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Critical Care

SESSION TITLE: Medical Student/Resident Critical Care Posters

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COVID-19-INDUCED LIVER INJURY: A CLINICAL DISTRACTION?

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INTRODUCTION: Patients with novel coronavirus disease 2019 (COVID-19) experience various degrees of liver function abnormalities. Liver injury can be multifactorial and heterogeneous and often requires extensive workup and close follow up (1).

CASE PRESENTATION: A 51-year-old male with a history of hypertension and diabetes was admitted with fever and breathlessness secondary to COVID-19 and started on hydroxychloroquine (HCQ) and azithromycin. His routine blood work, including a complete metabolic panel, was unremarkable. He was intubated for progressive hypoxia, and given the elevated ferritin-5088 ng/dL and D-Dimer >2500mg/L, he was started on high-dose heparin infusion for anticoagulation. His ferritin and D-Dimer continued to uptrend, and on day 10, he was noted to have a total bilirubin of 8.4 mg/dL. Highest peak values noted, aspartate aminotransferase-229, alanine aminotransferase-167, alkaline phosphatase-162, total bilirubin-22, and direct bilirubin-15.5. Tests for hepatitis A, B, C, HSV, EBV, hemolysis workup, and imaging studies were unremarkable. After a week, the liver enzymes and bilirubin down trended and returned to baseline spontaneously. No clear etiology was ascertained and was presumed multifactorial likely from viral hepatitis, sepsis or drug-induced cholestasis. His hospital course was also complicated by acute renal failure requiring renal replacement therapy, coagulopathy, and encephalopathy which coincided with liver injury trailing behind the peak of inflammatory markers. He underwent tracheostomy and percutaneous endoscopic gastrostomy tube placement prior to discharge after 7 weeks.

DISCUSSION: Emerging data support the hypothesis that liver injury in COVID-19 is often the result of SARS-CoV-2 directly binding to ACE2+ cholangiocytes, leading to cholangiohepatitis. In addition, cytokine storm may further exacerbate the hepatic injury in COVID-19 (2). Although our patient lacked any known risk factors for liver injury and preexisting liver disease, he developed a significant hyperbilirubinemia and transaminitis without sequelae. Hepatic congestion in ventilated patients, shock liver and particularly, drug-induced liver injury (DILI) remains an important consideration in COVID-19 patients. Initiating antiviral therapy and curtailing cytokine dysregulation at an early stage could be beneficial to curb the disease progression (3).

CONCLUSIONS: COVID-19 induced viral hepatitis is now being increasingly identified as a self-resolving complication and the physician should be mindful of it and in the right setting, it may only be a clinical distraction. We should be cognizant of other potential causes of liver injury in COVID-19 patients like concurrent infection, sepsis-induced and DILI.

Reference #1: Alqahtani SA, Schattenberg JM. Liver injury in COVID-19: The current evidence. *United European Gastroenterology Journal*. 2020 Jun;8(5):509-19.

Reference #2: Yang, Z, Xu, M, Yi, JQ, et al. Clinical characteristics and mechanism of liver damage in patients with severe acute respiratory syndrome. *Hepatobiliary Pancreat Dis Int* 2005; 4: 60-63.

Reference #3: Bangash MN, Patel J, Parekh D. COVID-19 and the liver: little cause for concern. *The Lancet Gastroenterology & Hepatology*. 2020 Jun 1;5(6):529-30.

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