Acute Liver Failure in a Pediatrician with COVID-19: a Case Report

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Abstract

Here, we report a case of acute liver failure and a drastic increase of liver enzyme in a pediatrician with COVID-19 infection without a history of preexisting liver disease. Unfortunately, the patient passed away several days after intensive care unit (ICU) admission.

Introduction

In December 2019, pneumonia cases with clinical signs and symptoms that closely resemble viral pneumonia were reported in Wuhan, Hubei, China (1). Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), the newly emerging coronavirus, was termed by the World Health Organization (WHO) shortly after the initial instances of lower respiratory tract infection, and the illness that resulted was called coronavirus disease 2019 (COVID-19) (2,3). In the twenty-first century, three viral epidemics have been linked to Coronaviruses (CoVs): SARS, the Middle East respiratory syndrome (MERS), and COVID-19 (4).

There is ample evidence of liver failure in COVID-19 patients (5–9). The presence of increased liver enzymes in SARS-CoV-2 patients was discovered in 75 of 148 patients (10). Aspartate aminotransferase (AST) increased in 62 percent of intensive care unit (ICU) patients compared to 25 percent of patients who did not receive ICU treatment, suggesting that more severe illness is associated with deterioration of liver enzymes in this population (1). A retrospective cohort study has indicated that acute liver damage is frequent in SARS-CoV-2 positive patients, although usually mild. However, a severe disease course should be expected in the 6.4 percent of individuals with severe liver damage. 67% of patients who tested positive for SARS-COV-2 had higher ALT levels than those who tested negative (11). Here, we report a case of acute liver failure in a pediatrician with COVID-19 infection without a history of preexisting liver disease. Unfortunately, the patient passed away several days after ICU admission. We desire to highlight the knowledge that requires further studies related to liver failure in COVID-19 patients.

Case presentation

On July 12, 2020, a 56-year-old man with fever, severe shortness of breath, cough, and A^+ blood group was admitted to M-ICU at the Ghaem Hospital in Karaj, Iran. He was a pediatrician. A polymerase chain reaction (PCR) test had been performed before he was admitted. The PCR findings indicated that the patient was positive in terms of the presence of SARS-COV-2 at the 24.5 cycle threshold (CT) value. The patient had a history of high blood pressure and had consumed the Valsartan tablet 80 mg. Generally, his medication regimen had included Hydroxychloroquine sulfate, Lopinavir/Ritonavir, Remdesivir, Reci-Gen (interferon beta-1a), Naproxen, Dexamethasone, Convalescent plasma, and Albumin during the period of hospitalization. On the first day of admission, primary laboratory findings revealed the elevated level of white blood cells (WBCs, 20900/microliter) with a high count of neutrophil (91% of WBCs), impaired liver

function; (SGPT or ALT, 127 U/L), acute inflammation; (ESR, 30 mm/hr, not shown in Table 1), negative D-Dimer, and increased fibrinogen (403 mg/dL, not shown in Table 1). On the 2nd post-admission day, lactate dehydrogenase (LDH, 477 U/L, not shown in Table 1) test was performed. Also, a drastic decrease was seen in WBCs count. The enzyme-linked immunosorbent assay results (ELISA) indicated that both SARS-COV2 IgM and SARS-COV2 IgG were negative. Renal function was normal (Table 1). On the next day, the level of total bilirubin was upper than the reference range. Furthermore, the coagulation system was normal. On this day, the patient received fresh frozen plasma (FFP). On the 5th post-admission day, the patient's partial pressure of oxygen (PO_2) and oxygen saturation (SO_2) were lower than normal, and his pulmonary capacity had reduced. One day later, the result of the hepatitis B surface antigen (HBsAg) test was reported as non-reactive or negative. Explicitly, the level of PO_2 reached under 40 mmHg. Meanwhile, renal function was disrupted. A portable chest X-ray (CXR) revealed that the image size of the heart and mediastinum was normal. Moreover, the ground-glass opacity (GGO) was seen in peripheral areas of both lung sides, especially the basal zone. There were no manifestations in the bony thorax. On the 7th post-admission day and the next day, the patient received Morphine Sulfate 10 mg/ml solution by injection each day. Concerning the result of the CXR, the diffused GGO in hemithorax was proved, and pleural effusion. Finally, the patient passed away due to respiratory failure, impaired liver function; (SGPT/ALT, 2500 U/L, SGOT/AST, 4200 U/L), and renal dysfunction. The GGO was apparent in peripheral areas of both lung sides, and lateral sinuses were closed. Drastically, the patient's WBCs count had increased on this day (Table 1).

Discussion

Liver function affected by SARS-COV-2 (9,10). However, there are currently few investigations on the pathogenesis of liver damage in patients with COVID-19. The liver biochemistry abnormalities in COVID-19 patients are moderate (1-2 times the upper limit of normal) increases of serum ALT and AST values, observed in an estimated 29-39% and 38-63% of patients, respectively (12–14). There is no consensus on the association between liver enzyme level elevations and mortality in COVID-19 patients. In this regard, some studies have reported that there is no apparent association (15,16).

On the contrary, several studies have revealed that elevations in the AST and ALT levels that are more than five times the upper limit of normal are linked with an increased risk of mortality (11,17–19). Here, our patient on day 11 had a higher level of AST (105 times the upper limit of normal) and ALT (more than 62 times the upper limit of normal). A tentative explanation is that patients might have a robust immune response. Another reason is that aggressive therapies and anti-viral medication may lead to liver damage (20). It is noteworthy that the possibility of drug-induced liver damage due to hepatotoxicity is associated with drugs used in the treatment of COVID-19 such as lopinavir, ritonavir, and hydroxychloroquine should be taken into consideration by clinicians (6).

It is crucial to develop effective treatment regimens for COVID-19 patients with the fewest side effect on the liver. Further observations will be required to understand the hepatic SARS-CoV-2 infection comprehensively. There is a salient question in this regard. Is the drastic increase in serum AST and ALT caused by hepatic SARS-CoV-2 infection?

Consent for Publication

Written informed consent was obtained from the patient's next of kin to publish this case report and any accompanying images.

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Table 1. Laboratory Data.

Reference range	Day1(Men)	Day2	Day3	Day4	Day5	Day6	Day7	Day8	Day9	Day10	Day11	
ΩBˆ (μΛ)	4000-11000	20900	13600	11000	14000	12300	9700	10500	9800	13800		2
Neutrophil count	55-70 (%)	91	94	92	93	95	92	96	97	93		9
SGPT (U/L)	Up to 41	127		128	90	73	23	50	62	40	24	2
SGOT (U/L)	Up to 37	33		56	29	35	64	23	27	33	32	4
ALP (U/L)	80-306	186		317	256	220	201	180	165	173	164	2
PT (Sec)				14.7			15.5	12	13.9			
PTT (Sec)	30-40			39			37	30	36			
Total bilirubin (mg/dL)	0.1-1.2	0.8		2.1			0.9	0.7				
Direct bilirubin (mg/dL)		0.3		0.6			0.3	0.2				
Ferritin (ng/mL)	30-400						>2000					
pO2	30-50					50.8	38.1			80.6	49.0	2
pCO2	40-52					42.4	40.9			24.2	10.8	7
Oxygen saturation	60-85					87.8	72.9			96.8	89.0	3
HCO3-	22-28					31.8	26.4			17.6	8.2	2
SARS-COV2 IgM	N: <0.9		0.04- N									
	BL: 0.9-1.1											
	P: >1.1											
SARS-COV2 IgG	N: <0.9		0.06- N									
	BL: 0.9-1.1											
	P: >1.1											
BUN (mg/dL)	7-24	25	17	18	25	26	31	29	26	24	24	5
Cr (mg/dL)	0.7-1.4	1.3	1.0	0.8	0.9	0.9	0.9	1.0	1.0	0.8	0.8	2
Albumin (g/dL)	3.5-5			3.6			3.4				3.1	
Phos. (mg/dL)	2.6-4.5										2.2	8
D-Dimer (ng/mL)	N: <500	N					N					

Reference range (Men)	Day1	Day2	Day3	Day4	Day5	Day6	Day7	Day8	Day9	Day10	Day11	
HBs Antigen	NR: <0.9						0.09- NR					
	BL: 0.9-1.0											
	R: >1.0											
Troponin HS (ng/L)	N: <19											3
	S: 19-100											
	P: >100											
CPK (U/L)	24-195											1
CPK-MB (U/L)	6%-25% of Total CK											5
PCR	Sensitivity: 65%	P- CT:24.5	P- CT:24.5	P- CT:24.5	P- CT:24.5	P- CT:24.5	P- CT:24.5	P- CT:24.5	P- CT:24.5	P- CT:24.5	P- CT:24.5	P

Abbreviations: N=Negative; P=Positive; BL= Border line; CT=Cycle threshold; NR=none reactive; R=Reactive; S=Suspicious.