Nawghare et al. Scr Med 2022 Dec;53(4):353-6. DOI:10.5937/scriptamed53-41392

CASE REPORT

A Case of COVID-19 Presenting as Acute Liver Failure

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Abstract

Although children with COVID-19 make up a small proportion of patients and have milder symptoms than adults, liver damage is a well-documented side effect of COVID-19 infection. Most liver damage caused by COVID-19 is modest. In this report, a case of a 6-year-old child who was hospitalised to a paediatric intensive care unit (PICU) with COVID-19 manifested as acute liver failure is described.

Key words: COVID-19; Acute liver failure; Children.

ARTICLE INFO

Received: 25 November 2022
Revision received: 20 December 2022
Accepted: 20 December 2022

Introduction

Coronavirus disease 2019 (COVID-19) is an illness caused by novel severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). The illness was initially noted in Wuhan in December 2019 and has since spread across the world. Children have been affected by the COVID-19, however they appear to have had a milder disease course and better prognosis than adults.[1] Despite the fact that pulmonary illness is the main clinical symptom, patients frequently show signs of damage to other organs, such as the gastrointestinal (GI) tract and liver, which sharply raises their mortality. Although these individuals frequently have modest liver enzyme increase, incidents of serious liver injury have also been documented. In this case report, a COVID-19 case that manifested as acute liver failure is described.

Case History

A 6-year-old boy presented to the hospital with complaints of fever for 6 days with mild upper right quadrant pain. Patient also had 2-3 episodes of vomiting associated with nausea and passage of high coloured urine for 3 days with hypersomnolence for 1 day. There was no history of cough, sore throat or breathlessness. His past medical history was unremarkable. Parents denied taking any recent over-the-counter or herbal drugs in the prior weeks.

On presentation, patient was vitally stable, temperature was 39.8 °C. Icterus was present. Patient was lethargic with disorientation in time. On examination, there was right hypochondriac tenderness and enlarged liver with liver span of 12 cm. There was no splenomegaly or ascites. Respiratory system evaluation revealed no abnormalities. At the time of his admission, his laboratory results were as follows: haemoglobin: 11.6 g/dL; leucocyte: 4300 cells/mm^3; platelets: 115,000 cells/uL; serum bilirubin: 4.1 mg/dL; aspartate transaminase (AST): 9116 U/L; alanine transaminase (ALT): 3797 U/L; alkaline phosphatase: 519 U/L; total protein: 6.8 g/dL; albumin 3.5 g/dL and international normalised ratio (INR): 2.9. An ultrasound of the abdomen showed minimal ascites and hepatosplenomegaly. IgM hepatitis A, IgM hepatitis E, the surface antigen of the hepa-

This article should be cited as follows: Nawghare P, Nair S, Jain S, Bansal S, Chandnani S, Rathi S, et al. A case of COVID-19 presenting as acute liver failure. Scr Med 2022 Dec;53(4):353-6.

titis B virus (HbSAg), anti-HCV, Epstein-Barr virus (EBV) DNA polymerase chain reaction (PCR) and cytomegalovirus (CMV) DNA PCR serological testing were conducted; all results were negative. IgM leptospira, dengue IgM and dengue nonstructural protein 1 (NS1), rapid malarial antigen tests were all negative. His autoimmune workup came out negative, as did his serum ceruloplasmin level. Prior to admission, a nasopharyngeal swab was performed as per standard hospital procedure and the reverse transcription polymerase chain reaction (RT-PCR) result showed COVID-19

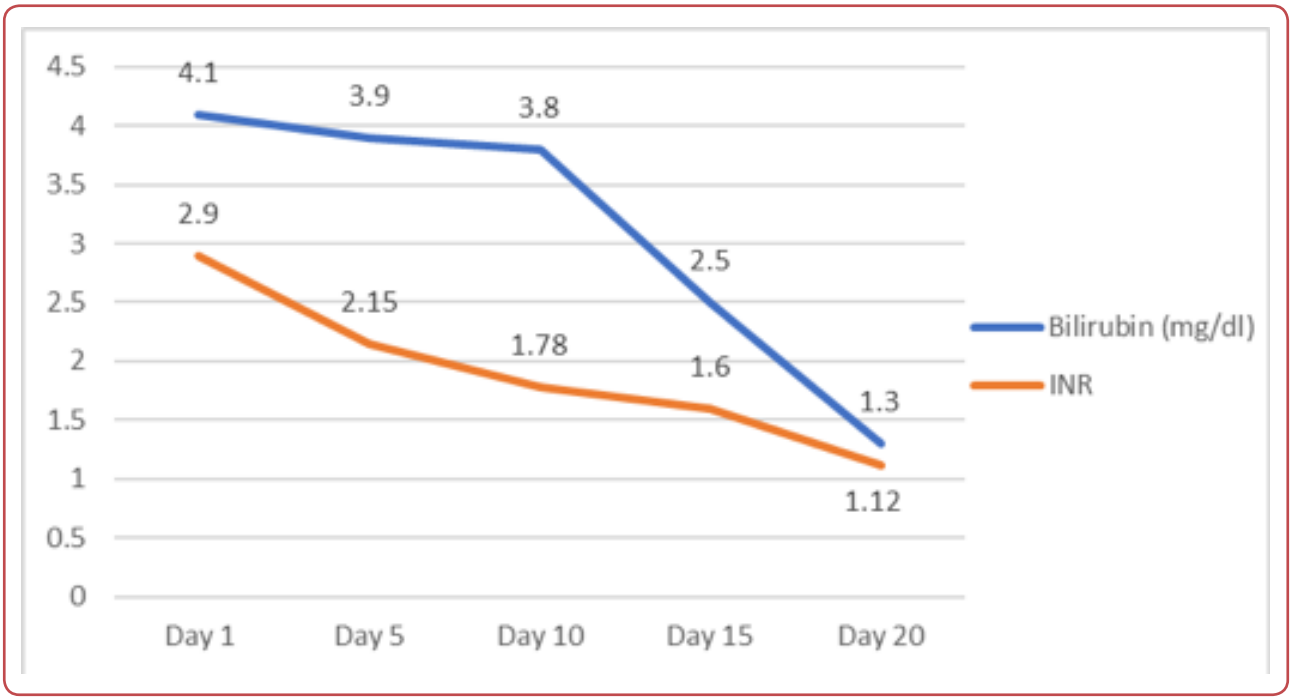

Figure 1: Serial serum bilirubin and international normalised ratio (INR) levels of the patient with COVID-19

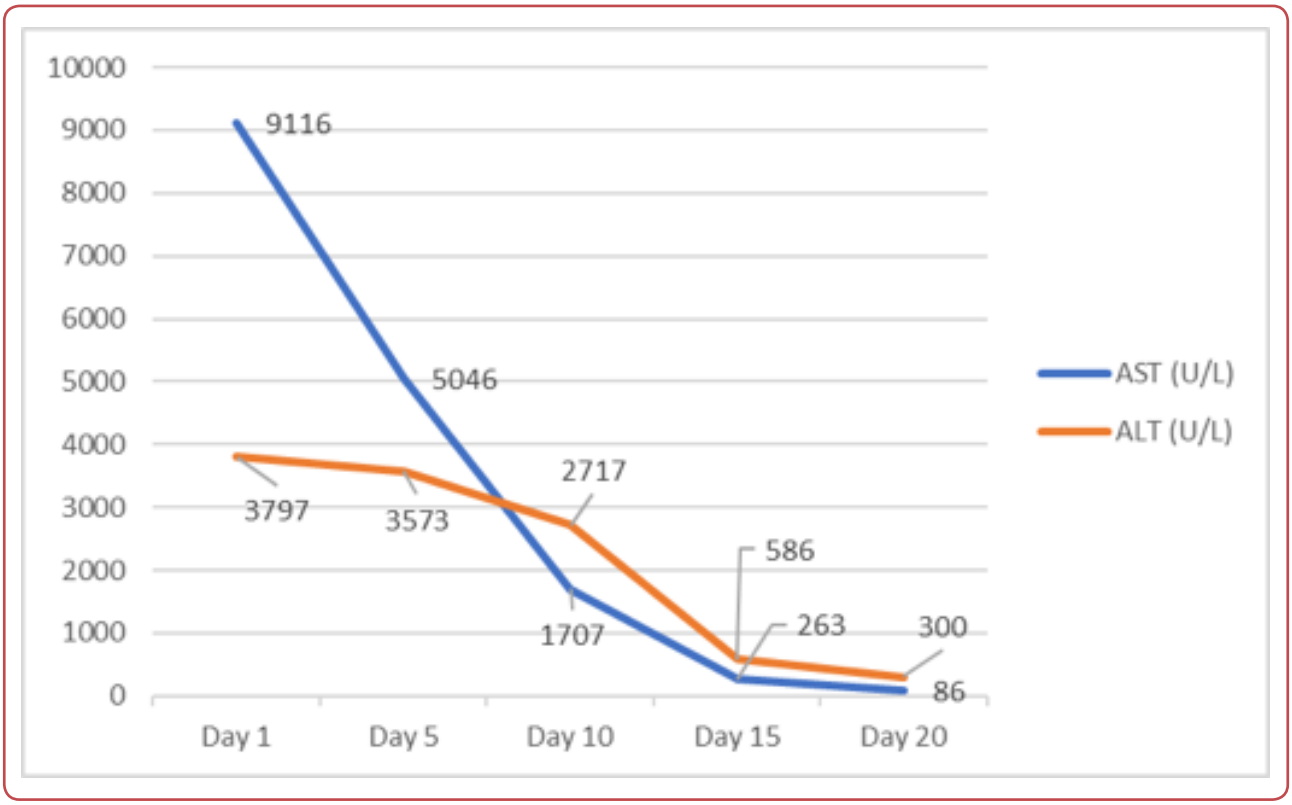

Figure 2: Serial aspartate aminotransferase (AST) and alanine aminotransferase (ALT) levels of the patient with COVID-19

infection. Patient had a peripheral oxygen saturation of 99 % at room air and no evidence of interstitial pneumonia was found on a chest X-ray.

The patient was managed conservatively with antibiotics, vitamin K supplementation and anti-hepatic encephalopathy measures. He improved during the hospital stay. Serial laboratory testing revealed a downward trend in liver enzymes (Figure 1), INR and serum bilirubin levels (Figure 2). After 20 days, a second nasopharyngeal swab for the SARS CoV-2 PCR test came out negative. After 23 days in the hospital, the patient was released in good clinical condition and was symptom-free from a hepatic and respiratory standpoint.

Discussion

Corona viruses are single-stranded RNA viruses that belong to the Coronaviridae family and the Orthocoronavirinae subfamily. In Wuhan, China, in December 2019, SARS-CoV-2, a new virus, was found to be the source of a cluster of pneumonia cases.[2] Due to the virus quick global spread and substantial number of hospital admissions and fatalities, the World Health Organization (WHO) has classified it as a global pandemic. COVID-19 is largely seen as a pulmonary illness and often appears with signs of viral pneumonia. Increasing evidence, however, points to the involvement of many organ systems, including the liver and GI tract, with more than 60 % of patients exhibiting GI symptoms (anorexia, diarrhoea, nausea and vomiting) and a sizeable fraction of cases exhibiting increased liver biochemistries.[3]

Liver damage in the context of COVID-19 may be complex. An infection of liver cells by a virus may cause liver damage directly. This virus enters the host cell by binding to the angiotensin converting enzyme 2 (ACE2) receptor,[4] where it multiplies and infects additional cells throughout the upper respiratory tract and lung tissue. According to Chai et al, ACE2 is expressed by bile duct cells as well as liver cells.[5] Additionally, in severely sick COVID-19 patients, immune-mediated inflammation such as cytokine storm and hypoxia brought on by pneumonia may exacerbate liver damage or ultimately result in liver failure. The majority of drugs used to treat moderate to severe COVID-19 patients, on the other hand, have distinct profiles of liver damage. Moreover, a previously undetected liver condition is frequently ambiguous.

The severity of liver damage can vary from asymptomatic biochemical abnormalities to, in rare instances, abrupt liver failure. The prevalence of liver damage in COVID-19 patients varied from 58 % to 78 %, with a larger percentage present in severe COVID-19 cases. Liver damage was mostly detected by raised AST, ALT and total bilirubin levels along with slightly reduced albumin levels.[5] AST and ALT elevations are typically moderate (ie, 5 times the upper limit of normal); nonetheless, very high aminotransferase levels and severe acute hepatitis have also been recorded.[6] In the pattern of elevation, AST is frequently higher than ALT. Although the ACE2 receptor is more usually expressed on cholangiocytes than hepatocytes, AST and ALT are more frequently increased than bilirubin or alkaline phosphate. When severe and non-severe COVID-19 patients were compared, those with severe illness had a higher frequency of liver function abnormalities such hypoalbuminemia, gamma-glutamyl transferase (GGT), aminotransferase elevations and bilirubin elevations.[7]

Upon evaluating the past literature, it was discovered that there was just one research that showed blood ALT and AST levels in severe COVID-19 patients climbed to 7,590 U/L and 1,445 U/L, respectively. SARS-CoV-2 infections in children are more likely to be asymptomatic and result in minor symptoms.[8] Amir Saeed et al described a case of a kid, age 11, who had COVID-19 and had acute liver failure.[9] Based on a study of the few available literature, presented case seems to represent a rare instance of COVID-19 infection manifesting as acute liver failure. In this period of COVID-19 infection, clinicians should be aware that abrupt liver failure may be the virus' early manifestation. Patients with COVID-19 risk factors who present with acute hepatitis should be isolated and tested for the virus. To ascertain how frequently this presentation occurs, more observational studies are required.

Acknowledgements

None.

Conflict of interest

None.

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