Acute Liver Failure in an Incidentally COVID-19 Positive Patient with no Respiratory Symptoms

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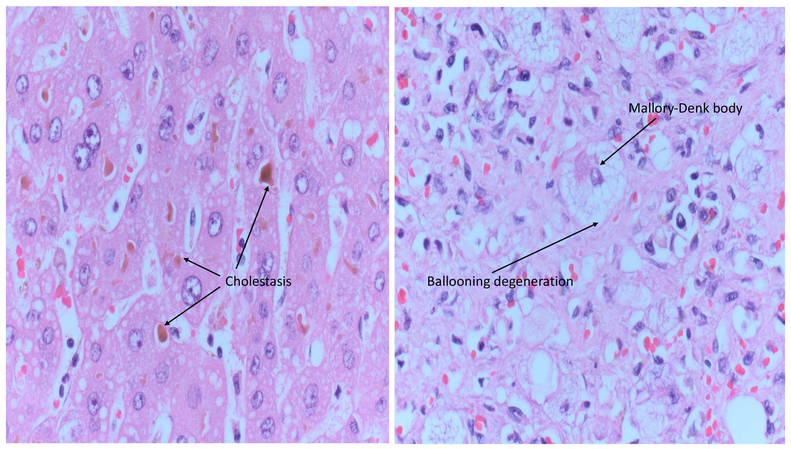
Introduction

- Given that COVID-19 uses angiotensin converting enzyme 2 as its primary viral entry receptor; which is present on multiple organs including the liver; it has the capacity to cause severe systemic disease.(1-4)
- Several studies have documented elevations in liver associated enzymes (LAE), in patients with COVID-19 ranging from 2 to 50 % above baseline. Currently, it is not entirely clear whether LAE elevations can be used for prognostication purposes.(5-7)
- Here we present a case of COVID-19 induced fulminant liver failure in an otherwise asymptomatic non-hypoxemic patient.

Figure1. (left) Liver biopsy at the time of autopsy showing cholestasis and mild mixed steatohepatitis
Figure 2. (right) Liver biopsy at the time of autopsy showing ballooning degeneration and Mallory Denck bodies



Case

- A 50 year-old male with no significant past medical history who had recently recovered from a mild COVID-19 infection ten days prior presents with symptoms of fatigue, abdominal pain, nausea and vomiting for three days. Physical exam revealed diffuse abdominal tenderness and scleral icterus. Patient denied recent travel, alcohol use, contact with animals, multiple sexual partners, drug/medication use.
- Lab findings were significant for elevation of aspartate transaminase, alanine transaminase, total bilirubin and INR of 4985 IU, 9895 IU, 11 mg/dl and >9 respectively. CT abdomen revealed periportal edema with perihepatic free fluid. In conjunction with marked hepatocellular transaminitis, these findings were concerning for acute hepatitis. However, viral panels including Hepatitis A, B, C, D, E, EBV, CMV and HSV were negative. Drug screens, autoimmune panel, ceruloplasmin level and fungal panels were also negative.
- Over the next 3 days patient developed lactic acidosis, shock, hepatic encephalopathy, liver and kidney failure that necessitated ICU admission. Deemed to be too unstable for a liver transplant, despite aggressive supportive measures, the patient died on the 5th day of hospital stay.
- A post-mortem evaluation of hepatic tissue revealed extensive cholestasis, mild mixed steatohepatitis, Mallory bodies, periportal fibrosis, ballooning hepatocytes and lymphocytic infiltrates throughout the parenchyma. (Figure 1 and 2)

Discussion

- Although COVID-19 infection presenting with hypoxemia have shown to present with elevated LAEs in the past, very few cases of fulminant liver failure in an otherwise non-hypoxemic patient have been documented.
- In the setting of no other explanation for acute liver failure, it could be hypothesized that COVID-19 induced viral hepatitis or hypercoagulability leading to a Budd Chiari-like syndrome could have potentiated liver failure in this patient.(8-9)

References

1. Huang C, Wang Y, Li X, et al. Clinical features of patients infected with 2019 novel coronavirus in Wuhan. China. Lancet. 2020;395(10223):497-506.
2. Wang D, Hu B, Hu C, et al. Clinical characteristics of 138 hospitalized patients with 2019 novel coronavirus–infected pneumonia in Wuhan, China. JAMA. 2020;323(11):1061.
3. Mehta P, McAuley DF, Brown M, et al. COVID-19: consider cytokine storm syndromes and immunosuppression. Lancet. 2020;395(10229):1033-1034.
4. Wang F, Wang H, Fan J, Zhang Y, Wang H, Zhao Q. Pancreatic injury patterns in patients with COVID-19 pneumonia. Gastroenterology. 2020.
5. Guan W-J, Ni Z-Y, Hu YU, et al. Clinical characteristics of coronavirus disease 2019 in China. N Engl J Med. 2020;382:1708-1720.
6. Jin XI, Lian J-S, Hu J-H, et al. Epidemiological, clinical and virological characteristics of 74 cases of coronavirus-infected disease 2019 (COVID-19) with gastrointestinal symptoms. Gut. 2020;69:1002-1009.
7. Fan Z, Chen L, Li J, Cheng X, Yang J, Tian C, et al. Clinical features of COVID-19 related liver damage. Clin Gastroenterol Hepatol. 2020.
8. Mazzeffi MA, Chow JH, Tanaka K. COVID-19 Associated Hypercoagulability: Manifestations, Mechanisms, and Management. Shock. 2021;55(4):465-471.
9. Pearson JD. Endothelial cell function and thrombosis. Baillieres Best Pract Res Clin Haematol 12 (3):329–341, 1999.