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ACUTE ON CHRONIC LIVER FAILURE WITH ALCOHOL WITHDRAWAL SYNDROME: A CASE REPORT

Athira G. H.[1], Ananthan J.[1], Dhanya Dharman*[2], Shaiju S. Dharan[3]

[1]Pharm D. Intern, Department of Pharmacy Practice, Ezhuthachan College of Pharmaceutical Sciences, Thiruvananthapuram.
[2]Associate Professor, Department of Pharmacy Practice, Ezhuthachan College of Pharmaceutical Science, Thiruvananthapuram.
[3]Principal and Head of Department of Pharmacy Practice, Ezhuthachan College of Pharmaceutical Science, Thiruvananthapuram.

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Dhanya Dharman
Associate Professor, Department of Pharmacy Practice, Ezhuthachan College of Pharmaceutical Science, Thiruvananthapuram.

ABSTRACT
Acute on chronic liver failure (ACLF) is a newly recognized clinical entity that describes acute hepatic decompensation in persons with preexisting liver disease. Acute liver failure (ALF) is a well-defined clinical syndrome with associated high mortality. Acute on chronic liver failure is also a rare syndrome with diverse etiology. ACLF can develop in patients with or without liver cirrhosis, where patients with decompensated cirrhosis display a higher risk of short-term mortality. Pathophysiological mechanism include systemic inflammation due to bacterial and fungal and acute hepatic insult with drug, alcohol and viral hepatitis.

KEYWORDS: Acute on chronic liver failure, liver cirrhosis, organ failure, acute decompensation.

INTRODUCTION

Acute on chronic liver failure (ACLF) is a clinical syndrome of sudden hepatic decompensation observed in patients in patients with pre-existing chronic liver disease and associated with one or more extrahepatic organ failures and increased mortality.[1-4] Regardless of the etiology of chronic liver injury, when a clinician follows the natural history of liver injury, the initial uncomplicated chronic liver disease leads to cirrhosis and, later, the decompensation of liver function with ascites, jaundice, portal hypertension with variceal bleeding, and hepatic encephalopathy. The three major feature characteristics of this syndrome occurs in the context of intense systemic inflammation, alcoholic hepatitis and single or multiple organ failure.

The etiology of ACLF would be related to a precipitating event in the context of a pre- existing liver condition. Hepatic causes include alcohol-related injury, drug-induced liver injury, viral hepatitis (A, B, C, D, and E), hypoxic injury, or liver surgeries including transjugular intrahepatic portosystemic shunt (TIPS) placement. Extra-hepatic causes mainly consist of bacterial infections and major surgery. An estimated 40% to 50% of patients are labeled as having an unrecognized precipitating event culminating in ACLF.

Recent trends in morbidity and mortality indicate that patients with acute on chronic liver failure comprise 5% of all hospitalizations for cirrhosis. When compared to the most common causes of hospitalization in the United States, the healthcare burden per patient is much higher in patients with ACLF. The mean cost of hospitalization for patients with ACLF is three times more than the cost of patients hospitalized with cirrhosis, and five times more than the cost of patients hospitalized with congestive heart failure.

The pathophysiology is based on the understanding that an acute precipitating event in a patient with a chronic liver condition that injures hepatocytes leads to the accumulation of a cascade of inflammatory cytokines and results in further hepatic injury in the presence of failure of hepatocyte regeneration. The resulting compromise in immune function and liver decompensation leads to further susceptibility to infections, multi-organ failure, and death.

Systemic inflammation with elevated leukocytosis, cytokines, and chemokines (including IL-6 and IL-8) are observed in patients with ACLF. This is usually absent in cirrhotic patients with ACLF.

Bacteria-induced pathogen-associated molecular patterns

(PAMPS) and virulence factors activate transcription factors are required for encoding cytokines in the inflammatory cascade. Endogenous inducers of inflammation, obtained as a result of denaturation of hepatocytes, called damage-associated molecular patterns (DAMPs), are also responsible for activating inflammatory cascades cooperating with Toll-like receptors (TLRs). This immunopathology is responsible for tissue, organ damage, and, ultimately, organ failure.

CASE PRESENTAION

A 54- year- old male patient admitted under gastroenterology department with complaints of abdominal pain and vomiting. His previous history shows that alcohol withdrawal seizure and alcoholic hepatitis. The patient had medical history of systemic hypertension for 5 years and was on T. TELMIKIND 40 mg and T.PANTOP 40 mg and he was taking lorazepam for past 3 years due to sleep disturbances.

Laboratory results showed a hemoglobin, 11.5 g/dL, blood urea nitrogen (BUN) 25 mg/dL, and serum creatinine 0.6 mg/dL. Liver function test should be abnormal SGOT, SGPT, ALP level should be elevated. During the admission time he was shown to have hyponatremia and hypokalemia .USG abdomen shown that chronic liver disease with cirrhosis, dilated portal vein with splenomegaly, ascites with GB wall edema. Abdominal sonogram showed fatty liver infiltration, mild splenomegaly, mildly distended gallbladder without calculi, and no biliary ductal dilatation.

He was treated with IV antibiotics and hepatoprotective agents and nutritional supports, INJ. THIAMINE 100 mg IV BD, INJ. CEFTRIAXONE 1gm iv BD, INJ. PHYTOMENADIONE 10ml OD, T. THIOTRESS 500mg, T. ADEMETIONINE 400mg, T. UDILIV 300mg, T. BECOSULEZ, SYP. LACTULOSE 25ml, INJ. PANTOPRAZOLE 40mg and other supportive measures were given. Patient was better after two weeks, electrolytes and liver function test was repeated and it was found to be normal. The patient was discharged on stable condition with T.CARVEDILOL 3.125mg OD, T. ADEMETIONINE 400mg BD, T. PENTOXIFYLLINE 400mg TDS, T. URSODEOXYCHOLIC ACID 300mg BD and T.ZOLPIDEM 10mg HS and was advised with repeat USG abdomen and AFP every 6 months for HCC screening.

DISCUSSION

ACLF patients manifest in various forms due to the heterogeneity of this patient population. Severe jaundice, coagulopathy, multi-organ failure with encephalopathy and renal dysfunction, and systemic inflammatory response syndrome are common findings. Proinflammatory cytokines, neutrophil dysfunction and sepsis are believed to play a major role in pathogenesis and prognosis. Elevated leukocyte counts and C-reactive protein have been found to be common as well as evidence of occult or overt infection in ACLF patients. Dysregulated inflammation is considered a critical hallmark and final common pathway of the various insults of ACLF.[5-8] At the time of presentation our patient however, had normal white blood cell count and no evidence of infection. Further research will help define this subgroup of patients.

Acute-on-chronic liver failure (ACLF) is a multifaceted condition with poor treatment options and high short-term mortality. ACLF can develop in patients with or without liver cirrhosis, where patients with decompensated cirrhosis display a higher risk of short-term mortality. Pathophysiological mechanisms include systemic inflammation due to bacterial and fungal infections and acute hepatic insult with drug, alcohol, and viral hepatitis. Cryptogenic factors also contribute to the development of ACLF.

CONCLUSION

The clinical outcome of patients with ACLF gets further complicated by the occurrence of variceal hemorrhage, hepatorenal syndrome, hepatic encephalopathy and systemic immune dysfunction. Acute on chronic liver failure (ACLF) is a serious condition which develops in patients with chronic liver disease (CLD) with compensated and decompensated cirrhosis. ACLF is defines of multi organ failure, and high risk of short-term mortality.

ACLF is a devastating syndrome which defines a subgroup of patients with chronic liver disease who develop organ failure with high mortality. ACLF is a clinically, pathophysiologically, and prognostic ally distinct entity. In ACLF, deranged host response to precipitating injury plays a pivotal pathophysiological role, such as SIRS. The degree of background immune paralysis and severity of organ failure determine the outcome of this syndrome. However, there are areas of uncertainty in defining ACLF, such as heterogeneity of ACLF, ambiguity in qualifying underlying liver disease, argument for infection or sepsis as a precipitating event, etc. Treatment strategies are limited to organ support but better understanding of the pathophysiology of ACLF is likely to lead to discovery of novel biomarkers and therapeutic strategies in the future.

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DECLARATION OF PATIENT CONSENT

Informed consent was obtained from the patient and his physician.

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CONFLICTS OF INTEREST
There is no conflicts of interest.

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