




# Thrombosis leading to acute abdomen in corona virus disease- 19: A case series

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## Abstract

Involvement of the gastrointestinal (GI) system in corona virus disease-19 (COVID-19) in form of diarrhea, loss of taste, nausea, and anorexia is common and associated with poor prognosis. COVID-19 is also associated with a hypercoagulable state that mainly involves the pulmonary vasculature. However, GI complications involving thrombosis are observed infrequently. We report two COVID-19 patients who had two different causes of acute abdomen. The first patient was a 49-year-old male diagnosed with an aortic thrombus along with a splenic infarct. He was diagnosed early and successfully managed with anticoagulants. The second patient was a 30-year-old male who developed pain in the abdomen and was found to have features suggestive of peritonitis. A contrast-enhanced computerized tomography (CECT) scan of the abdomen revealed dilated bowel loops. Immediate exploratory laparotomy was performed; he was found to have jejunal perforation with gangrene. Histopathological examination of the resected specimen showed inflammatory cells with edema and thrombotic vessels. However, he succumbed to sepsis and multiorgan failure. Therefore, it is important to investigate cases of acute abdomen in COVID-19 thoroughly and whenever indicated CT angiogram should be obtained.

**Keywords** Angiogram · COVID-19 · Disease management · Gastrointestinal · Intestinal perforation · Mesenteric ischemia · Multiple organ failure · SARS-CoV-2 · Splenic infarction · Thrombophilia

## Bullet points of the study highlights

### *What is already known?*

- Corona virus disease-19 (COVID-19) is known to cause gastrointestinal (GI) symptoms in 17.6% of patients.
- The most common symptoms are diarrhea, loss of taste, nausea, and anorexia.

### *What is new in this study?*

- Thrombotic complications involving GI tract are uncommonly reported.
- We report two cases of acute abdomen with different mechanisms because of thrombotic complications of COVID-19.

### *What are the future clinical and research implications of the study findings?*

- Acute abdomen in COVID-19 should lead us to a prompt workup in the form of computerized tomography (CT) angiography and prothrombotic workup to rule out thrombosis.

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## Introduction

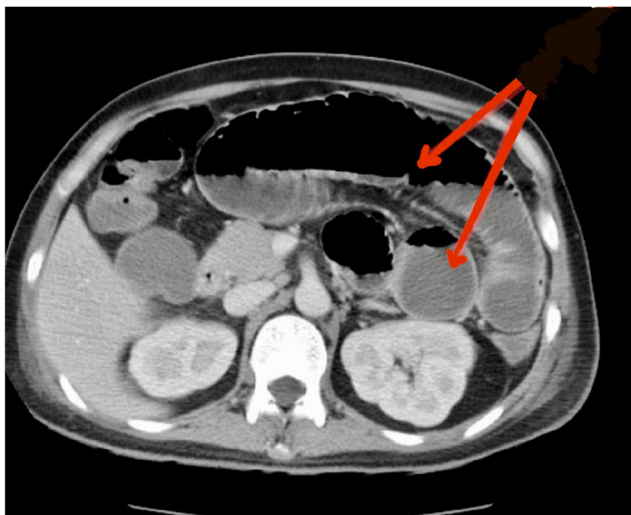
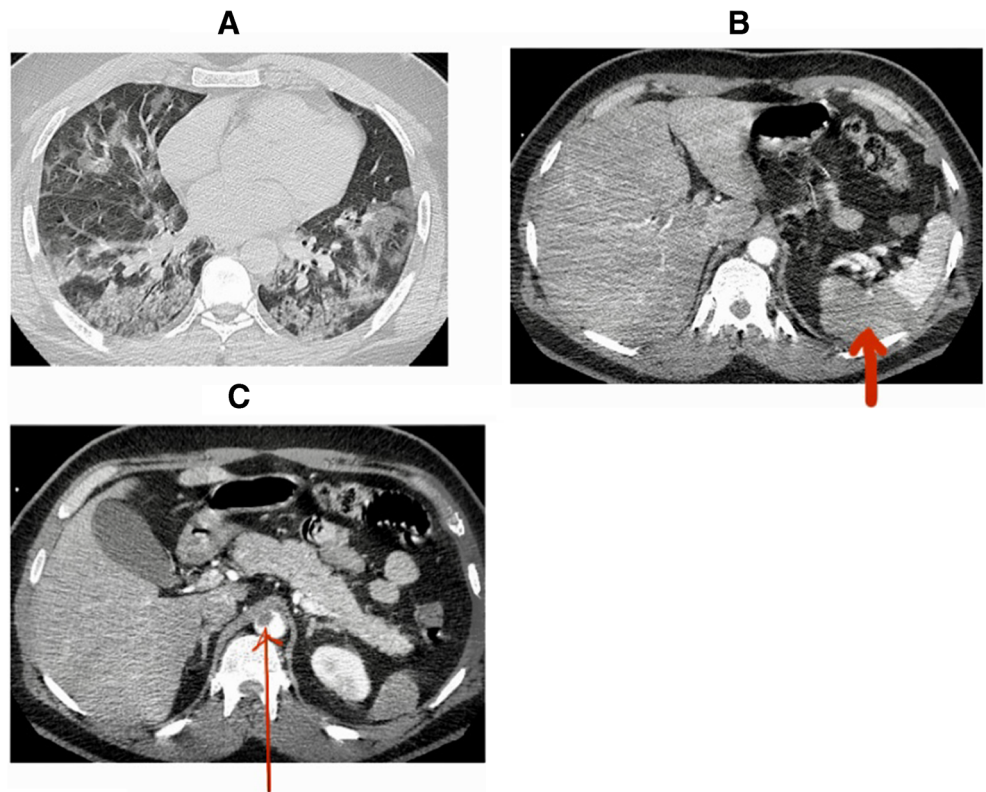
The threat of corona virus disease- 19 (COVID-19) is not over yet, as various parts of the globe are continuing to have COVID-19 cases. COVID-19 is a complex disease with the propensity to affect multiple systems both by direct viral invasion and by indirect mechanisms. Since the respiratory system comes first in direct contact with the virus, respiratory manifestations are seen most commonly. The most common symptoms are cough, sputum, shortness of breath, fever, etc., which may progress to acute respiratory distress syndrome (ARDS), shock, and death. However, gastrointestinal (GI) complications are not infrequently described. It is estimated that 9% to 17.6% of patients have some form of GI involvement [1–3]. Radiographically, thickening of the small and large bowel has been described [4]. Moreover, COVID-19 is also a prothrombotic condition, and thrombosis involving various vessels has been described. We hereby report two different causes of acute abdomen in which hypercoagulability and thrombosis played a central role. At our center until August 2021, 3583 patients with COVID-19 had been admitted. Of them, perhaps only two patients were diagnosed with abdominal thrombosis. We are presenting these two interesting cases:

**Case 1:** A 49-year-old, non-diabetic, non-smoker male presented with fever and dyspnea of 10-day duration to a local hospital where coronavirus disease reverse transcription-polymerase chain reaction (COVID RT–PCR) was performed, which came positive. He did not have any significant past medical or surgical history. He was a non-smoker, non-alcoholic, leading a non-sedentary lifestyle. On admission, he was tachypneic (respiratory rate 25/min) and hypoxic (SpO<sub>2</sub> 93% room air). His blood reports showed normal blood counts. High-resolution computerized tomography (HRCT) of the thorax revealed changes consistent with COVID-19 (Fig. 1). His serum ferritin, C-reactive protein, and fibrin-degradation product (FDP) were elevated with a positive D-dimer test. Treatment was started with oxygen support via a non-rebreather mask at 12 L per minute (L PM), intravenous glucocorticosteroids, antibiotics, and injection enoxaparin 60 mg once daily, subcutaneously. During the hospital stay, he had an acute episode of left-sided chest and left

upper quadrant abdominal pain, 2 days after admission. There was no history of vomiting or change in bowel frequency. Acute coronary syndrome (ACS) was ruled out by electrocardiogram, 2D echocardiography, and cardiac enzyme levels. Because of persistent pain radiating from the left hypochondrium, CT angiography of the thorax and abdomen was performed, which revealed an aortic thrombus and splenic infarct (Fig. 1). Cardiology consultation was taken and tablet aspirin 75 mg once a day and injection enoxaparin 60 mg twice a day were started. He improved symptomatically in 7 days after therapy initiation; oxygen support was gradually weaned off, and his COVID RT–PCR became negative. A follow-up scan was performed after a month and showed complete resolution of the aortic thrombus and contraction of the infarct; however, there was no evidence of liquefaction. A gastroenterology consult was sought, and he was managed conservatively. He was discharged on rivaroxaban, which was continued for 3 months. He is currently doing well and is on follow-up of more than 1 year.

**Case 2:** A 30-year-old, non-diabetic, non-smoker, male electrician who was COVID-19 positive was admitted with breathlessness and oxygen requirement of 5 litre PM. He complained of pain in the abdomen on day 5 of his illness. There was no history of altered bowel habits, vomiting or obstipation. On examination, patient was hemodynamically stable, and had tense abdominal distension with features of peritonitis. Contrast-enhanced computed tomography (CECT) abdomen showed dilated bowel loops. No thrombosis in aorta or major mesenteric vasculature could be detected (Fig. 2). Bowel wall enhancement pattern and thickness appeared normal. Peritonitis was suspected clinically, and emergency surgical exploratory laparotomy was performed, 3 days after admission which showed thickened bowel walls with submucosal edema, engorged mesenteric vessels, gangrenous jejunum, and four perforations located 25, 28, 34, and 42 cm from one resection end. There was no obvious thrombosis seen in major mesenteric vessels. Therefore, a diagnosis of ischemic jejunal perforation with peritonitis was made. Approximately 2 ft of the jejunum was removed, and an ileostomy was performed. Histopathology of the resected bowel revealed mixed inflammatory cell infiltrate with congested blood vessels,

**Fig. 1** (A) Axial high-resolution computerized tomography (HRCT) lung window images showing multifocal ground-glass opacities and consolidation patches in both lungs. Computerized tomography (CT) angiography images showing non enhancing posterior part of the splenic parenchyma (B) suggestive of an infarct and adherent thrombus in the anterior wall of the abdominal aorta above the level of the celiac axis. (C) However no obvious thrombus was seen in the splenic artery



**Fig. 2** Contrast-enhanced computerized tomography (CECT) of the abdomen of case 2 showing dilated jejunal loops

edema, and congestion. There was transmural mixed inflammatory cell infiltrate and multiple vascular thrombi in the submucosa on histopathology, suggesting gangrene (Fig. 3). The patient developed sepsis and multiorgan failure and could not survive.

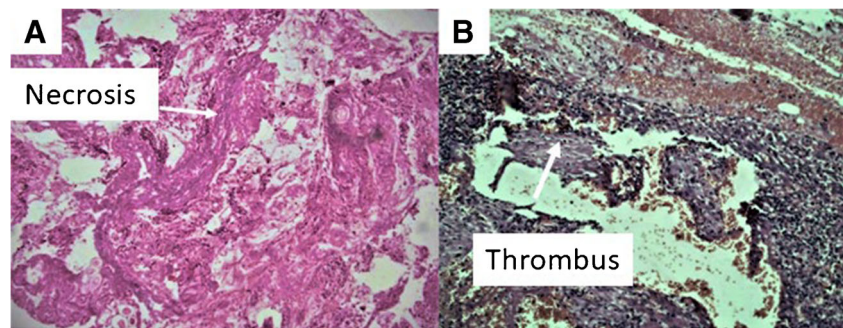
The clinical and laboratory parameters of both patients are summarized in Table 1.

**Discussion**

The incidence of thrombosis in critically ill COVID-19 patients is approximately 31% [5]. Pulmonary embolism has been the most common thrombotic event associated with COVID-19, sometimes despite prophylactic or therapeutic-dose anticoagulation [6]. Autopsy studies have also demonstrated pulmonary arterial thrombosis in COVID-19-related ARDS [7]. Severe acute respiratory syndrome corona virus-



**Fig. 3** Histopathology section from the gangrenous jejunum of case 2 shows (hematoxylin and eosin stained X 400) necrosis (a) and thrombosis of the vessel (b)



**Table 1** Clinical features of patients

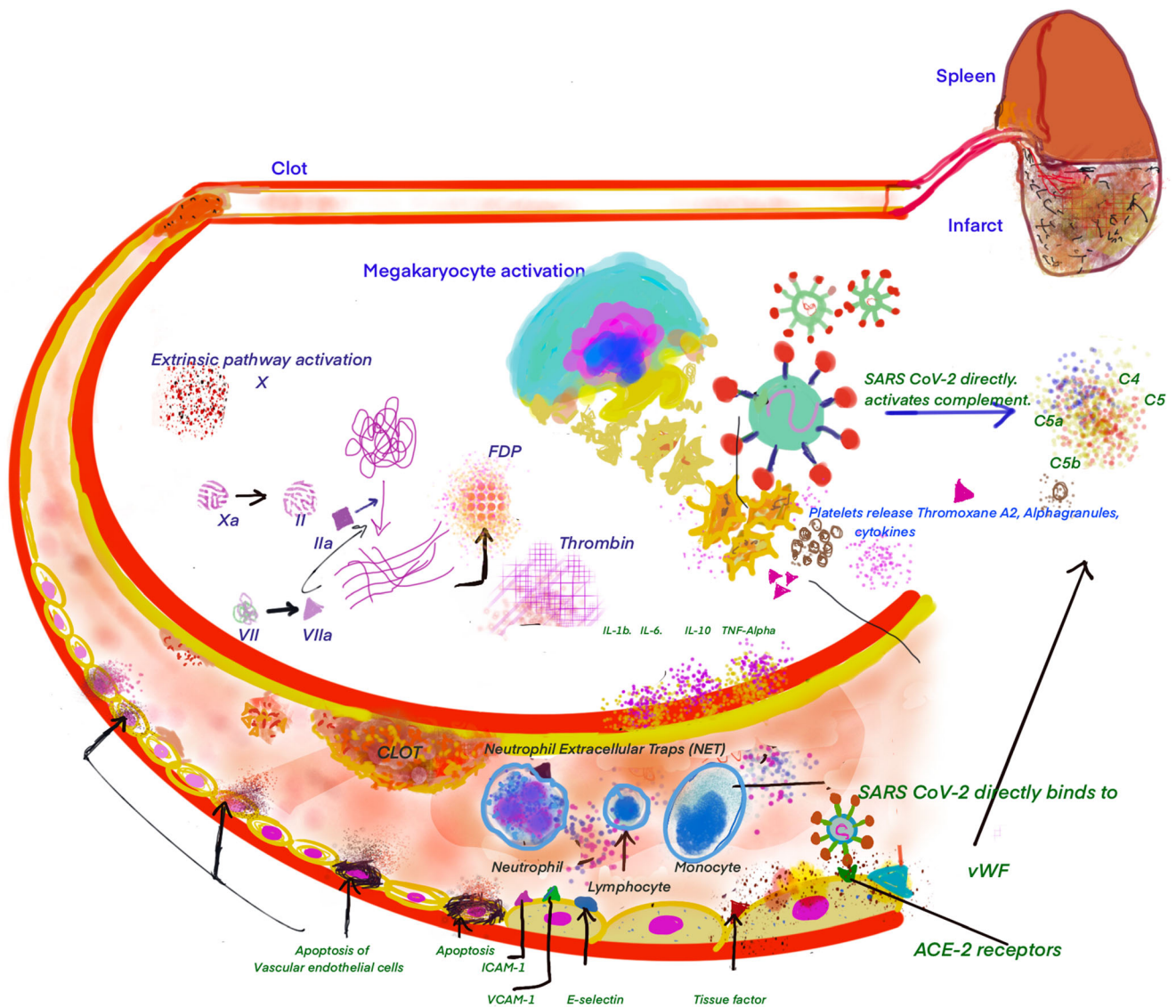
	Case 1	Case 2
Age (years)/sex	49/M	30/M
Hypertension	No	No
Diabetes	No	No
CAD	No	No
CLD	No	No
CVA	No	No
RF requiring dialysis	No	No
BMI (kg/m <sup>2</sup> )	26.7	23.9
Oxygen requirement at admission (L PM)	10-15	5-6
P/F ratio	80	112
PaCO <sub>2</sub> mmHg	39	35
Respiratory rate (per minute)	25	32
Hypotension	No	Terminally
Platelet(×10 <sup>3</sup> /mL)	172	165
D-dimer (mcg/mL)	6245	7234
FDP	Positive	Positive
Fibrinogen (mg/dL)	545	617
TEG	Hypercoagulable	Hypercoagulable
Ferritin (ng/mL)	1614	1213
CRP (mg/L)	165	121
HRCT thorax	14	8
CTSI		
ECG	Sinus tachycardia	Normal
Echocardiography	Normal	Normal
Doppler lower limb	No DVT	No DVT
Lung compliance	-	WNL
LMWH	Enoxaparin 60 mg BD	Enoxaparin 60 mg BD
Vasopressor initial	NA.	NA
Bleeding complications	None	None
Outcome	Discharged	Expired
Final diagnosis	COVID-19 Severe Pneumonia Aortic thrombus with splenic infarct	COVID-19 Severe Pneumonia Jejunal perforation peritonitis Thrombosis of feeding vessels

CAD coronary artery disease, COVID-19 corona virus disease - 19, CLD chronic liver disease, CTSI computerized tomography severity index, DVT deep vein thrombosis, HRCT high-resolution computerized tomography, ICU intensive care unit, L PM liters per minute, LMWH low-molecular-weight heparin, NA not applicable, P/F ratio PaO<sub>2</sub>/FIO<sub>2</sub>, WNL within normal limits, RF renal failure, TEG thromboelastography

2 (SARS-CoV-2) enters the gut through its attachment to angiotensin-converting enzyme 2 (ACE-2) receptors present in the epithelium of the esophagus and enterocytes. The most common GI symptoms are diarrhea, vomiting, anorexia, and abdominal pain [2]. Severe complications such as hemorrhage and perforation are rare. GI arteries are comparatively less affected by thrombosis. This result is similar to a study comprising 225 COVID-19 patients who underwent abdominal CT, of whom only two patients were found to have bowel ischemia [4]. A similar case of aortic thrombus with splenic infarct was reported in a 60-year-old COVID-19-positive female despite therapeutic anticoagulation [8]; similarly, a case of splenic artery thrombosis in a 60-year male was also reported [9]. Small bowel infarctions have been described in case reports [10, 11]. The cause of mortality in our second case was sepsis as a result of bowel perforation, in addition to COVID-19. Extrapulmonary involvement has been shown to increase mortality. There was no significant comorbidity in our patients to explain the hypercoagulable state. Both of our patients were receiving low molecular weight heparin (LMWH) in therapeutic doses. The development of thrombosis in patients already receiving LMWH has been documented, suggesting a multifactorial origin of hypercoagulability in COVID-19, which carries a poor prognosis.

SARS-CoV-2 attaches to ACE-2 receptors and damages the endothelium by direct infection. Viral ribonucleic acid (RNA) extensively damages the endothelium, activates platelets, activates the extrinsic pathway, and releases von Willebrand factor, thrombin, and fibrin (Fig. 4). In addition, hypoxia in severe pneumonia can stimulate thrombosis by increasing blood viscosity and a hypoxia-inducible transcription factor-dependent signaling pathway. The reported abnormal coagulation parameters include elevated D-dimer, FDPs, and low antithrombin values, showing prognostic value. A high index of suspicion is of paramount importance in managing COVID-19 patients with any persistent symptoms.

To conclude, these two cases of acute abdomen emphasize the importance of timely evaluation of abdominal symptoms and that CECT of the abdomen with CT angiogram is the investigation of choice if thrombosis is suspected clinically.



**Fig. 4** Severe acute respiratory syndrome corona virus-2 (SARS-CoV-2) attaches to angiotensin-converting enzyme (ACE-2) receptors present on the vascular endothelium and damages it by direct infection, which leads to apoptosis and release of tissue factor (TF) and von Willebrand factor (vWF). This leads to activation of the complement pathway and activation of neutrophils, monocytes, and lymphocytes, which lead to the release of interleukin-1 (IL-1), interleukin-6 (IL-6), interleukin-10 (IL-10), and tumor necrosis factor-alpha (TNF- $\alpha$ ). SARS-CoV-2 also directly activates platelets and megakaryocytes, which activates the

extrinsic coagulation pathway, which leads to the formation of fibrin from fibrinogen. Fibrin degradation products (FDPs) are also released. Vascular cell adhesion molecule (VCAM-1), intercellular cell adhesion molecule (ICAM-1), and E-selectin present in the endothelium play an important proinflammatory role. Therefore, a combination of endothelial damage and inflammation leads to a hypercoagulable state that leads to microvascular or macrovascular clots, which cause blockage of vessels and infarction or gangrene

**Declarations**

**Patient consent** The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published, and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

**Conflict of interest** ZH, AK, PA, ZN, AN, SJ, and SM declare no competing interests.

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