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Ischemic Bowel as a Thrombotic Complication of COVID-19: A Case Report

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Abstract

Background: Coronavirus disease 2019 (COVID-19) is now recognized as a multisystem illness associated with a significant risk of thrombotic complications due to a hypercoagulable state. While venous thromboembolism is well documented, gastrointestinal ischemia remains a rare but severe manifestation. The incidence, clinical course, and outcomes of COVID-19-associated ischemic bowel are not well characterized, though available reports suggest high mortality. Proposed mechanisms include endothelial injury, inflammatory-mediated coagulation activation, and elevated prothrombotic factors. *Case*: A 67-year-old male presented to the emergency department with several days of generalized weakness and exertional dyspnea. Initial diagnostic considerations included anemia, dehydration, electrolyte abnormalities, and infection. Further evaluation revealed acute COVID-19 infection complicated by thrombotic disease resulting in ischemic bowel. His hospital course was prolonged and complex, marked by multiple complications and progressive functional decline. Despite aggressive medical management and supportive care, his prognosis remained poor. He was ultimately discharged to a long-term acute care facility with a tracheostomy tube, enteral feeding dependence, and continued mechanical ventilation. *Conclusion*: This case underscores the prothrombotic potential of COVID-19-associated hypercoagulability and highlights ischemic bowel as a rare yet devastating complication. Clinicians should maintain a high index of suspicion for thrombotic events in patients with COVID-19, even when initial symptoms are nonspecific, as delayed recognition may result in significant morbidity and mortality.

Keywords

Thrombosis, COVID-19, Hypercoagulable, Ischemic

1. Introduction

Coronavirus disease 2019 (COVID-19), first reported in December 2019, was initially described as primarily respiratory illness but is now widely recognized as a multisystem disease. Emerging evidence suggests that infection with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is associated with a significantly increased risk of thrombotic complications [1]. Although this association is well established, the true incidence of thrombosis in patients with COVID-19 remains difficult to quantify due to the wide spectrum of clinical presentations, ranging from asymptomatic infection to severe critical illness.

More robust data exist regarding thrombotic events among hospitalized patients with COVID-19, suggesting a correlation between disease severity and thrombotic risk. However, many individuals experience mild or no symptoms and may present later, or to different clinical settings after developing thromboembolic complications [2]. As a result, while the relationship between severe COVID-19 and thrombosis is clear, the incidence of thromboembolic disease in asymptomatic or mildly symptomatic patients remains poorly defined. Akel *et al.* described patients who were diagnosed with pulmonary embolism at the time of hospitalization for COVID-19, supporting the association between SARS-CoV-2 infection and an increased risk of thromboembolic complications [3].

Gastrointestinal ischemia represents a rare but severe manifestation of COVID-19-associated thrombosis. Uhlenhopp *et al.* summarized fifteen cases of COVID-19-related ischemic colitis reported across nine publications and described two additional cases. Among the seventeen patients reported, eight died, eight survived, and one outcome was not reported [2]. This highlights both the high mortality and rarity of this complication. Despite these reports, the overall clinical course, management, and outcomes of COVID-19-associated ischemic bowel remain poorly characterized, representing a gap in the current literature.

The underlying pathology of COVID-19-associated thrombosis is not fully understood. Proposed mechanisms include endothelial damage caused by direct viral invasion of endothelial cells, activation of the intravascular complement system and endothelial dysfunction secondary to an exaggerated inflammatory response, commonly referred to as a cytokine storm [4]. Additional contributing factors may include elevated coagulation factors such as factor VIII and fibrinogen, increased blood viscosity, thrombotic microangiopathy, and venous stasis [1] [4].

Recognition of this rare complication is critical given its high morbidity and mortality. We report the case of a patient with COVID-19 who initially presented with generalized weakness and exertional dyspnea and subsequently developed acute ischemic bowel secondary to COVID-19-associated hypercoagulability.

2. Case Presentation

A 67-year-old male with a past medical history of benign prostatic hyperplasia, chronic kidney disease, and anemia with prior transfusions presented to the

emergency department (ED) with several days of generalized weakness and exertional dyspnea. Initial vitals revealed hypotension (BP 87/55 mmHg) and tachycardia (HR 105 bpm). Physical examination was initially unremarkable. Laboratory evaluation demonstrated severe normocytic anemia, acute kidney injury on chronic kidney disease, mild hyponatremia, and metabolic acidosis (Table 1). Urinalysis showed positive nitrites and leukocyte esterase. SARS-CoV-2 polymerase chain reaction (PCR) testing was positive. Chest radiograph and electrocardiogram (ECG) showed no acute abnormalities. The patient received one unit of packed red blood cells in the ED and was being prepared for admission.

Shortly after admission orders were placed, while still in the ED, the patient developed sudden-onset, diffuse abdominal pain accompanied by hemodynamic instability. Computed tomography (CT) of the abdomen and pelvis revealed portal venous gas, gastric pneumatosis, pneumatosis intestinalis, and marked small bowel distension without a discrete obstructing lesion (Figures 1-3). These findings were concerning for acute bowel ischemia. General surgery was emergently consulted, and the patient was taken to the operating room for exploratory laparotomy.

Table 1. Key laboratory findings on admission and prior to long-term acute care transfer.

Parameter	Admission (1/6)	LTC Transfer (1/30)	Reference Range
WBC ($\times 10^3/\mu L$)	6.3	14.7 H	4.0 - 10.0
Neutrophils (%)	84 H	86.9 H	40 - 70
Lymphocytes (%)	6.4 L	6.0 L	20 - 50
RBC ($\times 10^6/\mu L$)	2.62 L	2.59 L	4.2 - 5.4
Hemoglobin (g/dL)	6.2 L	6.8 L	13.5 - 17.5
Hematocrit (%)	20.9 L	21.7 L	41 - 51
MCV (fL)	79.6 L	84.1	80 - 100
MCH (pg)	23.7 L	26.3 L	27 - 33
MCHC (g/dL)	29.8 L	31.3 L	32 - 36
RDW (%)	14.2	16.6 H	11.5 - 14.5
Platelets ($\times 10^3/\mu L$)	223	324	150 - 450
MPV (fL)	7.3	6.4 L	7.5 - 11.5
PT (s)	12.6 H	13.7 H	11 - 13.5
INR	1.11	1.21 H	0.9 - 1.1
aPTT (s)	31.6	33.3	25 - 35
D-dimer (ng/mL)	—	36,767 H	<500

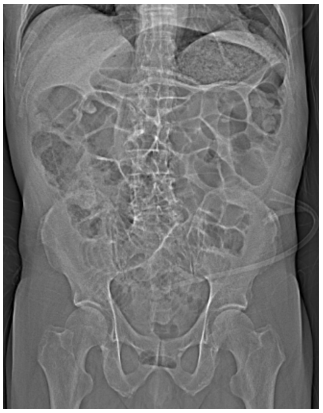

Figure 1. Abdominal radiograph demonstrating diffuse small bowel distention with branching lucencies consistent with portal venous gas and pneumatosis intestinalis, concerning for bowel ischemia.

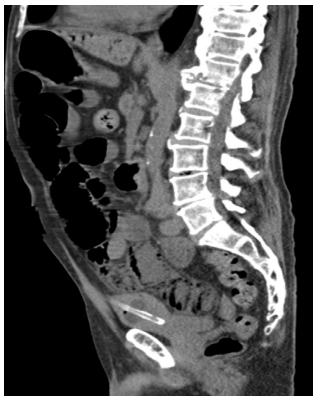

Figure 2. Saggital CT abdomen/pelvis showing dilated small bowel loops with extensive intramural and mesenteric venous gas, suggestive of acute mesenteric ischemia.

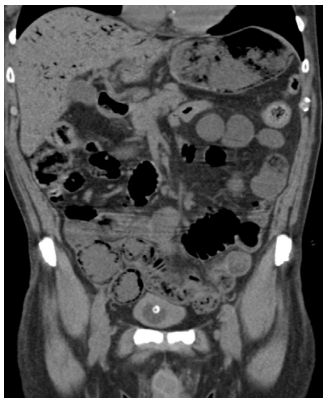

Figure 3. Coronal CT abdomen/pelvis demonstrating multifocal pneumatosis intestinalis with associated portal venous gas and bowel wall thickening, consistent with ischemic bowel.

Intraoperatively, a segment of the mid-jejunum near the ligament of Treitz demonstrated ischemic changes and was resected with healthy margins. Direct

visualization of a thrombus did not occur during surgery; however, the dark red bowel tissue and well-demarcated borders of the resected bowel were consistent with acute thrombosis. Pathologic examination of the resected bowel demonstrated transmural ischemia with thrombotic changes. The initial differential diagnosis for bowel ischemia included chronic kidney disease and anemia, but COVID-19 was ultimately considered the primary contributing factor due to the associated virus-induced hypercoagulable state.

Due to intraoperative hypotension requiring vasopressor support, the abdominal fascia was loosely closed, and the patient was transferred intubated to the intensive care unit (ICU) with plans for re-exploration. On hospital day one, the patient returned to the operating room for evisceration at the surgical site. Small bowel anastomoses were performed, the abdominal fascia was repaired, and a Jackson-Pratt drain was placed.

During his ICU stay, the patient experienced septic shock requiring vasopressors and stress-dose corticosteroids, acute hypoxic respiratory failure necessitating prolonged mechanical ventilation, and worsening renal failure requiring hemodialysis. A percutaneous endoscopic gastrostomy (PEG) tube was placed for nutritional support. Blood cultures grew *Enterococcus faecalis*, and he was treated with broad-spectrum antibiotics per infectious disease recommendations. The patient also developed gastrointestinal bleeding manifesting as melena and recurrent anemia, which was managed with proton pump inhibitor therapy and transfusions as indicated.

Laboratory evaluation revealed progressive leukocytosis, persistent anemia, significant lymphopenia, and reactive thrombocytosis (**Table 1**). Notably, D-dimer measured on hospital day twenty-four was markedly elevated at 36,767 ng/mL, indicating a profound hypercoagulable state consistent with COVID-19-associated coagulopathy. Although early inflammatory and hypercoagulable markers, including D-dimer, fibrinogen, and C-reactive protein, were unavailable, limiting definitive temporal correlation between SARS-CoV-2 infection and thrombotic events, the severity of the thrombotic complication, imaging findings, and markedly elevated D-dimer later in the course strongly support COVID-19 as a major contributing factor. Specific thrombophilia testing was not performed, and other risk factors for ischemic bowel cannot be entirely excluded.

Later during his hospitalization, recurrent low-grade fevers and leukocytosis raised concern for line-associated infection; however, imaging was unrevealing and repeat blood cultures remained negative. Temporary cessation of hemodialysis was possible as the patient's renal function was improving, as evidenced by increasing urine output and down trending creatinine levels. The patient also experienced failed extubation requiring reintubation due to suspected laryngeal edema and subsequently underwent tracheostomy placement. Despite resolution of septic shock and stabilization of infectious parameters, he remained ventilator-dependent with profound deconditioning, protein-calorie malnutrition, and persistent anemia.

One of the main goals of care was establishing an anticoagulation strategy for this patient over the course of his complex treatment. The initial treatment was prompt surgery, but due to the severity of injury, the patient was started on unfractionated heparin to prevent recurrence of bowel ischemia postoperatively. The patient was later transitioned to rivaroxaban.

After prolonged multidisciplinary care, he was deemed medically stable and transferred to a long-term acute care facility with tracheostomy, mechanical ventilation dependence, and enteral feeding. This case highlights the profound prothrombotic effects of COVID-19, which likely contributed to the development of ischemic bowel and significant morbidity.

3. Discussion

This case is noteworthy because it highlights a rare complication of COVID-19 manifesting as ischemic bowel in a patient who initially presented with vague symptoms of weakness and exertional dyspnea. Although COVID-19 is most associated with respiratory manifestations, this case demonstrates the rapid clinical deterioration that can occur secondary to COVID-19-associated hypercoagulability. It underscores the importance of maintaining awareness of thrombotic complications, even in patients without severe symptoms, and highlights the complex management required once ischemic complications develop.

COVID-19 is gradually being recognized as a systemic prothrombotic condition, with reported complications including venous thromboembolism, arterial thrombosis, stroke, and microvascular ischemia [5]. Proposed mechanisms include endothelial injury mediated by viral invasion, activation of inflammatory and complement pathways, and elevations in procoagulant factors such as fibrinogen and factor VIII [4] [5]. While thrombotic events are most reported among critically ill patients, emerging evidence suggests that patients with mild or atypical presentations may still be at risk [6]. Gastrointestinal ischemia remains a rare manifestation, but reported cases demonstrate high morbidity and mortality, emphasizing the severity of this complication [2].

Despite growing recognition of COVID-19-associated thrombosis, challenges remain in the early diagnosis of gastrointestinal ischemia due to nonspecific symptoms and overlapping clinical features with more common causes of abdominal pain, weakness, and abnormal lab findings. This diagnostic ambiguity may result in delays in imaging, intervention, and escalation of care, thereby worsening outcomes. The rarity of reported cases further limits clear guidance regarding optimal screening, monitoring, and management strategies.

This case contributes to the existing literature by illustrating that catastrophic gastrointestinal ischemia can occur in patients with initially vague symptoms and minimal respiratory involvement. Unlike many previously reported cases involving critically ill patients, this presentation suggests that clinicians should maintain a broad differential diagnosis even in less severe presentations of COVID-19. These findings expand the current understanding of the clinical spectrum of

COVID-19-associated thrombosis and support earlier diagnostic evaluation in patients presenting with limited respiratory symptoms and nonspecific weakness.

Furthermore, this case highlights the potential for rapid thrombotic progression even in the absence of severe respiratory compromise, reinforcing emerging evidence that COVID-19-induced hypercoagulability may manifest unpredictably across organ systems. Though the onset was more acute, the outcome mirrors that of previously reported COVID-19-associated bowel ischemia, a condition that continues to demonstrate poor prognosis across the existing literature.

Clinicians should therefore maintain heightened awareness of thrombotic complications in patients with SARS-CoV-2 infection, particularly those presenting with nonspecific symptoms or unexplained clinical deterioration. Early recognition and appropriate intervention may improve outcomes by prompting earlier imaging, anticoagulation consideration, and surgical consultation when indicated. Initial evaluation may include expanded laboratory workups to include inflammatory and hypercoagulable markers, such as D-dimer, fibrinogen, and C-reactive protein to assist in risk stratification and clinical decision-making.

4. Conclusion

This case demonstrates that ischemic bowel is a rare but serious complication of COVID-19-associated hypercoagulability, which can occur even in patients presenting with mild or nonspecific symptoms. Clinicians should maintain a high index of suspicion for thrombotic complications in patients with SARS-CoV-2 infection, particularly when unexplained weakness, abdominal symptoms, or abnormal lab findings are present. Early recognition, prompt diagnostic evaluation, and timely intervention may improve outcomes. This case also highlights the need for further research to better define risk factors, screening strategies, and management approaches for rare thrombotic complications of COVID-19.

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Conflicts of Interest

The author declares no conflicts of interest regarding the publication of this paper.

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