CASE REPORT

COVID-19 in A Liver Cirrhosis Patient

Lia Sasmithae

Departement of Internal Medicine, Faculty of Medicine, Universitas Palangkaraya/Mas Amsyar Hospital, Katingan

Corresponding author:
Lia Sasmithae. Departement of Internal Medicine-Faculty of Medicine, Universitas Palangkaraya/Mas Amsyar Hospital.Jl. Bukit Raya IX No.32 Palangkaraya Indonesia. Phone: +62-12-53176556; Facsimilie:-. E-mail: liasasmithae032@gmail.com.

ABSTRACT

The COVID-19 pandemic is a novel disease and posed a great challenge in the current healthcare system. The exact impact of the COVID-19 virus on the liver is still unknown. However, in a patient with chronic liver disease, most COVID-19 infections will affect the survival rate and initiate liver decompensation. This study reported a 50-years-old man who complained about bloody vomit and black tarry stool with COVID-19 infection. Physical examination findings included hematemesis, pale conjunctiva, ascites, collateral vein; and from the rectal toucher, there was melena. There was no fever, cough, or shortness of breath. The laboratory and radiological examinations showed that there were normochromic normocytic anemia, hypoalbuminemia, slightly increased ALT/AST, HBsAg (+), and abdominal ultrasound findings were liver cirrhosis with ascites. The patient was screened for the COVID-19 antigen swab test (+), further confirmed by the COVID-19 PCR swab test (+). The treatment given for hematemesis and melena was Gastric Cooling; the patient was fasted, then received somatostatin PPI drip, Vitamin K injection, PRC transfusion, lactulose, ceftriaxone, ascites fluid puncture, and albumin transfusion. After the bleeding resolved, the patient received spironolactone and propranolol. The treatments for COVID-19 were Azithromycin, Favivirapir, Vitamin D, Vitamin K, and Zinc. The patient was hospitalized for 11 days and then improved.

Conclusion: This study reported a case of a 50 years-old man with ruptured esophageal varices due to liver cirrhosis with concomitant COVID-19 infection and improved with comprehensive therapy despite the limited facilities at the hospital.

Keywords: COVID-19, liver cirrhosis, ruptured esophageal varices

ABSTRAK


Kesimpulan dilaporkan sebuah kasus laki-laki, 50 tahun dengan rupture varises esofagus karena sirosis hepatis disertai infeksi Covid-19 dan membaik dengan terapi yang komprehensif meskipun dalam keterbatasan fasilitas pemeriksaan di rumah sakit.

Kata kunci: Covid-19, sirosis hepatis, ruptur varises esofagus

INTRODUCTION

COVID-19 is a novel disease that caused a high morbidity and mortality rate and is also identified in patients with comorbidity, including liver cirrhosis. The virus that caused COVID-19 disease is SARS-CoV-2 (Severe Acute Respiratory Syndrome Coronavirus 2). Patients infected with COVID-19 mostly come with complaints of fever, and with respiratory symptoms including pneumonia and acute respiratory distress syndrome (ARDS) which increase deaths from COVID-19.

Over time, the symptoms and manifestations of COVID-19 infection are increasingly varied and develop into extrapulmonary manifestations, one of which is gastrointestinal and hepatobiliary manifestations. Several studies have linked the expression of the angiotensin-converting enzyme 2 (ACE2) receptor found on hepatocytes and cholangiocytes with the ability to directly infect the SARS-CoV-2 virus.

Patients with liver cirrhosis are highly vulnerable to SARS-CoV-2 infection due to decreased immune system. Preliminary studies reported that 2-11% of patients infected with COVID-19 have pre-existing liver disease. Nevertheless, the actual prevalence rate from liver cirrhosis patient that infected with Covid-19 is not yet known.

This case report aimed to report a rare case, namely a patient with liver cirrhosis who was infected with COVID-19 and to examine the involvement of the virus in the occurrence of liver decompensation.

CASE ILLUSTRATION

A 50-years-old man came to the emergency room with bloody vomit 10 times and a black tarry stool one time. The patient also complained of abdominal pain. In 2019, he had been diagnosed with liver disease and undergone a procedure to remove fluid from his abdomen, but he never received routine treatment. It was the first time the patient had experienced bloody vomit and black tarry stool. The patient had no previous complaints of fever, cough, shortness of breath. The history of consuming alcohol and traveling was denied. From the general condition and vital signs, it was found that the consciousness was comatose, the patient was looked moderately ill, Glasgow Coma Scale 4-5-6, blood pressure 152/80 mmHg, heart rate 60 beats/minute, respiratory rate 24 times/minute, temperature 37.1°C, SpO₂ 97% without oxygen supplementation.

Physical examination findings included anemic conjunctiva, collateral vein at the abdomen, ascites, and positive rectal toucher (RT) test for melena. Laboratory examination found normochromic normocytic anemia (Hb 6 gr/dL, MCV/MCH 83 pg/26.3 pg), thrombocytopenia (89,000/ul), leucopenia (3,700/ul), positive HBsAg, hypoalbuminemia (2.3 g/dl), AST/ALT (56/44 U/l). The patient was screened for Covid-19 infection with Covid-19 antigen swab test (+), further confirmed with PCR swab test showed a positive result (+3) (FAM 32.387; HEX 34.2017; Cys 25.012). The results of laboratory tests after undergoing treatment and therapy showed improvement, namely Hb 8.6 g/dL, MCV/MCH 83.1 pg/27.4 pg, platelets 108.000/ul, after undergoing albumin transfusion to 2.5 g/dL, PCR evaluation was carried out on day 10 treatment showed negative results (FAM -; HEX -; 31105).

Ascites fluid analysis showed a conclusion that contained transudate ascites fluid. USG examination showed a liver cirrhosis appearance with ascites. The plain chest radiograph was normal. From the history taking and physical examination, and additional testing, the patient was diagnosed with liver cirrhosis Child-Pugh classification B with ruptured esophageal varices and confirmed Covid-19 infection. The patient was treated in the Covid-19 isolation ward and was fasted. The patient was given oxygen supplementation.
with nasal canule 2 lpm, NGT installation, gastric cooling per 8 hours until the bleeding stop; if it was clean for three times, it could be continued with a liquid diet six times 200cc. A urinary catheter was placed to monitor the patient's fluid balance. In addition, the patient was treated with Pantoprazole injection 2 x 40 mg, somatostatin drip for one day (because this drug was not available), Ceftriaxone injection 1 x 1 g for prophylaxis of hepatic encephalopathy, Vitamin K injection 3 x 1 ampoule, Tranexam Acid injection 3 x 1 ampoule, Sucralfate syrup 3 x C2, and Lactulose 3 x C1. A paracentesis was carried out as much as 4,5 L. The treatments given for Covid-19 in this patient were Azithromycin 1 x 500 mg, Favirapir loading dose 1600 mg/12 hours/oral for the first day, then continued with 2 x 600 mg until the fifth day, Vitamin D 1 x 400 mg, Zinc 1 x 1 tablet, Vitamin C 3 x 500 mg.

Seven days after received antiviral drugs, a PCR swab test for evaluation was performed. After undergoing treatment in the isolation ward for ten days, the patient was transferred and observed in the regular ward. The patient was also given medicines to prevent portal hypertension, including spironolactone 1 x 200 mg and beta-blocker propranolol 3 x 10 mg. During the evaluation, there was no bloody vomit, black tarry stool, and ascites; then, the patient was allowed to be discharged from the hospital and continued self-isolation at home.
The occurrence of bloody vomit, black tarry stool, ascites, and collateral vein might be aggravated due to COVID-19 infection or portal hypertension because of liver failure itself; to date, it was still unknown.

Jin et al reported that gastrointestinal manifestation was found in 11.4% of patients with COVID-19 infection, and 28% of these patients did not have any feature of respiratory system, this study is in accordance with the current case report where the patient did not show any features or symptoms regarding the respiratory system.10 Cheung et al revealed that the cumulative prevalence of gastrointestinal manifestation was around 17.6%, whereas Pan et al reported a higher percentage of 20.5%.11 Clinical symptoms of COVID-19 associated with the gastrointestinal system include anorexia, diarrhea, nausea and vomiting, abdominal pain, and gastrointestinal bleeding. Pan et al revealed that anorexia was the most reported symptom (76.8%), followed by diarrhea (34%), vomiting (3.9%), and abdominal pain (1.9%).12 Upper and lower gastrointestinal were rare gastrointestinal symptoms but could potentially be severe. Gastrointestinal bleeding prevalence in COVID-19 patients was unclear. Gastrointestinal bleeding could occur without features of the respiratory system.9

The impact of SARS-CoV-2 infection on patients with a history of chronic liver diseases, such as hepatitis B or C virus infection, is still not clearly understood. Patients with hepatic cancer or liver cirrhosis are more vulnerable to SARS-CoV-2 infection due to immunocompromised conditions.9 Exposure of SARS-CoV-2 to the liver was based on ACE2 receptors distribution in hepatocytes and cholangiocytes.7 Study conducted by Chai et al reported that ACE2 receptor more expressed in cholangiocytes (59.7%) than in hepatocytes (2.6%).13 The presence of ACE2 receptors in cholangiocytes supports evidence regarding retrograde liver damage due to viral entry into the biliary tree cells.14,15 SARS-CoV-2 infection probably binds directly to cholangiocytes and induces cytopathic effects. Disorder in cholangiocyte’s function causes damage in hepatobiliary cells. Covid-19 infection can aggravate comorbid conditions, such as chronic liver disease, which can progress to liver decompensation and acute liver failure and increase patient mortality rate.16,17

In this study, the patient was given treatments for COVID-19 infection as well as ongoing acute bleeding. The COVID-19 treatments provided included vitamin C, vitamin D, Zinc, Azithromycin for five days, and antiviral, specifically Faviravirapir, for five days. For the treatments of gastrointestinal bleeding, namely hematemesis-melena, the patient received somatostatin only for one day due to limited drug supply; and high doses of PPI and PRC transfusion were also given. Hematemesis was resolved after five days of treatment. After the bleeding stopped, spironolactone and propranolol treatment were started. The patient was evaluated with a PCR swab test on the seventh day of treatment and gained negative PCR results after received treatments for COVID-19. The patient was then transferred to the regular ward for observation and evaluated for three days, then the patient was discharged and was advised to be referred for endoscopic examination.

As a conclusion, this study reported a case, a 50 years-old man presenting with bloody vomit and black tarry stool with confirmed mild COVID-19 infection and hematemesis-melena due to ruptured esophageal varices because of liver cirrhosis child-pugh classification B post necrotic hepatitis B.

The COVID-19 disease can exacerbate the comorbid condition, such as chronic liver disease, which can progress to liver decompensation and acute liver failure along with high mortality. In this case, the possibility of liver decompensation in the form of hematemesis-melena and ascites may be aggravated by the presence of the COVID-19 virus infection; therefore, comprehensive management and further studies regarding involvement of the COVID-19 infection in liver disease are needed.

REFERENCES


