

## Single Case

# Ischemic Colitis in a Patient with Severe COVID-19 Pneumonia

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## Keywords

COVID-19 · Ischemic colitis · Thrombembolism · Acute abdomen

## Abstract

At the time of the current COVID-19 pandemic, on a daily basis, we encountered patients suffering from various manifestations of this infection. The most common are respiratory symptoms. Many of the patients require acute hospital care, and a smaller group of them are hospitalized in intensive care units. A subset of these critically ill patients demonstrates clinically remarkable hypercoagulability and thus a predisposition to venous and arterial thromboembolism, manifested by thrombotic events ranging from acute pulmonary embolism and splanchnic vascular ischemia to extremity ischemia. The article describes a case of a patient with COVID-19 pneumonia complicated by massive bleeding into the gastrointestinal tract due to ischemic enterocolitis in connection with COVID-19 infection.

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## Introduction

Even though the classical presentations of COVID-19 infection are with upper and lower respiratory tract symptoms [1, 2], over time, there have been numerous extrapulmonary presentations like gastrointestinal manifestations that were noticed. According to some studies, around 50% of SARS-CoV-2 patients present with GI symptoms like diarrhea, nausea,



**Fig. 1.** Chest X-ray: diffusely thickened lung pattern, paracardial to confluent on the left – character of inflammatory infiltration. The left diaphragm is blurred (photo: author's archive).

vomiting, and abdominal pain [3–6], followed by anorexia, anosmia, and dysgeusia [7]. Due to lack of knowledge of the pathophysiology of the gastrointestinal form of SARS-CoV-2 infection, together with the ambiguous conclusions of diagnostic imaging methods in patients suffering from this infection, it is a challenge when deciding on the right therapy – whether to proceed conservatively or operate.

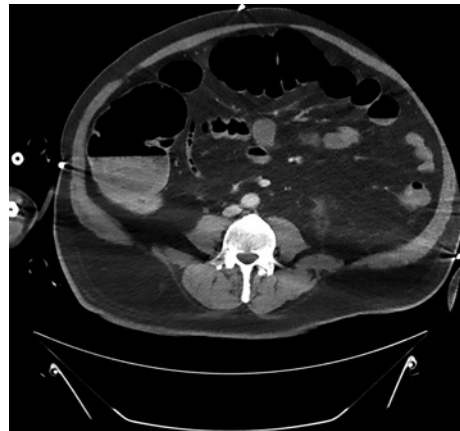
### Case Report

A 67-year-old patient was presented to the emergency department on 11 January 2021 with a history of 4 days of worsening dyspnea, irritating cough with an expectoration of brownish sputum, and general weakness. The patient suffers from metabolic syndrome, is being treated for diabetes and hyperlipidemia, and intermittently uses PPI. At admission, the patient was subfebrile, complained of nausea, did not vomit, negated diarrhea or chest pain, and had no loss of taste or smell.

Initially, blood saturation of 92% O<sub>2</sub> without oxygen, borderline blood pressure of 115/60 mm Hg, and mild tachycardia around 100 rpm were measured. There was a rapid test performed for COVID-19, which was positive. Furthermore, a PCR test was performed, which confirmed the previous result. A chest X-ray subsequently showed bilateral pneumonia (Fig. 1). Laboratory analysis of the blood revealed an elevation of inflammatory markers (CRP 168.9 mg/L, leukocytes 4.0 10<sup>9</sup>/L, PCT 7.13 µg/L, Hb 143 g/L, thr 272 10<sup>9</sup>/L, INR 0.93), decompensated diabetes mellitus (glycemia 18.9 mmol/L), and normal renal and liver function.

The patient was acutely admitted to the ward reserved for COVID-19-positive patients and was treated in a barrier regime. Antibiotic therapy was started; corticoids, bronchodilators, LMWH were added to medication, and diabetic medication with a good effect on glycemia was adjusted.

During hospitalization, the patient started to feel shortness of breath and had an irritating cough. Despite the raising of oxygen therapy (with max. 15 L/min O<sub>2</sub> mask with reservoir) desaturation gradually up to 76% O<sub>2</sub>, the patient needed transfer to the intensive care unit. Deep venous thrombosis and pulmonary embolism were excluded. HFNO therapy was introduced; corticoids and antibiotics were continued; and the dose of LMWH was increased. On the 6th day of hospitalization, progression occurred, arterial astrup blood gas analysis showed severe hypoxemia. There was indicated translation to the anesthesiology



**Fig. 2.** CT angiography of abdomen with i.v. and p.o. contrast – area of distended caecum up to 80 mm, without wall thickening, with dense liquid intestinal content (photo: author's archive).

and resuscitation department and intubation, construction of tracheostomy, escalation of ATB, and nutrition via nasogastric tube were started. Temporarily, circulatory support of vasopressors for signs of septic shock was required. The patient's condition slowly improved; catecholamines were withdrawn, febrile episodes subsided, and inflammatory markers decreased. Sedatives were discontinued; the patient regained consciousness, but low muscle strength remained a limitation.

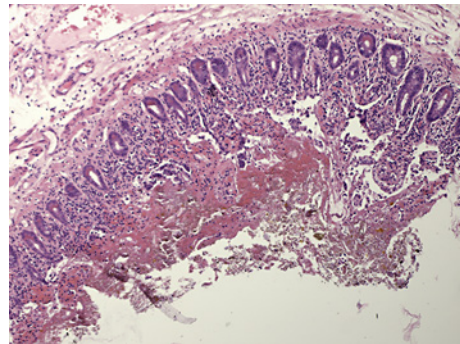
From day 21 of hospitalization, the patient was considered noninfectious. Transfer to a rehabilitation care ward was planned.

However, on the 23rd day of hospitalization, the day of the planned transfer, there was a sudden rapid deterioration in the patient's overall condition; he developed circulatory instability with tachypnea, fibrillation, accompanied by massive enterorrhagia. Laboratory blood analysis showed a drop of hemoglobin to 81 g/L, and the clinical picture corresponded to septic shock. The patient was intubated again, and circulatory resuscitation was started, supported with catecholamines up to 40 mL/h; multiple blood substitutes were given, hemostatic therapy was started, and LMWH therapy was discontinued. Abdominal ultrasound was performed with the finding of undilated small-bowel loops, meteoric colon, here and there with borderline width 6–7 cm, with no obvious wall thickening and no detection of free fluid in the abdominal cavity.

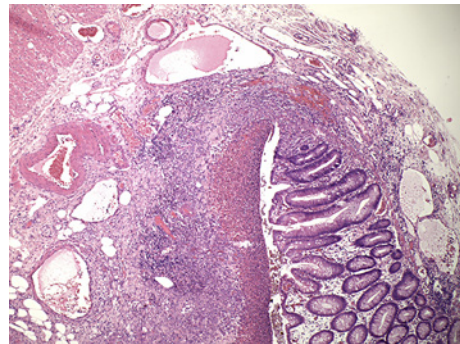
Moreover, a gastroenterologist was contacted who performed gastrofibroscopy and colonoscopy. Gastroscopy showed normal mucosa from the visualized esophagus to the D2 part of the duodenum with no signs of bleeding; colonoscopy was performed only to the area of the hepatic flexure. The clarity of the colon wall was significantly aggravated due to the presence of intestinal contents and admixed with coagula. No source of bleeding or mural lesion was found in the evaluated range. CT angiography of the abdomen showed liquid content in the cecum and suspected coagulum in the aboral portion of the sigmoid colon and ampulla recti. The cecum and sigmoideum were distended to 80 and 60 mm, without wall thickening. No clear source of bleeding was evident (Fig. 2).

Due to the general condition and signs of continued bleeding, acute abdominal revision was indicated. The abdominal cavity was inspected by midline laparotomy; it was free of effusion. The dominant finding was a distended colon, especially in the cecum area with translucent hemorrhagic filling. The small bowel was free of translucent blood and distention throughout. Desufflation of the cecum was performed over the stump of the appendix, verifying its hemorrhagic filling. Subsequently, a revision of the small intestine was performed from a short enterotomy in the area of the terminal ileum, which was filled with enteral contents only, without signs of bleeding. Pathology was therefore presumed to be in the area

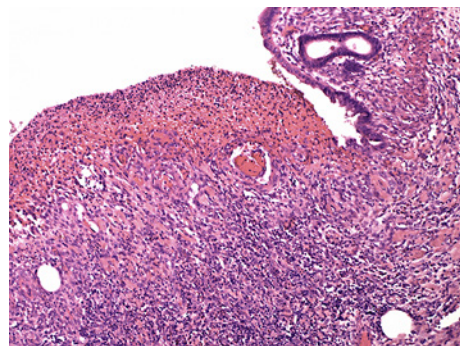
**Fig. 3.** Detail of the small-intestine mucosa: erosions and neutrophilic infiltration of the superficial epithelial layer – possible sign of ischemic enteritis. Standard hematoxylin-eosin staining, magnification, ×40 (photo: author’s archive).



**Fig. 4.** Detail of colonic mucosa with visible sharp cutoff changing into ulceration. Standard hematoxylin-eosin staining, magnification, ×40 (photo: author’s archive).



**Fig. 5.** Detail of colonic ulceration richly permeated with erythrocytes. Visible is a vessel with fibrin thrombus in the lumen (thrombi in vessels together with changes such as erosions or ulcerations are assuming ischemic etiology of the lesion). Standard hematoxylin-eosin staining, magnification, ×100 (photo: author’s archive).



between the colonoscopically reached hepatic flexure and the cecum. It was decided to perform a right-sided hemicolectomy with side-to-side ileotransversoanastomosis.

Histological examination of the resected tissue revealed ischemic enteritis and colitis with mucosal ulcerations (Fig. 3–5). There was no evidence of infectious or IBD inflammation and no evidence of a Dieulafoy lesion. There was also no sign of dysplasia or malignancy in the extent examined.

In the subsequent postoperative period, the patient’s general condition gradually stabilized. Weaning was without major complications. There was a progressive adjustment of the intestinal passage. The patient was loaded with enteral nutrition via a nasogastric probe. The patient was hospitalized until day 42, when he was transferred to a post-acute intensive care unit. Two weeks later, the patient was readmitted for recurrent GIT bleeding. Colonoscopy showed a small ulceration in the area of ileotransversoanastomosis. The condition was managed conservatively with hemodynamic therapy, and the patient was transferred back to the post-acute intensive care department without further signs of bleeding.

## Discussion

The first case of COVID-19 was reported in Wuhan, China, in December 2019, when severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) was recognized. Since then, the virus has spread worldwide. On 11 March 2020, COVID-19 was declared a global pandemic by the WHO [1]. Initially, very little was known about the virus, its pathogenesis, and manifestations. In the last few months, physicians around the world have been intensively analyzing and studying the disease, with the multiplying literature and articles describing the various unexpected manifestations and unpredictable complications of the disease.

The target viral receptors for SARS-CoV-2 are angiotensin-converting enzyme-2 receptors, which are abundantly present in alveolar cells, but are also known to be present in glandular cells of gastric, small intestine, colonic, and rectal epithelium, which may have a major impact on the development of gastrointestinal symptoms [8]. Furthermore, coagulopathy, accompanied by elevated D-dimer and fibrinogen levels, has been associated with an increase in morbidity and mortality in patients with COVID-19 [9]. The incidence of ischemic colitis in patients with SARS-CoV-2 infection appears to be closely associated with this coagulopathy [10].

Gastrointestinal bleeding has been described in 2–13% of patients hospitalized with COVID-19 [11–13]. Endoscopy remains the diagnostic and often therapeutic method of first choice.

The most common sources of upper GIT bleeding in patients hospitalized with COVID-19 include gastric, duodenal, or esophageal ulcerations and esophagitis. In the lower GIT, cases of rectal ulceration (associated with rectal catheter insertion), colitis, and diverticular bleeding are recorded. Whether GI bleeding in these patients is primarily due to COVID-19 disease, indirectly due to treatment-related effects, or a combination of both has not been fully elucidated [14].

SARS-CoV-2 is associated with the development of coagulopathy, hypercoagulability, and thromboembolic complications, especially in patients with severe COVID-19 infection. Coagulopathy in COVID-19 patients is a well-known and described phenomenon that is associated with a poor overall prognosis. Patients with COVID-19 regularly have significantly elevated D-dimer and fibrinogen levels, which also puts them at high risk for microcirculatory and macrocirculatory thromboses [15]. Recent research suggests that the hypercoagulable state of COVID-19 patients is more likely to be the result of a “cytokine storm” than a manifestation of progression of disseminated intravascular coagulation [16]. The patient in our case report had fluctuating D-dimer and fibrinogen levels during hospitalization, and these levels were above normal throughout the hospitalization. On admission to the hospital, a prophylactic dose of LMWH (Fraxiparine 0.4 mL every 24 h) was administered; however, in light of the high D-dimer levels with signs of hypercoagulable state, the dose was then increased to therapeutic. Anticoagulation therapy was stopped on day 23 of hospitalization due to massive enterorrhagia with sudden circulatory instability and significant drop in hemoglobin. Due to failure of diagnostic methods and because of continued bleeding, the patient was indicated for acute surgical revision.

The hypercoagulable state most often causes pulmonary embolism or deep vein thrombosis and less frequently presents as mesenteric ischemia. Deep venous thrombosis and pulmonary embolism have typical clinical manifestations even in critically ill patients and are among the expected complications, especially in the ICU setting. In the case of our patient, ECHO of the heart and duplex sonography of the deep veins of the lower extremities were performed during hospitalization to exclude these complications. However, colonic ischemia remains a diagnostic challenge, especially in patients with severe COVID-19 infection under ICU care as the characteristic abdominal pain is either masked or virtually absent in intubated and sedated patients [17]. Imaging modalities such as abdominal CT quite often demonstrate focal necrosis of the bowel caused by small-vessel thrombosis, suggesting that the ischemia is due to in situ thrombosis rather than a thromboembolic event [18, 19]. Thrombosis in situ

is also advocated by the fact that we observe elevated von-Willebrand factor levels in these patients, leading to endothelial damage and activation and subsequent thrombosis. In addition, the target viral receptor of the new coronavirus is the angiotensin-converting enzyme-2 receptor, which is abundantly expressed not only in alveolar cells but also in the GIT which may also explain endothelial tropism and the resulting endothelial dysfunction, endothelial damage, and thrombosis [20, 21].

The severe course of COVID-19 may also lead to nonocclusive colonic ischemia as shock and hemodynamic compromise are common in COVID-19 pneumonia. In patients requiring inotropic support, intense vasoconstriction of small vessels and reduced blood flow through the mesenteric arteries occur via alpha-adrenergic stimulation, further impairing blood supply to the cells of the intestine [8]. Noradrenaline and high levels of adrenaline cause intense vasoconstriction by their stimulation of adrenergic receptors. Other pharmacological compounds that reduce splanchnic blood flow include vasopressin, phenylephrine, and digoxin. However, pharmacological inotropic support does not always cause vasoconstriction; low doses of dopamine (3–8 µg/kg/min) and adrenaline (0.05–0.10 µg/kg/min) have been shown to increase mesenteric blood flow, providing adequate vasopressor support while protecting the intestine from ischemia [22].

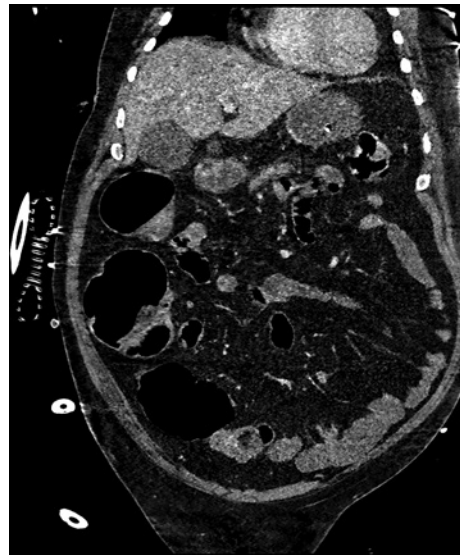
Ischemic colitis is a condition caused by a decrease in blood supply to the colon (possible etiologies listed above), resulting in mucosal damage, ischemia of intestinal cells, and necrosis [23]. Usually, only a specific area of the colon is affected by ischemia. Due to the limited collateral vascularization are most at risk of developing ischemia splenic flexure and recto-sigmoid junction (so-called “watershed” areas) [24]. Evidence of right-sided ischemic colitis in COVID-19 patients in the literature is more rare [25].

Colonic ischemia may be temporary or irreversible, depending on its extent and duration. Colitis is one of the most mild and therefore reversible conditions. Colitis manifests itself within 3 days of the onset of compromise blood supply, when mucosal edema is absorbed, resulting in mucosal ulceration. These ulcerations take several months to heal, although the patient may be free of any clinical symptoms [26]. More severe ischemic damage may lead to full-width necrosis of the bowel wall.

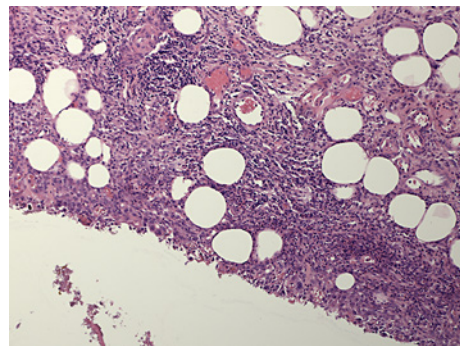
The diagnosis of colonic ischemia must be thought of in patients who develop severe abdominal pain accompanied by GIT bleeding in the immediately following 12–24 h. Early colonoscopy is recommended in these patients [27]. Common but less specific symptoms include painless diarrhea, nausea, or vomiting. Clinically, varying degrees of abdominal palpatory tenderness may be observed depending on the extent of bowel injury, and even peritoneal irritation, signs of SIRS, which are sepsis, tachypnea, and tachycardia, may be present, along with continued leukocyte elevation, metabolic acidosis, lactate, inorganic phosphate, and alkaline phosphatase elevation [28–34].

Treatment of ischemic colitis depends on the severity of manifestation. Milder cases can be managed mainly with supportive care – bowel rest, i.v. fluids, antibiotics, and close observation if there is no evidence of necrosis, gangrene, or perforation of the bowel. Surgical intervention and colonic resection is indicated in cases where imaging proves colon necrosis or for patients with right-sided colon involvement [35–37].

Our patient’s acute condition could have been result of bowel ischemia during COVID-19 infection, which led to ischemic ulcerative colitis accompanied by massive bleeding. A nonocclusive etiology is highly unlikely given the fact that the patient did not receive vasopressors for 9 days prior to the bleeding complication nor did he show signs of sepsis. Another possible cause of the bleeding could have been lipomatosis of the Bauhin valve (Bauhin syndrome, Fig. 6, 7), which was demonstrated by histopathological examination of the resection. This rare disease is usually asymptomatic but can manifest with nonspecific abdominal pain, constipation, diarrhea, and bleeding [38, 39].



**Fig. 6.** CT angiography of the abdomen with p.o. and i.v. contrast – area of the caecum with apparent lipomatosis of the Bauhin's valve (photo: author's archive).



**Fig. 7.** Detail of the area of Bauhin's valve with lipomatosis: a visible partition of the intact crypts of the colonic mucosa and ulceration. Inflammatory and reparative changes at the base of the ulceration affect the adipocytes. Standard hematoxylin-eosin staining, magnification,  $\times 100$  (photo: author's archive).

The clinical manifestations and complications of COVID-19 disease are diverse. In this case report, we described a case of a rare gastrointestinal complication, massive bleeding due to ischemic ulcerative colitis. In patients with severe COVID-19 pneumonia, we should always assume a hypercoagulable state. In critically ill patients with the need for medical circulatory support, it is necessary to remember the possibility of ischemic complications and eventually to recognize and treat these complications early.

### Statement of Ethics

This retrospective review of patient data did not require ethical approval in accordance with local/national guidelines. Written informed consent was obtained from the patient for publication of the details of their medical case and any accompanying images.

### Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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## Author Contributions

All the authors of this paper have directly participated in the planning, execution and analysis of this study; have been involved in drafting the manuscript and revising it critically for important intellectual content; and have read and approved the final version submitted. Ilona Krejčová is the author of the article, collected data, did analysis and interpretation, and wrote the paper. Alena Berková made substantial contribution to conception and design of the paper, did acquisition of data, and wrote the paper. Laura Kvasnicová made substantial contribution to conception and design of the paper, did acquisition of data, and wrote the paper. Petr Vlček, Lenka Veverková, and Igor Penka did analysis and interpretation of data. Dušan Zoufalý and Vladimír Červeňák did acquisition of data.

## Data Availability Statement

All data generated or analyzed during this study are included in this article. Further inquiries can be directed to the corresponding author.

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