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Assessment of clinical profile of patients with acute on chronic liver failure

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ABSTRACT

Background: Acute-on-chronic liver failure is a clinical syndrome of sudden hepatic decompensation in patients with pre-existing chronic liver disease that is associated with one or more extra-hepatic organ failures and increased mortality.The present study was conducted to assess clinical profile of patients with acute on chronic liver failure.

Materials & Methods: 68 acute on chronic liver failure patients age ranged 18- 58 years of both gendersunderwent estimation of haemogram, liver function tests (LFT), renal function tests (RFT), serum electrolytes (Na/K), viral serology (HIV/HBsAg/Anti-HCV/IgM HAV/IgM HEV), urine routine/microscopic examination and culture, stool routine/microscopic examination and culture, blood culture, ascitic fluid analysis, chest X-ray and ultrasonography abdomen.

Results: out of 68 patients, males were 40 and females were 28. Common clinical features were haematemesisin 45, jaundicein 32, abdominal pain in 51, malena in 14, abdominal distension in 41, breathlessness in 30 and decreased urine output in16 patients. The difference was significant ($P< 0.05$). Common aetiological factors were pallor seen in 15, icterusin 39, fetor hepaticus in 12, pedal oedema in 17, petechiae in 27, asterixis in 30 and splenomegaly in 16 patients. The difference was significant ($P< 0.05$).

Conclusion: Common clinical features were haematemesis, jaundice, abdominal pain, malena, abdominal distension, breathlessness and decreased urine output.

Key words: Chronic liver failure, Haemogram, Liver function tests

INTRODUCTION

Acute-on-chronic liver failure (ACLF) is a clinical syndrome of sudden hepatic decompensation in patients with pre-existing chronic liver disease that is associated with one or more extra-hepatic organ failures and increased mortality.[1] ACLF is characterized by a rapidly deteriorating course in a previously diagnosed or undiagnosed chronic liver disease with a potential for reversibility.[2]

Global literature says most of ACLF in India is caused by infections. With improvement of socio-economic status aetiological cause should be studied for a change in trend. Different factors affect the incidence and prevalence of ACLF in different geographical areas.[3]ACLF occurs as a natural history of chronic liver disease. There is a risk for multiple organ failure and an increased mortality. The Asia-Pacific Association for the Study of the Liver (APASL) found reactivation of hepatitis B and superinfection with hepatitis E virus infection on NAFLD are important causes or ACLF in that region, but a significant proportion of cases continue to be due to alcoholic hepatitis.[4]

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Both the precipitating event and the pre-existing liver disease have geographical disparities. These events could be either liver-related (superimposed viral hepatitis, alcoholic hepatitis, portal vein thrombosis (PVT), drug-induced liver injury (DILI)), or non-liver-related (surgery, infections, trauma).[5] There is no specific precipitating event to be found in about 40% of patients with ACLF as well. ACLF is a dynamic syndrome, which may improve, worsen, or have a mild protracted course allowing us to evaluate for a possible liver transplant. The etiology of the precipitating factor causing ACLF does not alter the prognosis.[6] The present study was conducted to assess clinical profile of patients with acute on chronic liver failure.

MATERIALS & METHODS

The present study was conducted among 68 acute on chronic liver failure patients age ranged 18-58 years of both genders. All were informed regarding the study and their written consent was obtained.

Data related to patients such as name, age, gender etc. was recorded. In all patients, a detailed history and clinical examination was performed. All patients underwent estimation of haemogram, liver function tests (LFT), renal function tests (RFT), serum electrolytes (Na/K), viral serology (HIV/HBsAg/Anti-HCV/IgM HAV/IgM HEV), urine routine/microscopic examination and culture, stool routine/microscopic examination and culture, blood culture, ascitic fluid analysis, chest X-ray and ultrasonography abdomen. Results of the study thus obtained were subjected to statistical analysis P value less than 0.05 was considered significant.

RESULTS

Table I Distribution of patients

Total- 68		
Gender	**Males**	**Females**
Number	40	28

Table I shows that out of 68 patients, males were 40 and females were 28.

Table II Assessment of clinical features

Clinical features	Number	P value
Haematemesis	45	0.05
Jaundice	32	
Abdominal pain	51	
Malena	14	
Abdominal distension	41	
Breathlessness	30	
Decreased urine output	16	

Table II, graph I shows that common clinical features were haematemesis in 45, jaundice in 32, abdominal pain in 51, malena in 14, abdominal distension in 41, breathlessness in 30 and decreased urine output in16 patients. The difference was significant (P< 0.05).

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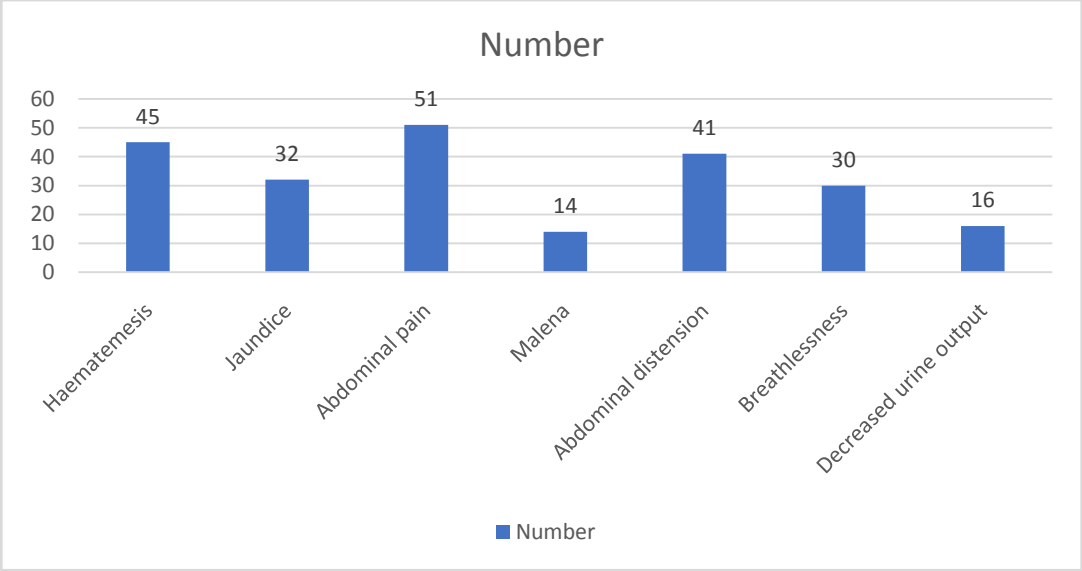

Fig I Assessment of clinical features

Table III Aetiological factors in ACLF patients

Etiology	Number	P value
Pallor	15	0.01
Icterus	39	
Fetor hepaticus	12	
Pedal oedema	17	
Petechiae	27	
Asterixis	30	
Splenomegaly	16	

Table III, graph II shows that common aetiological factors were pallor seen in 15, icterus in 39, fetor hepaticus in 12, pedal oedema in 17, petechiae in 27, asterixis in 30 and splenomegaly in 16 patients. The difference was significant (P< 0.05).

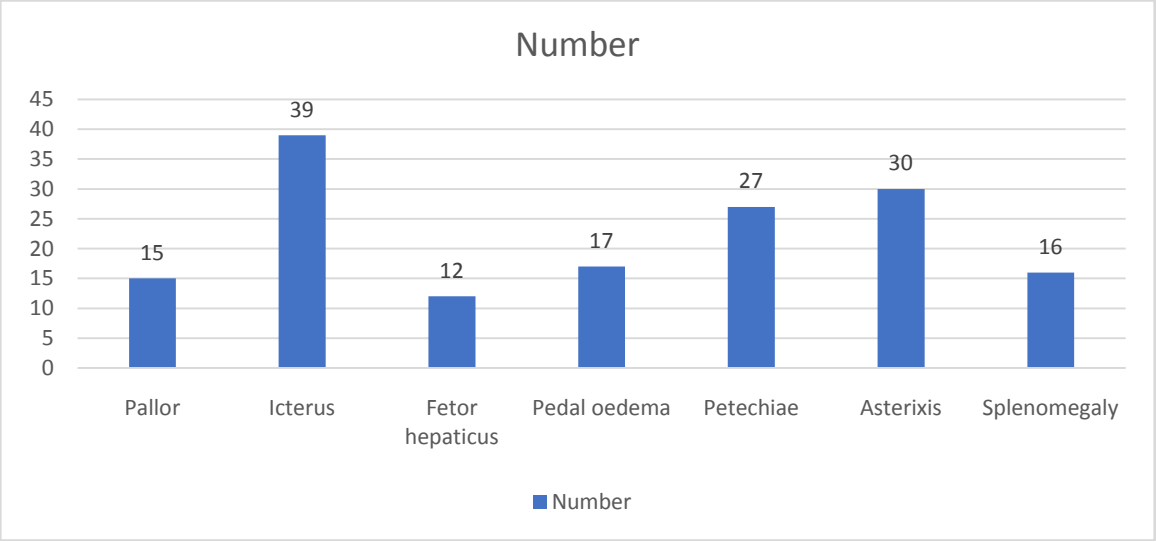

Fig II Aetiological factors in ACLF patients

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DISCUSSION

ACLF occurs as a natural history of chronic liver disease. There is a risk for multiple organ failure and an increased mortality.[7]In non-cirrhotic chronic liver disease the precipitating event is a major hepatic injury, such as reactivation of chronic hepatitis B, superimposed acute hepatitis A or hepatitis E infection in the East, or drug-induced liver injury superimposed upon non-alcoholic fatty liver disease in the West.[8] Alcoholic hepatitis or drug-induced hepatitis are common precipitating events in compensated cirrhosis while infections are common in decompensated cirrhosis.[9] Same study suggested that in acute viral hepatitis, underlying diabetes and CLD are at higher risk for development of liver failure. The study demonstrated that patients with underlying diabetes are prone for Drug-Induced Liver Injury (DILI) and further to liver failure. Thereby they concluded ACLF is a common precipitant in NAFLD patient with superadded DILI Almost 50% of patients of cirrhosis admitted to the hospital have evidence of infection or sepsis and a further 25% develop nosocomial infections with high inpatient hospital mortality.[10]The present study was conducted to assess clinical profile of patients with acute on chronic liver failure.

In present study, out of 68 patients, males were 40 and females were 28. Jha et al[11]describe the clinical profile of ACLF and the effect of dual acute insult on the natural history.Patients with jaundice diagnosed to have ACLF were prospectively enrolled. Patients were evaluated for the clinical presentation, etiology of acute decompensation and underlying chronic liver disease, and inhospital mortality. They compared the clinical profile of patients who had dual acute insult with those of single/unknown insult.Fifty-two patients with ACLF (mean age 38.6 ± 16.7 years; M/F 41:11) were included. Hepatitis virus infection (46.1 %) and bacterial infection (36.5 %) were the most common acute insults. Hepatitis virus infections were the sole acute insult in 34.6 % and associated with another injury in 11.5 %. Bacterial infections were identified as acute insult in 19 patients (sole acute insult in 13). Drugs, autoimmune disease, surgery, malaria, and dengue were other acute injuries identified. The cause of acute decompensation was unknown in 11.5 %. Mortality (66.6 % vs. 51.1 %) was higher in patients with dual insult (n=9) as compared with single/unknown insult (n=43).

We found that common clinical features were haematemesisin 45, jaundice in 32, abdominal pain in 51, malena in 14, abdominal distension in 41, breathlessness in 30 and decreased urine output in16 patients. Singh et al[12] in their study determined the clinical, laboratory and etiological profile of patients with ACLF.Out of 300 chronic liver disease patients, 58 met the criteria of ACLF. Their clinical, laboratory, etiological profile and outcome were studied.The most common etiology of underlying chronic liver disease was alcohol (81%). Most common acute insult in patients with alcoholic liver disease was severe alcoholic hepatitis (54%). Most common (75.8%) type of patients were of decompensated cirrhosis. Remaining 24.2% of patients were not decompensated before presentation. Seven days mortality was 13.7% (8 out of 58). Patients who died had more than 2 organ failure and MELD >25. Two out of 8 patients who died, were not decompensated prior to illness. Fifteen required repeat admission within 1 month.Most common cause of acute insult in ACLF was alcohol which is preventable. Prognosis was worst in patients who were decompensated prior to illness, had organ failure and high MELD score.

We observed that common aetiological factors were pallor seen in 15, icterusin 39, fetor hepaticus in 12, pedal oedema in 17, petechiae in 27, asterixis in 30 and splenomegaly in 16 patients. Yadav et al[13] in their single center prospective observational study included 50 ACLF patients. Out of 50 patients, male 47 (94%) were predominant. Alcohol was most common

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etiology for chronic liver disease, 35 (70%) followed by chronic hepatitis B (CHB), 6 (12%). Active alcoholism was most common precipitating event 33 (66%), followed by sepsis in 15 (30%), CHB flare in 5 (10%), acute hepatitis A in 3 (6%), ATT-DILI in 2 (4%). More than one precipitating event in 14 (28%). Spontaneous bacterial peritonitis was present in 26%, hepatic encephalopathy in 40%, renal dysfunction in 24%, UGI bleed in 18% cases. One month mortality was 20%. Three months mortality was 42%. For 1 month mortality younger age (<39 years), high INR (>3.07) and low hemoglobin (<9.3 g/dL) were statistically significant. For 3 months mortality total bilirubin (>21.65 mg/dL) and low hemoglobin (<9 g/dL) were statistically significant.

CONCLUSION

Authors found thatcommon clinical features were haematemesis, jaundice , abdominal pain, malena, abdominal distension, breathlessness and decreased urine output.

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