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A case of COVID 19 with concomitant infection with hepatitis A

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Symptoms of COVID-19 range from mild to severe with pulmonary manifestations being predominate, however, liver injury is not rare (1). There might be a reciprocal influence between COVID-19 and hepatic disease (2). While high levels of liver enzyme is associated with an increased prevalence of severe complications, search for other etiologies of hepatic disease should be not be ignored (3). We report a case of COVID-19 presented with acute fulminant hepatitis A (HAV) without previous history.

A 21-years-old man with history of contact with COVID-19^{+} patient presented to emergency department with chills, high grade fever, abdominal pain, vomiting, diarrhea, myalgia, icterus and sore throat without cough, dyspnea, shortness of breath and chest pain since 4 days ago. He had history of swimming in dirty water 2-weeks ago. 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 CT scan and abdominal ultrasonography were normal. COVID-19-RT-PCT-swab test was positive. Laboratory findings were suggestive of significant liver dysfunction (table 1).

Serologic tests were reactive for anti-HAV Ig (reactive IgM and non-reactive IgG) suggesting acute HAV. HBsAb was positive due to previous vaccination. Patient was admitted in ICU received supportive standard care, lactulose therapy for hepatic encephalopathy with serial monitoring of hepatic function panel. Patient's condition and liver profile was improved significantly and discharged after 10-days.

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 worsen outcomes and even fulminant hepatitis. Although mechanism is unclear but 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: 5600 cells/mm^3 (N: 4000–10000)	Fasting blood sugar: 84 mg/dl (N: 70-99)
Hemoglobin: 17 g/dl (N: 14-18)	Creatinin: 0.3mg/dl (N: 0.7-1.4)
Platelet count: 124000 cells/mm^3 (N: 150000–450000 K/µl	Urine analysis: protein: +1 bilirubin:+2
Neutrophil: 56%,	K: 5mg/L (N: 3.6-5.2)
Lymphocyte: 26%	AST: 4480 IU/L (N: <38)
Monocyte: 12%,	ALT: 6655 IU/L (N: <41)
Eosinophil: 5%	Alk phosphatase: 285 IU/L (N: <290)
Basophil: 1%	Albumin: 4.19 g/dl (N: 3.5–5.2)
LDH: 2375 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 1h: 23 mm/hr (N: 0-15)
Na: 137 Meq/l (N: 135-145)	Immunoglobulin level: normal range
Serological tests for 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 serum ceruloplasmin: negative	International normalised ratio: 2.69 (N: 1-1.2).
Blood cultures for bacteria and fungi: negative	Partial thromboplastin time: 44 (N:31-41)

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