Alcoholic Liver Disease and COVID-19 Pneumonia: A Case Series

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Abstract

The novel coronavirus 2019 (COVID-19) was reported by the World Health Organization in December 2019, and since then it has progressed into a worldwide pandemic, causing significant morbidity and mortality. Gastrointestinal symptoms of COVID-19 and elevated liver chemistries are seen in up to 50% of infected patients. Recent reports have suggested a high mortality rate for COVID-19 in patients with pre-existing liver disease, having an associated mortality of 39.8%. Alcoholic liver disease is a significant cause of morbidity and mortality in New Mexico (USA), and we report here the clinical course and characteristics of three cases of patients with alcoholic cirrhosis who were admitted to our hospital with COVID-19.

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Introduction

The first case of novel coronavirus 2019 (COVID-19) in the USA was reported on January 20, 2020.1 The USA now has the largest number of confirmed cases in the world; as of July 15, 2020, there were 3,448,625 cases of coronavirus diagnosed in the country, with 136,699 deaths.2 While liver abnormalities have been reported in patients with coronavirus, most of the related biomarker elevations are mild, with a predominantly hepatocellular elevation ranging from 14–53% and slightly elevated bilirubin in 14–53% of patients.3,4

There is limited evidence on the outcomes of COVID-19 in patients with alcoholic liver disease. A meta-analysis showed 3% prevalence of chronic liver disease in patients with COVID-19.5 Bangash et al.6 considered that abnormal liver biochemistries do not necessarily arise from the liver alone; in fact, several other reasons, such as COVID-19-induced myositis as well as collateral liver damage from induction of a dysregulated immune response and drug-related liver injury, are more likely to explain deranged liver biochemistries in COVID-19.

In data collected from seven Chinese studies, mortality occurred in only 0–2% of patients with chronic liver disease; however, the nature and severity of the liver disease were unknown.3 The SECURE-Cirrhosis registry and COVID-HEP reported on 334 patients with cirrhosis, out of which 102 (31%) patients had alcoholic cirrhosis; however, outcomes were not defined by etiology of cirrhosis.7 Our hospital is a tertiary care center in a state with disproportionately excessive alcohol use and alcohol-related liver disease deaths (22 per 100,000 population)8 compared with other USA states. In this case series, we describe the clinical presentation, management, and outcomes of patients with alcoholic cirrhosis and COVID-19.

Case report

A retrospective chart search was performed (under institutional IRB 20-186) for patients with a past medical history of alcoholic cirrhosis and diagnosis of COVID-19. The patients had been consecutively admitted to the University of New Mexico Hospital from December 1, 2019, to April 23, 2020. Informed consent was waived as part of the institutional IRB. Only patients with a laboratory-confirmed (reverse transcription-PCR-positive) diagnosis for COVID-19 were included.

Information was collected regarding comorbidities, social history, vital signs, demographics, clinical characteristics, symptomatology, alcohol use, lab results, imaging characteristics, and clinical management details. The authors manually analyzed the data.

Case 1

A 32-year-old male with a past medical history of alcoholic cirrhosis, and class I obesity [body mass index (BMI) of 30.5], presented intubated and sedated after he was found unresponsive at home. Baseline model for end-stage liver disease (MELD) labs were not available; however, per his family, the patient was a heavy drinker and was actively drinking alcohol up to 2 days before.

On evaluation, the patient was in multiorgan failure, attributed to septic shock due to COVID pneumonia. He required significant vasopressor support, acute hypoxic respiratory failure due to acute respiratory distress syndrome (ARDS), requiring mechanical ventilation, and acute kidney injury (AKI) requiring continuous renal replacement therapy (CRRT). His physical exam was significant for bruising on his...
extremities, diffuse anasarca, and jaundice. Liver biochemistries on presentation were an aspartate aminotransferase (AST) level of 276 U/L, alanine aminotransferase (ALT) level of 60 U/L, and alkaline phosphatase (ALP) level of 269 U/L. Total bilirubin was elevated to 15 mg/dL and direct bilirubin to 10.4 mg/dL. MELD-sodium (MELD-Na) on presentation was 36, and Child-Pugh class was C. Coagulation profile was deranged as well, with a prothrombin time (PT) 29s and international normalized ratio (INR) of 2.4. D-dimer was elevated at 6951 mg/mL, and platelet count was 92,000/mL.

He was also severely hyponatremic, with serum sodium of 116 mg/dL, and anuric, with serum creatinine of 4.81 mg/dL. Lactate dehydrogenase was elevated to 609 U/L. A nasopharyngeal swab returned positive for COVID-19. Serologies for hepatitis A, B, and C were performed and were negative.

On day 3 of hospitalization, the patient’s cardiopulmonary status began to improve, with decreased fraction of inspired oxygen (FiO2) and positive end-expiratory pressure (PEEP) requirements. Unfortunately, he continued to be oliguric and in hepatic failure, with an AST of 317 U/L, ALT of 110 U/L, bilirubin of 17.8 mg/dL, and INR of 1.64 (Table 1). Also, he experienced an acute gastrointestinal bleed, with a drop in hemoglobin from 12.4 g/dL on admission to 7 g/dL, with coffee-ground contents in his nasogastric tube on suction and melena requiring 3 U of packed red blood cells as well as vitamin K administration. Octreotide and proton pump inhibitor infusions were started, and on day 2, overt bleeding had stopped, and no endoscopic intervention was performed. He also received a 10-day course of azithromycin and hydroxychloroquine, according to an ongoing hospital-based clinical trial (unpublished data). A computed tomography scan of his abdomen showed extensive ascites and a nodular-appearing liver as well as dilated bowel loops suggestive of shock bowel. Unfortunately, his septic shock continued to worsen with leukocytosis, poor oxygenation, and elevated lactate levels, and care was withdrawn per family wishes on day 13.

### Case 2

A 34-year-old male with a known history of alcoholic cirrhosis, Class II obesity (BMI of 35) and active alcohol use was brought into the hospital intubated and sedated, after...
A 44-year-old man with Class III obesity (BMI of 41.7) and alcoholic cirrhosis complicated by esophageal varices, as well as a history of spontaneous bacterial peritonitis and pulmonary hypertension was transferred to our hospital for acute hypoxemic respiratory failure secondary to COVID-19, requiring intubation and vasopressor support. His physical exam was significant for diffuse anasarca and jaundice.

Liver biochemistries on admission showed an AST of 73 U/L, ALT of 19 U/L, ALP of 147 U/L, and bilirubin of 1.71 mg/dL. Platelet count was low at 88,000/μL. He was started on CRRT due to oliguric AKI, azithromycin, and hydroxychloroquine were administered per an ongoing clinical trial (unpublished data). While his liver biochemistries remained stable, he developed severe encephalopathy, and his cardiopulmonary status continued to worsen. Due to his poor prognosis and lack of improvement, his family decided to withdraw care, and he died 6 days after admission.

**Discussion**

Our cases represent the only patients presenting with alcoholic cirrhosis and COVID-19 to our tertiary hospital through the study period, representing a 100% mortality rate. The severity of liver disease at baseline was not known in cases 1 and 2, as they were transferred from an outside facility with no available medical records; however, case 3 had decompensated cirrhosis with varices as well as a history of spontaneous bacterial peritonitis. All patients were obese, with an average BMI of 35.7 (BMI >30 in all) and were actively drinking before presentation to a peripheral hospital with abdominal pain and shortness of breath. His physical exam was unremarkable. He had severely deranged liver biochemistries, with an AST of 4969 U/L, ALT of 6350 U/L, ALP of 160 U/L, and bilirubin of 5.1 mg/dL. MELD-Na on presentation was 32 and Child-Pugh class was C. Creatinine kinase (CK) was elevated to 2095 U/L and lactate was elevated to 13 mEq/dL, suggesting ischemic hepatitis in the setting of shock. Platelet count was low at 54,000/μL, and his coagulation panel was abnormal, with an INR of 1.4. A nasopharyngeal swab was positive for COVID-19. Tests for markers of acute and chronic hepatitis A, B and C were negative. During the day, his hemodynamic instability worsened, requiring the addition of a fourth pressor and stress-dose hydrocortisone. CRRT was initiated, and inhaled nitric oxide was administered due to persistent hypoxemia. Despite all interventions, he continued to deteriorate and died of septic shock, presumed due to COVID-19 pneumonia.

**Case 3**

A 44-year-old man with Class III obesity (BMI of 41.7) and alcoholic cirrhosis complicated by esophageal varices, as well as a history of spontaneous bacterial peritonitis and pulmonary hypertension was transferred to our hospital for acute hypoxemic respiratory failure secondary to COVID-19, requiring intubation and vasopressor support. His physical exam was significant for diffuse anasarca and jaundice.

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