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A case of COVID-19 with concomitant infection with hepatitis A

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Dear Editor,

Symptoms of COVID-19 ranging from mild to severe and pulmonary manifestations are the most common. However, liver injury is not rare (1) as there might be a reciprocal influence between COVID-19 and hepatic disease (2). While high levels of liver enzymes are associated with an increased prevalence of severe complications, the search for other etiologies of hepatic disease should be not be ignored (3). We report a case of COVID-19 that presented with acute fulminant hepatitis A (HAV) without a previous history.

Case report

A 21-year-old male with history of contact with a COVID-19^{+} patient presented to the Emergency Department with chills, high-grade fever, abdominal pain, vomiting, diarrhea, myalgia, icterus and sore throat without a cough, dyspnea, shortness of breath and chest pain of four days duration. He had a history of swimming in dirty water two-weeks previously. Past

medical history was non-significant. On examination, he was sleepy and had tachycardia, scleral icterus, jaundice, right upper quadrant tenderness and encephalopathy grade I. Lung computed tomography (CT) scan and abdominal ultrasonography were normal. A COVID-19-RT-PCT-swab test was positive. Laboratory findings were suggestive of significant liver dysfunction (Table 1). Serologic tests were reactive for anti-hepatitis A virus (HAV) Ig (reactive IgM and non-reactive IgG), suggesting acute HAV. HBsAb was positive due to previous vaccination. The patient was admitted to the Intensive Care Unit (ICU) and received supportive standard care and lactulose therapy for hepatic encephalopathy with serial monitoring of hepatic function panel. The patient's condition and liver profile improved significantly and he was discharged after ten days.

Discussion

Fulminant HAV is exceptionally rare, especially in persons with no underlying disease. However, it is possible that superimposed COVID-19 infection in patients with mild liver disease increases the risk of a worse outcome and even fulminant hepatitis. Although the mechanism is unclear, immune overriding in addition to cytotoxic effects could be responsible (4,5). Clinicians should have a high level of suspicion in COVID-19^{+} patient and perform close monitoring of liver functions and mental status.

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Table 1. Emergency Department laboratory evaluation

Parameters	*Parameters*
WBC: 5,600 cells/mm^3 (N: 4,000-10,000)	Fasting blood sugar: 84 mg/dl (N: 70-99)
Hemoglobin: 17 g/dl (N: 14-18)	Creatinine: 0.3 mg/dl (N: 0.7-1.4)
Platelet count: 124,000 cells/mm^3 (N: 150,000-450,000 K/µl)	Urine analysis: Protein: +1 Bilirubin:+2
Neutrophil: 56 %	K: 5 mg/l (N: 3.6-5.2)
Lymphocyte: 26 %	AST: 4,480 IU/l (N: < 38)
Monocyte: 12 %	ALT: 6,655 IU/l (N: < 41)
Eosinophil: 5 %	ALP: 285 IU/l (N: < 290)
Basophil: 1 %	Albumin: 4.19 g/dl (N: 3.5-5.2)
LDH: 2,375 IU/l (N: < 480)	Direct bilirubin: 6.1 mg/dl (N: < 0.3)
Total bilirubin: 85.5 mg/dl (N: < 1.2)	CRP titer: 10 mg/l (N: < 15)
Amylase: 62 IU/l (N: < 90)	ESR 1 h: 23 mm/h (N: 0-15)
Na: 137 Meq/l (N: 135-145)	Immunoglobulin level: normal range
Serological tests: Hepatitis C: negative Hepatitis E: negative HIV: negative Cytomegalovirus: negative Herpes simplex: negative Brucellosis: negative	Autoimmune hepatitis assessment: Serum protein electrophoresis: negative Antinuclear antibody: negative Anti-smooth muscle antibody: negative
Wilson disease assessment:	International normalized ratio: 2.69 (N: 1-1.2)

Serum ceruloplasmin: negative	
Blood cultures for bacteria and fungi: negative	Partial thromboplastin time: 44 (N:31-41)