Ruptured Liver Abscess Post Severe COVID-19 Infection: A Case Report

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Received April 16, 2023; Accepted July 28, 2024.

Key words: SARS-CoV2 – COVID-19 – Vaccination – Liver abscess

Abstract: Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV2) caused imminent acute infection of respiratory tract known as Coronavirus disease 2019 (COVID-19). Complications of hepatobiliary system especially liver often found in post-acute COVID-19 patients. However, there are only few studies specifically discussing about liver abscess in patients who had history of contracted COVID-19. We present a case of a 54-years-old gentleman with no previous medical illness and no history of vaccination, who was presented with ruptured liver abscess post COVID-19 infection Category 4 (symptomatic with lung infection and the need of oxygen supplementation). Percutaneous drainage was performed to drain the abscess and collections.

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https://doi.org/10.14712/23362936.2024.25

Introduction

SARS-CoV2 is classified in Coronaviridae family, a single-stranded RNA virus. Angiotensin-converting enzyme 2 (ACE2) receptor became the main target of SARS-CoV2 (Nardo et al., 2021). ACE2 receptors also exist in other organs, such as the liver, heart, kidneys, pancreas and nerve sheaths (Li et al., 2020a; Skok et al., 2021). Liver abnormalities in post-infected SARS-CoV2 patients were shown in the other studies. Hepatocytes and cholangiocytes, which also contain the virus-specific ACE2 receptors, are directly susceptible to SARS-CoV-2 infection. According to expression profiling, cholangiocytes express ACE2 twenty times more than hepatocytes do. Compromised hepatic transaminases, as a sign of liver involvement, can result from direct injury of these cells (Yang et al., 2020).

Case report

A 54-year-old man developed sudden onset of the right hypochondriac and epigastric pain on day 22 post-COVID-19 infection Category 4. The nature of pain was described as colicky and radiated to the back, associated with vomiting and also loss of appetite. He did not have a history of liver disease or other systemic illnesses. He was not vaccinated with the COVID-19 vaccine prior to the infection. Initially, he was admitted to Covid Center Hospital before being transferred to our center for further management. During the transfer, the patient required respiratory support to maintain the oxygen requirement. He also completed the steroid regime for COVID-19 treatment for two weeks before the transfer.

On examination, there was tenderness at the right hypochondriac and epigastric region. Complete blood count showed a high total white count (29.2×10^9/l). In contrast, other biochemistry profiles, such as liver function test, showed elevation in aminotransferase enzymes and hypoalbuminemia features which were albumin 22 g/l, total bilirubin 42 umol/l, alkaline phosphatase (ALP) 4.37 ukat/l, alanine transaminase (ALT) 2.76 ukat/l, and aspartate transaminase (AST) 0.65 ukat/l. The C-reactive protein (CRP) also was on the higher side (151.0 mg/l).

Ultrasound of the hepatobiliary system revealed an ill-defined heterogenous hypoechoic lesion at segment V of the liver measuring 4×11.2×9.8 cm with a liquefied area within. A heterogeneous hypoechoic lesion was also present at left subphrenic region measuring approximately 6.0×16.6 cm. There was also the presence of echogenic and internal debris within with minimal peri splenic free fluid (Figure 1).

Further evaluation with a 4-phase liver CT (computed tomography) revealed a ruptured liver abscess with multiple loculated collections. There was a well-defined lobulated hypodense lesion with no significant enhancement in the right lobe of the liver, measuring approximately 11×9×10.4 cm (Figure 2). The lesion continued with

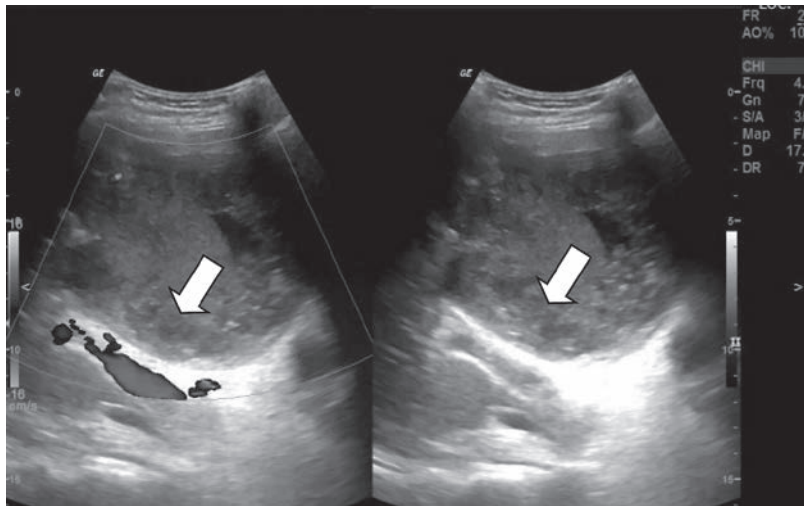

Figure 1 – Ultrasound hepatobiliary-hypoechoic lesion at segment V (arrows). Presence of echogenic and internal debris within with minimal peri splenic free fluid.

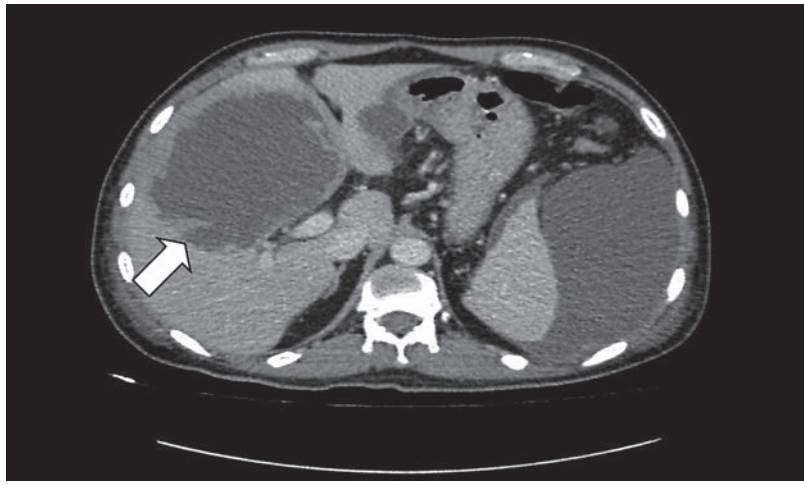

Figure 2 – Computed tomography liver 4 phases revealed a well-defined lobulated hypodense lesion with no significant enhancement in the right lobe of the liver measuring approximately 11×9×10.4 cm (arrow).

an elongated loculated collection between the stomach and duodenum (D1 and D2) and the left lobe of the liver, measuring about 2.8 cm in maximum thickness. There was also a loculated collection at the hepatorenal region. At the left hypochondrium, the subcapsular splenic collection at the superolateral aspect of the spleen extended to the subphrenic region, measuring about 13.5×9.6×14 cm.

Given the patient's symptoms and ongoing infection in the setting of post-COVID-19 infection, percutaneous drainage under ultrasound guidance was done. About 800 ml thick, exudative and yellowish pus was drained out during the procedure. An immediate start of intravenous meropenem during his stay in the ward was indicated. Neither pus nor blood cultures showed any growth. No acid-fast bacteria (AFB) were seen in direct smears. A serial hepatobiliary ultrasound done post-drainage showed improvement in terms of the reduced size of the collections and resolution of the left subphrenic collection. The patient was discharged after two weeks. He was well during the follow-up at the surgical outpatient clinic.

Discussion

Previous studies showed that severely ill patients with COVID-19 tended to develop gastrointestinal complications and had higher percentages for liver function derangements combined with low serum albumin level (Tian and Ye, 2020). This patient presented with a sudden onset of abdominal pain 22 days after the confirmed COVID-19 Category 4. There was clear evidence of elevation in aminotransferase enzymes and hypoalbuminemia with a raised total white count and CRP level.

Direct cytotoxicity of SARS-CoV2 virus by viral replication resulted in liver damage. This situation resulted in a hypoxic state brought on by respiratory failure, coagulopathy-induced vascular alterations, endothelial tissue inflammation, drug-induced liver damage and a history of liver disease exacerbations. Hypoxia and inflammation, which are frequent in severe COVID-19 instances, are crucial in the process of hepatocellular ACE2 expression (Nardo et al., 2021). The dysfunction of cholangiocytes, which can result in impaired bile production, inflammation, fibrosis, and liver dysfunction, might increase the expression of ACE2 in liver tissue, which may be one of the mechanisms of liver damage brought on by SARS-CoV2 infection (Banales et al., 2019). The SARS-CoV-2 induces direct damage to the biliary ducts by binding to ACE2 on cholangiocytes (Zhang et al., 2020). The other mechanisms of liver damage include hyperinflammation seen with cytokine storm and hypoxia-associated metabolic derangements (Li et al., 2020b). As our patient had been previously diagnosed with severe COVID-19 infection, hypoxia and inflammation led to extrapulmonary SARS-CoV2 dissemination that contributed to the liver abscess formation as there was a significantly raised ACE2 expression in the liver tissue.

It was proposed that possible factors behind the development of liver abscesses include suppression of immunity by COVID-19 infection itself and usage of steroids (Sahney et al., 2022). For our patient, we postulated that the predisposition to develop the liver abscess might have been related to COVID-19 itself and could have been accentuated by the previous use of steroids during their treatment for the severe infection of COVID-19.

As this patient did not have any medical illnesses and symptoms of liver abscess disease prior to COVID-19 infection, there was no available imaging for him. The very first imaging was done when the patient complained of the sudden onset of abdominal pain, as mentioned in the case report. Radiology images revealed a ruptured liver abscess with multiple loculated collections. Percutaneous drainage under ultrasound guidance was performed. No AFBs were seen in direct smear. We thus concluded that the patient was not diagnosed with extrapulmonary tuberculosis (TB). No microbes were found in the pus culture, as mentioned in the other case report of a COVID-19 patient with liver abscess (Dhadiala and Whatkar, 2021). As this was the first episode of abdominal symptoms, we concluded that this patient did not have any pre-existing liver abscess before their COVID-19 infection based on the symptoms' onset. The radiology images also showed evidence of ruptured liver abscess even though no common microbes were found in the cultures or smears. We hypothesized the liver abscess in our patient was associated directly with the COVID-19 infection according to these factors.

In our case, the core management focused on combination of drainage and targeted antibiotic therapy. As there are studies regarding liver abscess in post-COVID-19 patients still lacking, precautions for COVID-19 infection should still be taken. Our case report outlined the prompt diagnosis, pathophysiology, and approach to liver abscess after COVID-19 infection.

Conclusion

Due to the extremely small number of reported clinical cases, the pathophysiology and optimal management of liver abscess after COVID-19 have not been determined. As reported in our case, the pathophysiology of liver abscess after COVID-19 infection may result from the development of liver necrosis due to a specific interaction of the SARS-CoV2 with the liver parenchyma through ACE2 expression. Understanding the pathophysiology processes can guide the novel treatment of the disease in the future.

References

Banales, J. M., Huebert, R. C., Karlsen, T., Strazzabosco, M., LaRusso, N. F., Gores, G. J. (2019) Cholangiocyte pathobiology. *Nat. Rev. Gastroenterol. Hepatol*. **16(5)**, 269–281.

Dhadiala, S. K., Whatkar, A. A. (2021) Rare case of liver abscess in COVID 19 positive. *Int. J. Curr. Res.* **13(6)**, 17932–17934.

Li, R., Qiao, S., Zhang, G. (2020a) Analysis of angiotensin-converting enzyme 2 (ACE2) from different species sheds some light on cross-species receptor usage of a novel coronavirus 2019-nCoV. *J. Infect*. **80(4)**, 469–496.

Li, H., Liu, L., Zhang, D., Xu, J., Dai, H., Tang, N., Su, X., Cao, B. (2020b) SARS-CoV-2 and viral sepsis: Observations and hypotheses. *Lancet* **395(10235)**, 1517–1520.

Nardo, A. D., Schneeweiss-Gleixner, M., Bakail, M., Dixon, E. D., Lax, S. F., Trauner, M. (2021) Pathophysiological mechanisms of liver injury in COVID-19. *Liver Int*. **41(1)**, 20–32.

Sahney, A., Wadhawan, M., Agarwal, N., Berry, N., Dadwal, V., Vardani, A., Kumar, A. (2022) A case series of amoebic liver abscess in patients with COVID-19 infection. *J. Clin. Exp. Hepatol*. **12(3)**, 1017–1020.

Skok, K., Stelzl, E., Trauner, M., Kessler, H. H., Lax, S. F. (2021) Post-mortem viral dynamics and tropism in COVID-19 patients in correlation with organ damage. *Virchows Arch*. **478(2)**, 343–353.

Tian, D., Ye, Q. (2020) Hepatic complications of COVID-19 and its treatment. *J. Med. Virol*. **92(10)**, 1818–1824.

Yang, R. X., Zheng, R. D., Fan, J. G. (2020) Etiology and management of liver injury in patients with COVID-19. *World J. Gastroenterol*. **26(32)**, 4753–4762.

Zhang, C., Shi, L., Wang, F. S. (2020) Liver injury in COVID-19: Management and challenges. *Lancet Gastroenterol. Hepatol*. **5(5)**, 428–430.