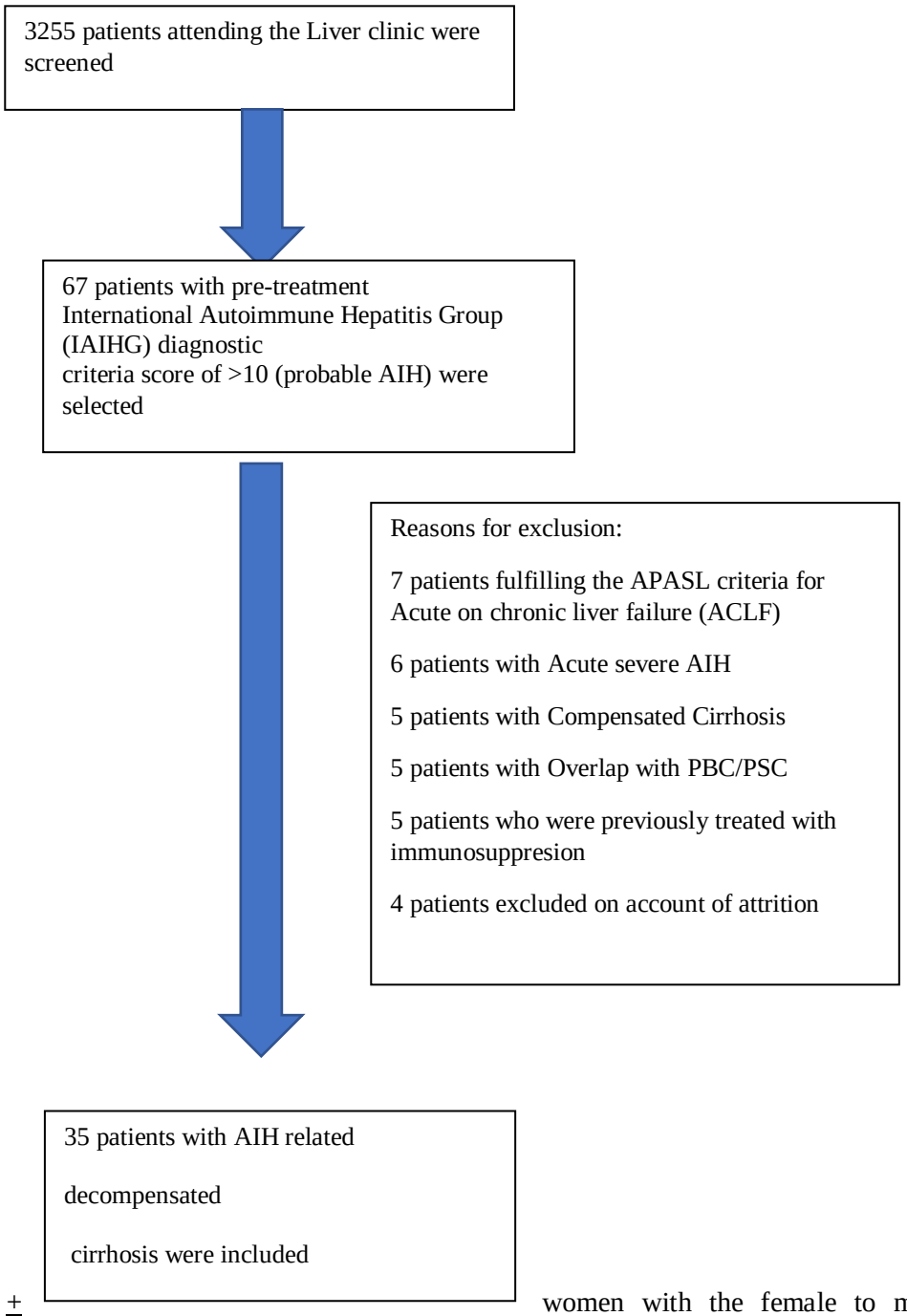
**Follow-up:**

The Child–Turcotte–Pugh (CTP) score and the model for end-stage liver disease (MELD) score were calculated from the baseline parameters and the changes in Child–Turcotte–Pugh (CTP) score (Delta CTP) and MELD score (Delta MELD) were calculated at the end of 1 year of follow up. Serious adverse events, such as sepsis, further decompensation like development of encephalopathy, variceal bleed, AKI and mortality were recorded during follow up.

**Study outcomes:**

The Primary outcome was to study the demographic profile of patients with AIH related decompensated cirrhosis. Secondary outcome were as follows: 1) 1 year transplant-free survival ,2) occurrence of infections and its impact on outcomes and 3) changes in CTP and MELD scores at the end of one year.

## RESULTS




The mean age was 45.4 ± 14.6 years. Nineteen (54.3%) of the patients were women with the female to male ratio being 1.2:1. Ascites was present in 32 (91.4%)

patients, being the most common form of decompensating event at the presentation. Encephalopathy (22.8%) and variceal bleed (20%) were the other forms of decompensation of presentation. More than one form of decompensating event at presentation was seen in 12 (34.2%) patients. Variceal bleed was more commonly seen in males (p-0.017). There was no significant difference in the presence of ascites and encephalopathy between the two sexes (p-0.4 and p-0.3). Twenty-eight (80%) patients were started on prednisolone as per protocol on presentation. Out of this, 5 (14.2%) patients received Prednisolone alone and 23 (65.7%) patients received a combination of Prednisolone and Azathioprine (AZA; n=22) or Mycophenolate mofetil (MMF; n=1). Seven (20%) patients were not started on any of the immunosuppressants. The mean CTP and mean MELD were 9.9 $\pm$ 1.6 and 14 $\pm$ 4.6 respectively at the presentation. Out of 35 patients, 30 survived (85.7%) at the end of one year. The most common cause of death was sepsis. Spontaneous bacterial peritonitis (SBP) was the most common form of infection (17.1%). Seven patients were not started on any medical therapy of which 6 survived (85.7%). Five patients were treated with steroids alone, out of which 4 survived (80%). Whereas, 23 patients who were treated with a combination of steroids and azathioprine or MMF, 20 patients survived (86.9%). Females had a higher survival rate when compared to male patients but the difference was not statistically significant (p-0.096). Thus, there was no significant difference in the survival rates based on the treatment received by the patient of AIH (p -0.99). Of the 32 patients who presented with ascites, 27 patients survived (p-0.4). Seven patients had variceal bleed on admission, of which 6 patients survived at the end of 1 year (p-0.9). Whereas, of the 8 patients having encephalopathy on presentation, only 3 survived at the end of 1 year (p-0.0001) making it a significant factor in the contribution leading to mortality in this cohort of patients. A total of 5 patients succumbed at the end of 1 year of which all the 5 patients had both ascites and encephalopathy whereas only 1 patient had variceal bleed. A total of 13 (37.1%) patients developed infections during the study period. Out of which, 11 patients were on immunosuppressants (39.2 %; n = 28). The rates of infections between those who received immunosuppressants when compared to those who did not receive immunosuppressants was not significant (p– 0.6). The MELD decreased by a mean of 3.12 points from baseline at the end of one year in all the patients. The decrease in MELD at the end of one year in patients who received and in those who did not receive immunosuppressants was 2.96 and 3.30 respectively, however, the change in MELD did not differ depending upon the treatment modality offered (p - 0.9).

## DISCUSSION:

In the present study we studied the demographic data, types of presentation, treatment imparted, response and survival at the end of one year in patients with AIH related decompensated cirrhosis. Patients with AIH related decompensated cirrhosis constitute a subgroup with poor prognosis and a high mortality rate. These subsets of patients pose as a difficult therapeutic challenge even today without liver transplantation. The available treatment guidelines do not give special recommendations regarding the use immunosuppressants in these patients [21,24]. "To give immunosuppressants or not to give?" still remains an unanswered question even today. Ascites was the most common form of decompensating event at presentation in our study and variceal bleed as the presenting event at decompensation was more commonly seen in males. Decompensation in the form encephalopathy at the baseline significantly predicted mortality at the end of one year. Overall survival was 85.7 % at the end of one year with females having a higher survival rate when compared to male patients but the difference was not statistically significant. The survival rates were not significantly different between those who received immunosuppressants when compared to those who did not receive immunosuppressants. The most common cause of death was sepsis with spontaneous bacterial peritonitis being the commonest form of infection. The rates of infections between those who received immunosuppressants when compared to those who did not receive immunosuppressants was not significant. The change in MELD, CTP, bilirubin, albumin, INR and creatinine did not differ depending upon the treatment modality offered. In our study, the presence of encephalopathy at presentation and higher MELD significantly predicted mortality at the end of one year in our study in patients with autoimmune hepatitis related decompensated cirrhosis. Fewer studies in the past have evaluated the outcomes of steroids in patients with autoimmune hepatitis related decompensated cirrhosis. Wang et al. demonstrated the reversal of clinical decompensations in 62% of patients and Amarapurkar et al demonstrated a biochemical remission in 68% of these patients [19,25]. These results are in contrast to our study which shows no mortality benefits in treating autoimmune hepatitis related decompensated cirrhotics with immunosuppressants. The study by Sanchit et al also shows that patients with decompensated cirrhosis and poorer outcomes with corticosteroids [18]. Thus to conclude our study highlights the fact that treatment of AIH related decompensated cirrhosis with immunosuppressants did not improve the overall survival but may in fact increase the risk of infections in this patient subset. This is a unique study as there have been not much data from the Western India regarding the demographic profile and evaluating the role of

immunosuppressants in patients with AIH related decompensated cirrhosis. This is also the first study negating the use of immunosuppressants in patients with AIH related decompensated cirrhosis. All the patients included in our study were histologically confirmed to have autoimmune hepatitis, which was not done in the previously conducted studies on these subset of patients [18,19,25]. In our study, Azathioprine and MMF were used as maintenance agents and none of the patients had any serious adverse effects or had to discontinue these medications. This makes it the first study where in these agents were successfully used in patients with AIH related decompensated cirrhosis. However, larger studies are needed to confirm these findings. The limitations of our study are the retrospective observational nature of data compilation and the relatively small (35) sample size. Another limitation is the absence of a control group of patients who were not treated with immunosuppressants. In our study, the number of patients not treated with immunosuppressants is too small to ideally compare with those who had received immunosuppressants. Further studies are needed to confirm the survival benefits of immunosuppressants in AIH related decompensated cirrhosis. There was a limited period of follow-up duration in our study and hence long-term outcomes in terms of overall survival and relapse of disease cannot be deduced. Our study also highlights an important fact that the use of immunosuppressants in patients with AIH related decompensated cirrhosis is not value and also adds to the risk of infections in this subset of patients. However, further prospective studies are needed in this area to guide us regarding the use of immunosuppressants in these patients.

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**Table 1: Baseline characteristics among patients with autoimmune hepatitis related decompensated cirrhosis**

| **Parameters** | **Number(%)** |
|---|---|
| ***Gender*** <br>• Females | • 19(54.3%) |
| ***Ascites*** <br>• Present | • 32(91.43) |
| ***Variceal bleed*** <br>• Present | • 7(20) |
| ***Encephalopathy*** | |

| | |
|---|---|
| • Present | • 8(22.86) |
| ***No co-morbidities*** | • 15(42.86) |
| ***Comorbidities*** | |
| • Diabetes Mellitus | • 9 (25.71) |
| • Diabetes + Hypertension | • 3(8.57) |
| • Hypertension | • 3(8.57) |
| • HIV | • 2(5.71) |
| • Dyslipidemia | • 1(2.86) |
| • Hypertension | • 1(2.86) |

**Table 2: Baseline laboratory parameters among patients with autoimmune hepatitis related decompensated cirrhosis**

| **Parameters** | **Mean ± SD** |
|---|---|
| **Hemoglobin** | 9.0600±2.2026 |
| **Total leucocyte count** | 5824.2857±2920.8926 |
| **Platelet count** | 106285.7143±54502.8717 |
| **Total bilirubin** | 3.0914±1.3156 |
| **Direct bilirubin** | 1.8057±1.0751 |
| **SGOT** | 122.8571±89.2271 |
| **SGPT** | 84.9143±49.3549 |
| **ALP** | 243.7714±125.0054 |
| **Total Protein** | 6.4686±0.7012 |
| **Serum Albumin** | 2.9114±0.08942 |
| **BUN** | 14.9143±9.4632 |
| **Creatinine** | 1.0114±0.4490 |
| **S. Sodium** | 133.5143±4.4283 |
| **S. Potassium** | 3.9629±0.5542 |

| | |
|---|---|
| **INR** | 1.4934±0.2718 |
| **CTP** | 9.8857±1.6046 |
| **CTP 1** | 5.2286±2.4142 |
| **MELD** | 13.9820±4.5537 |
| **MELD 1** | 8.4571±4.5071 |

**Table 3: Types of infections**

| **Parameters** | **Number(%)** |
|---|---|
| ***Infections*** | |
| • Spontaneous bacterial peritonitis | • 6 (17.1) |
| • Urinary tract infection | • 4(11.4) |
| • Pneumonia | • 2(5.71) |
| • Septicemia | • 1(2.86) |

**Table 4: Treatment modality offered and outcome**

| | |
|---|---|
| ***Treatment modality*** | |
| • Steroids+ Immunomodulators | • 23(65.71) |
| • No immunosuppresants | • 7 (20) |
| • Steroids | • 5(14.29) |
| ***Compliance*** | |
| • Yes | • 32(91.43) |
| • No | • 3(8.57) |
| ***Overall survival*** | 30(85.71) |

**Table 5: Parameters compared and statistical significance**

| **Parameters compared** | **P value** |
|---|---|
| **Survival with treatment modality** | 0.9220 |
| **Delta MELD with treatment modality** | 0.9635 |

| | |
|---|---|
| **Rate of infections with treatment modality** | 0.59 |
| **1st presentation as Ascites and survival** | 0.4661 |
| **1st presentation as encephalopathy and survival** | 0.0001 |
| **1st presentation as variceal bleed and survival** | 0.9999 |

**Abbreviations:**

1. ACLF: Acute-on-chronic liver failure
2. AIH: Autoimmune hepatitis
3. anti LKM1: anti-liver kidney microsomal types I
4. ANA: Anti-nuclear antibodies
5. APASL: Asian pacific association for the study of Liver
6. ASMA: Anti-smooth muscle antibodies
7. AZA: Azathioprine
8. CTP: Child–Turcotte–Pugh (CTP) score
9. IAIHG: International Autoimmune Hepatitis Group
10. INR: International normalised ratio
11. KF: Kayser Fleischer ring
12. MELD: Model for end-stage liver disease
13. MMF: Mycophenolate mofetil
14. PBC: Primary biliary cholangitis
15. PSC: Primary sclerosing cholangitis
16. SBP: Spontaneous bacterial peritonitis