Bowel Necrosis in Patient with Severe Case of COVID-19: A Case Report

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Case Report

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Abstract

Background: In patients who are critically ill with COVID-19, multiple extrapulmonary manifestations of the disease have been observed, including gastrointestinal manifestations.

Case Presentation: We present a case of a 65 year old man with severe COVID-19 pneumonia that developed hypercoagulation and peritonitis. Emergent laparotomy was performed and we found bowel necrosis in two sites.

Conclusions: Although rare, the presentation of COVID-19 with bowel necrosis requires emergency treatments, and it has high mortality rate.

Background

The novel coronavirus disease, caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), has spread worldwide becoming a pandemic and is currently causing more than 900,000 deaths. Indonesia is one of the countries with the highest number of registered cases and deaths.(1)

The Coronavirus belongs to a family of RNA viruses that can cause respiratory infection with varying symptoms, which usually includes cough, fever, fatigue, headache and myalgia after 2–14 days of exposure. This virus enters the human cells through the angiotensin-converting enzyme-2 (ACE-2) receptor, which is also expressed in the gastrointestinal tract epithelium. GIT involvement can be characterized by abdominal pain, hyporexia, nausea, and vomiting with a variable incidence ranging from 5% to 50% of cases.(2,3)

In addition, COVID-19 may also predispose to venous and arterial thromboembolic diseases due to excessive inflammation, hypoxia and diffuse intravascular coagulation through the triad of hypercoagulation, blood stasis, and endothelial injury. This occurrence of vascular thickening and thromboembolic resulting in increased hypoxemia with predictive value of adverse outcomes associated with D-dimer levels.(2,4)

In this case report, we describe a patient with COVID-19 who developed a bowel necrosis that required emergent surgical treatment.

Case Presentation

A 65-year-old male patient with past medical history for diabetes mellitus was brought to the hospital for having 7 days of fever and cough, and recent onset of dyspnea. The patient was given 15 lpm of oxygen with non-rebreathing mask, and oxygen saturation became 90%. Laboratory tests were as follows: haemoglobin 16,2 g/dL; leucocyte 9.000/μL; lymphocyte 3%; thrombocytes 222.000 /μL; CRP 0,2 mg/dL; D-dimer 0,78 mg/L; HbA1c 8,0%. His chest x-ray showed severe bilateral pneumonia (Fig. 1) and his real-
time PCR on nasopharyngeal and oropharyngeal swabs were tested positive for SARS-COV-2. The patient was diagnosed with COVID-19 with severe case and received COVID-19 regimen.

After seven days of hospitalized, the patient suddenly complained of epigastric pain. His respiratory rate was increased, oxygen saturation was 88-90% with 15 lpm of oxygen, and there was tenderness in the epigastric. Laboratory results shows an increase in leucocyte 15.100/μL; D-dimer 14,04 mg/L and Albumin 1,4 g/dL. A contrast abdominal CT scan was performed, infarction was found in both kidneys and no abnormalities in the abdominopelvic organ (Fig. 2). Abdominal pain was suspected due to mesentery ischemia caused by hypercoagulation. Anticoagulant therapy with low-molecular weight heparin 25.000 IU/day were administered; with the addition of tramadol 100 mg/day. On the next day, the patient felt less abdominal pain, D-dimer decreased to 8.54/μL and the medications were continued.

Three days after his epigastric pain, the patient complained of had a worsened abdominal pain with marked distension, inability to pass gas or defecate. On physical examination, signs of peritoneal irritation were found. His leucocyte increased to 33.100 /μL; aPTT 71,4 seconds (control aPTT 24.1 seconds). Abdominal X-ray showed free air in the abdominal cavity and distension of the large bowel (Fig. 3). We diagnosed it as intestinal perforation in COVID-19.

The patient underwent an emergent laparotomy. The operation was performed under general anaesthesia, after opening the stomach, air came out of the abdomen and the bowel was soak with reddish black feses. We explored it and obtained a brownish liquid about 200 cc. During the surgery, we found necrosis in two locations: first, we found round necrosis, 3 cm in diameter, yellowish black in antimesenterial site. In the second location, 20 cm whole intestinal necrosis was found at 1 meter from the first site (Fig. 4). We did bowel resection at each site of necrosis and end-to-end anastomoses by using manual suturing (Fig. 4). The histology showed a small bowel tissue with extensive haemorrhagic infarction and perforation.

Right after surgery, the patient was admitted to the ICU and was put on ventilator, with anticoagulant and other medications continuing. Six days after surgery, his condition continued to get worsen and the patient passed away in the ICU.

**Discussion And Conclusions**

Several researchers have reported the link between COVID-19 and generalized organ damage, including organs in the abdominopelvic. This association is thought to be because SARS-CoV-2 binds to the angiotensin-converting enzyme 2 (ACE-2) receptor found in alveolar epithelial cells in the lung, and these receptors are also detected in gastrointestinal epithelial cells resulting in infection and local viral replication, and increase cytotoxic effect.(4,5) In addition, some authors also think that organ damage in some COVID-19 patients may be caused by severe systemic inflammation caused by upregulation of cellular and natural immunity. SARS-CoV-2 infection triggers the activation of T lymphocytes and the inflammatory signaling pathway which ultimately results in the release of multiple proinflammatory cytokine, such as granulocyte-macrophage colony-stimulating factor (GM-CSF), interleukin (IL) -2, IL-6, IL-
7, IL-10, and tumor necrosis factor-α (TNF- α). (6) This cytokine cascade can eventually result in extensive cell damage, necrosis, and injury to multiple organs and may partly explain the different multisystem symptoms in patients with confirmed viral infections, including gastrointestinal necrosis. (6,7)

Intestinal necrosis is a late stage discovery characterized by cell death due to reduced blood flow to the digestive tract. This serious condition is often fatal and can lead to vascular occlusion, colitis, obstruction, or infection. In adults, the most common cause of intestinal necrosis is acute mesenteric occlusion, and, less commonly, perforation, chronic ischemia, inflammatory disease, and other mechanical disorders. (8,9) In our case, the bowel necrosis we found had patent blood vessels and did not involve mesenteric necrosis, therefore, we think that it might be due to microvascular thrombosis and the inflammation associated with hypercoagulability in this patient. Several studies have recently investigated the association between COVID-19 and hypercoagulability which is usually characterized by high D-dimer. (10,11) Some patients may show prolonged thrombin time and prothrombin time, and shortened aPTT. Several other studies have shown aPTT prolongation in patients with confirmed COVID-19. (11)

Bowel necrosis without adequate intervention and proper surgical management will lead to near 100% mortality. The operative mortality rate for acute mesenteric ischemia has been reported to be 47%. Patients who survive the initial event have a high probability of postoperative complications. (12,13) Sepsis can cause hypotension and end-organ damage, especially kidney and liver failure. For those who survive the early intervention, mortality continues to increase from comorbidities. (13)

Although rare, the emergent surgery for bowel necrosis and COVID-19 infection presents unique challenges for clinicians.

**List Of Abbreviations**

- **ACE-2** angiotensin-converting enzyme-2
- **aPTT** activated partial thromboplastin time
- **COVID-19** Coronavirus Disease 2019
- **CRP** C-Reactive Protein
- **GM-CSF** granulocyte-macrophage colony-stimulating factor
- **ICU** Intensive Care Unit
- **IL** interleukin
- **PCR** Polymerase Chain Reaction
Declarations

Ethics approval and consent to participate

This article approved by committee ethics of Pondok Indah Puri Indah Hospital.

Consent for publication

Written informed consent was obtained from the patient's next of kin for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Availability of data and materials

Not applicable.

Competing interests

The authors declare no competing interests.

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Authors’ contributions

DAS and WH analysed and interpreted the patient data. ST wrote the paper with input from all authors. All authors discussed and contributed to the final manuscript.

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