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Managing an Acute Bowel Ischemia as a Complication of a Covid-19 Infection. A Case Reports

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Abstract

Introduction*: There is enough evidence suggesting that severe COVID-19 infection is associated with a high rate of thrombotic complications. A proportion of such patients may present with a bowel ischemia which differs in some aspects from non-COVID-19 patients and may be a challenge for the surgeon.*

Patients and Methods: *We describe a case of a 63-year-old man with a confirmed COVID-19 infection, who entered the ICU due to acute severe respiratory distress syndrome. The patient gradually showed clinical improvement. Unexpectedly, he developed an acute abdomen. Abdominal computed tomography (CT) imaging combined with Computed Tomographic Angiography (CTA) revealed hypoperfused segments of small bowel with patent main mesenteric vessels, although he was already on full-dose anticoagulant therapy with LMWH (enoxaparin).*

***Results**: He was operated on an urgent basis and patchy bowel necrosis was found. An extensive small bowel resection with an end-to-end anastomosis was performed. The patient succumbed on the second postoperative day due to multiorgan failure*

***Conclusion:** Although acute mesenteric ischemia is a rare complication of COVID-19 infection, surgeons should be vigilant of this entity, even in cases where the main vessels present as patent, because of the catastrophic consequences of an undetected or late diagnosis. Urgent surgical exploration should be considered without any hesitation.*

***Keywords:** Acute mesenteric ischemia, COVID-19 complications, small vessels thrombosis*

Introduction

Although the lung is the most prominent organ to be first insulted in COVID-19 patients with respiratory failure, there is growing evidence that several other organs end up suffering from secondary micro - and macro -thrombotic complications, due to virus-induced coagulopathy [1,2,3]. Thus, coagulation test screening including the measurement of D-dimer levels, fibrinogen levels, PT and aPTT have been suggested. Consistently elevated D-dimer, as well as fibrinogen levels, have been associated with higher mortality rates and poor outcome, while anticoagulant therapy appears to confer a better prognosis [4,5,6,7,8]. Nevertheless, numerous reports indicate a significant number of venous thromboembolic events despite thromboprophylaxis, whilst the optimal dose and duration of therapy is unclear [9,10].

Bowel ischemia has been reported in a single-center study of 141 COVID-19 patients among which, 4 developed intestinal ischemia attributed to small vessel thrombosis related to COVID-19 infection. There have also been reported cases of superior mesenteric, portal vein and celiac artery thrombosis [11,12,13,14].

We report a fatal case of acute bowel ischemia due to microvascular thrombosis, in a 63-year-old man with confirmed COVID-19, while treated with full-dose anticoagulant therapy with LMWH (enoxaparin).

Patient and Method

A 63-year-old man with no comorbidities and a recent travel history to Great Britain was brought to the emergency department with high fever (up to 39 °C) and dyspnoea. The patient reported fever, dry cough, and diarrhea during the last seven days. He was diagnosed with COVID-19 via a positive Reverse transcription- polymerase chain reaction (RT-PCR) nasopharyngeal swab test and was admitted to the internal medicine department, where he was treated in isolation.

Clinical findings, as well as chest imaging, indicated severe SARS- CoV-2 infection with diffuse, bilateral patchy ground-glass opacities and pleural effusions. His initial laboratory studies revealed lymphopenia of 0.5 K/μL, elevated D-dimer levels of 6mg/L (reference range 0-0.5 mg/L), high fibrinogen levels of 711 mg/ dl (reference range 200-400 mg/dl) and elevated CRP.

He was put on Piperacillin/Tazobactam in addition to azithromycin, hydroxychloroquine, and lopinavir/ritonavir. On the third day after admission, his condition deteriorated with severe hypoxia for which he was intubated, and transferred to the ICU.

He remained in critical condition for several days with severe respiratory distress syndrome. Meanwhile, he received tocilizumab after completing a 10-day therapy with azithromycin and a 14-day therapy with hydroxychloroquine and lopinavir/ritonavir. In the meantime, he presented with multiple episodes of paroxysmal atrial fibrillation (AF), for which was effectively treated either with esmolol or amiodarone. Thus, from the very early beginning of his stay in the ICU, he started receiving full-dose anticoagulant treatment with LMWH (enoxaparin). Repeated bedside examinations with Transthoracic Echocardiography (TTE) showed no structural or functional heart abnormalities.

Furthermore, it is worth noting that the patient underwent a septic shock followed by multi-organ failure on the fifteenth day of his stay in the ICU, which was correlated to Ventilator-Associated Pneumonia (VAP) due to A. Baumanii PDR. He was treated effectively with targeted antibiotic therapy and hydrocortisone according to protocol.

On his recovery from the shock, he was kept on glucocorticoid therapy (hydrocortisone 200mg/d), and low doses of levothyroxine due to concurrently presented hypothyroidism. The patient gradually made some progress concerning his hemodynamic stability and weaning from mechanical ventilation. Also, a sample of tracheal aspirate was tested negative for SARS -CoV-2 via Reverse transcription- polymerase chain reaction (RT- PCR), with zero viral load.

Nevertheless, the patient abruptly presented with signs of peritoneal irritation, abdominal distention and tenderness. He was soon shocked and needed high-dose vasopressors. The CT scan and Computed Tomographic Angiography (CTA) revealed areas of small bowel hypoperfusion together with intramural pneumatosis (Fig.1), without major vessel embolism or thrombosis (Fig.2). Laboratory analysis at that time revealed leukocytosis of 19.1K/μL, elevated platelet count of 574K/μL, and further elevation of D-dimer levels up to 8mg/dl.

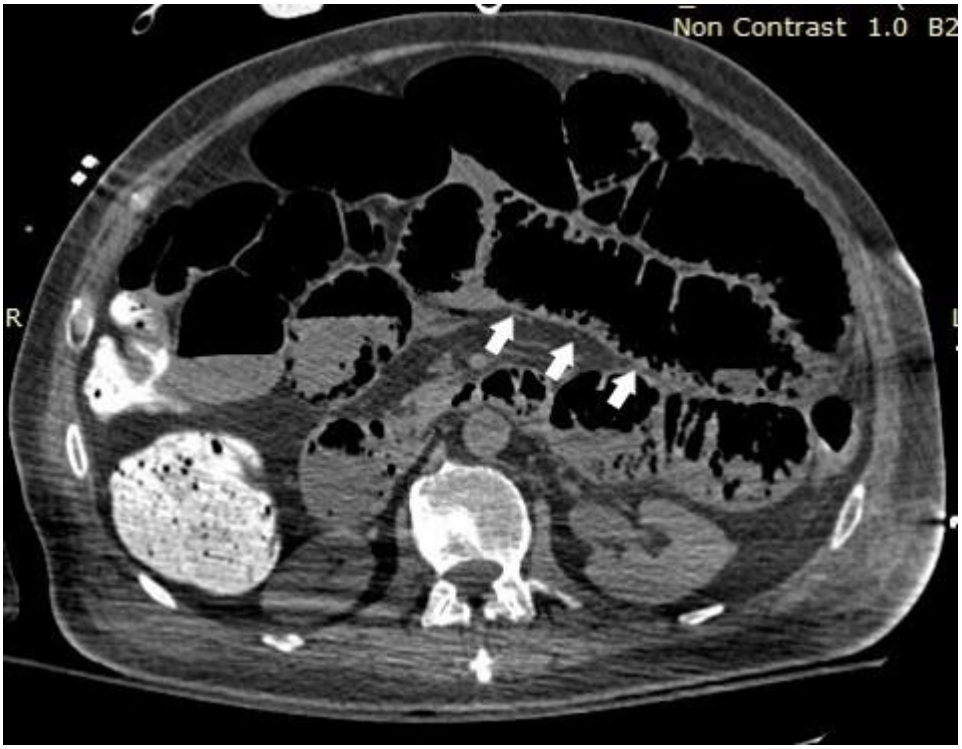

Figure 1. CTA showing areas of small bowel hypoperfusion together with intramural pneumatosis (white arrows).

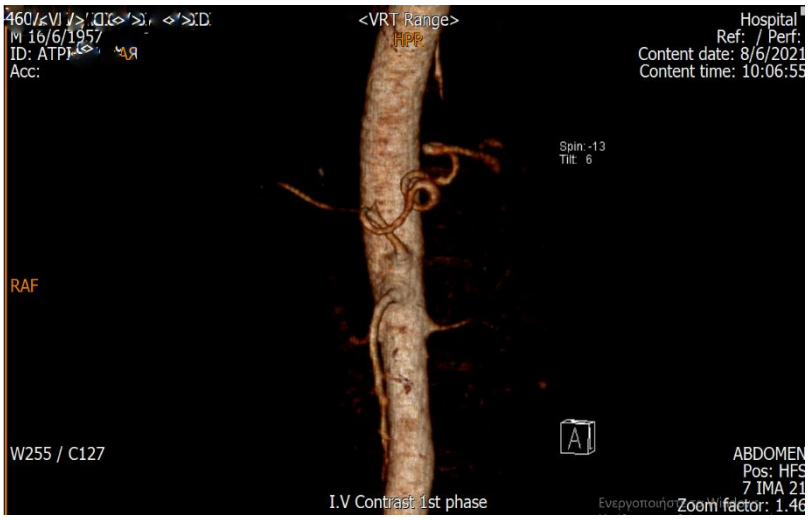

Figure 2. CTA 3d reconstruction depicting patent major vessel, without embolism or thrombosis.

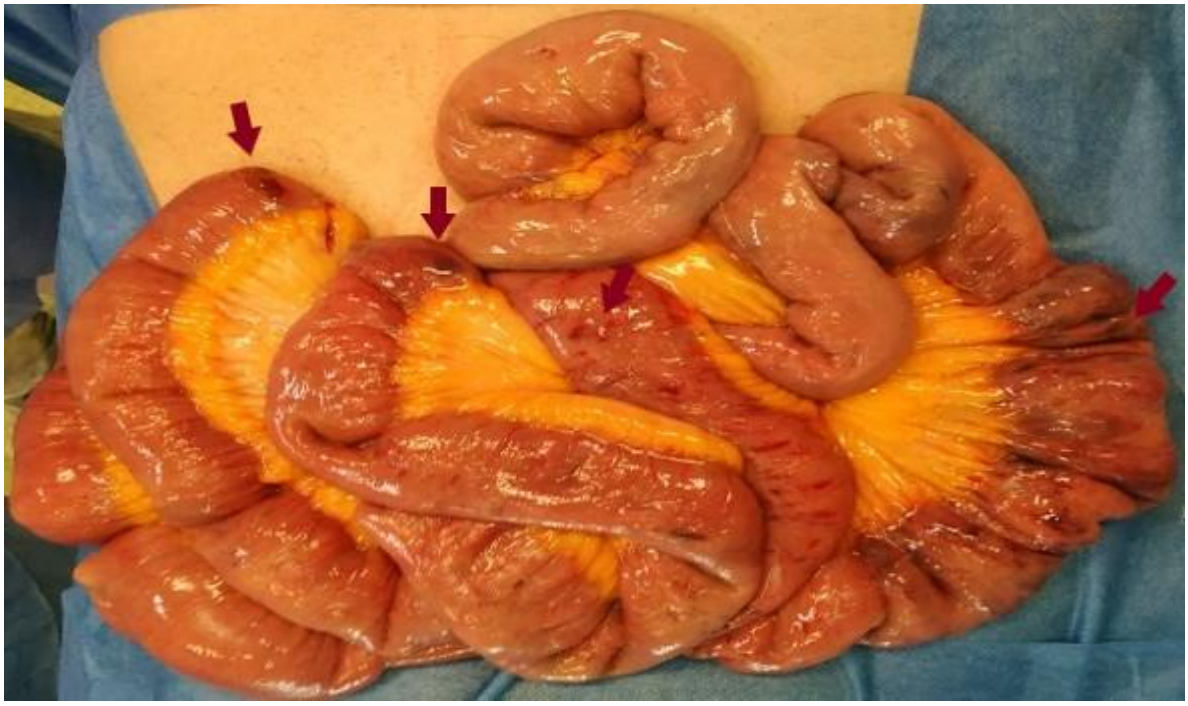

Figure 3. Circumferential and patchy ischaemic areas in the small bowel with a dark red-blue hue, indicative of peripheral microvascular thrombosis of distal jejunum and ileum (black arrows).

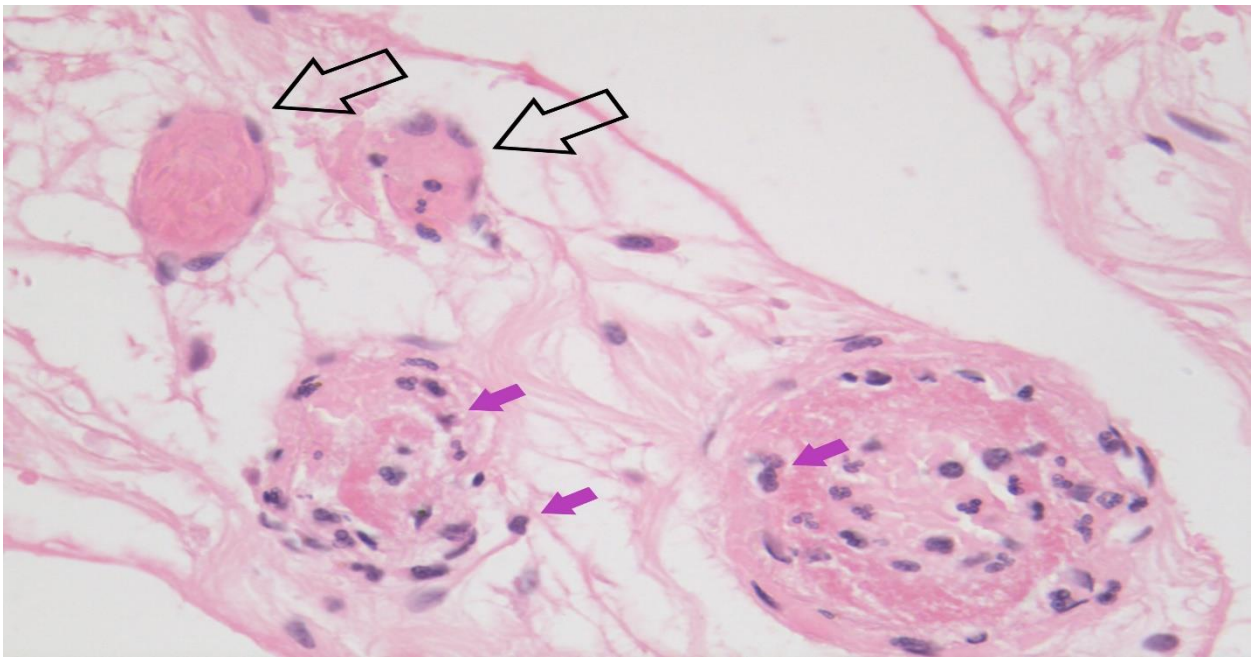

Figure 4 A small vessels emboli (white arrows), b. mononuclear cell infiltration in the wall and lumen of small vessels (black arrows).

We decided to proceed with an exploratory laparotomy, which revealed both circumferential and patchy ischaemic areas in the small bowel with a dark red-blue

hue, indicative of peripheral microvascular thrombosis of distal jejunum and ileum (Fig.3). An extensive resection of the small bowel together with a jejunostomy and ileostomy was performed. Despite postoperative intensive care, the patient rapidly decompensated, developed multiorgan failure and succumbed 48h later.

Histopathological examination of the resected small intestine, revealed sites of small blood vessels emboli and mononuclear cell infiltration in the wall and lumen of small vessels, together with early thrombosis (Fig.4).

Discussion

Thrombotic events have been recognized as a major complication and one of the less frequent causes of death in patients with severe COVID-19 infection [15].

Our case refers to acute segmental intestinal ischemia in the context of microvascular thrombosis, despite the fact that our patient was fully anticoagulated with LMWH since his admission to the ICU, due to episodes of paroxysmal AF.

Intestinal ischemia is caused by blood flow insufficiency, resulting in a supply-demand mismatch in visceral organs. The most common causes of acute mesenteric ischemia are a) arterial embolism in 40-50 % of cases, b) arterial thrombosis from ruptured atheroma in a previously stenotic mesenteric vessel in 20-35% of cases c) arterial dissection in < 5% of cases d) non-occlusive mesenteric ischemia-hypoperfusion in 10-15% of cases and e) venous infarction in 5-15% of cases [16,17].

Despite the fact that our patient had episodes of AF, embolic occlusion was not documented in CTA of the abdomen, or intraoperatively. Also, a major vessel thrombotic occlusion, dissection, or venous mesenteric thrombosis was not found.

Our patient was also not in shock or low flow state from any cause, and he was not receiving any vasopressors prior to the acute event, so the ischemic necrosis cannot be attributed to mesenteric hypoperfusion.

Impaired coagulation mechanism due to COVID-19 infection was highly suspected as the cause of microvascular thrombosis in our patient, based on markedly elevated D-dimer levels, despite the fact that prothrombin time, activated partial thromboplastin time and platelet count remained within the normal range. It is worth mentioning that D-Dimer levels were never normalized throughout his ICU stay, even though he was under full-dose anticoagulation therapy with LMWH.

There is an emerging body of evidence recognizing predisposition of severe COVID-19 patients to thrombotic disease [18,19].

According to available data, except for blood flow stasis due to prolonged immobility, and patient-associated risk factors, the underlying etiology of arterial thrombosis and thrombotic microangiopathy in severe COVID-19 cases may be related to a prothrombotic state in the context of hyperinflammatory reaction and "cytokine storm" [20,21].

Previous data have established a synergistic relationship between inflammation and pro-coagulant changes, by means of disruption of normal antithrombotic and anti-inflammatory endothelial function, leading to dysregulation of the coagulation process, microvascular thrombosis, and tissue damage [22].

There are also alternative views considering the vascular complications in this disease setting. According to them, direct viral infection of the endothelial cell and diffuse endothelial inflammation underlines the pathogenesis of vascular complications in severe COVID-19 patients. The resulting endothelial dysfunction causes more vasoconstriction, organ ischemia, oedema and a procoagulant state [23].

It has been also postulated that even autoimmune mechanisms may be involved, based on the presence of antiphospholipid antibodies, most commonly lupus anticoagulant (LA) in some cases of severe COVID-19 patients [24,25].

Although DIC (disseminated intravascular coagulation) and SIC (sepsis-induced coagulopathy) are frequently identified (both or individually) in severe COVID-19 as the underlying pathophysiological mechanisms for thrombosis, this is not always the case. Many patients, including ours, often sustain thrombotic events without concomitant DIC or SIC laboratory profile [26,27].

The most frequent GI symptoms in non-critically ill COVID-19 patients are diarrhea, nausea, vomiting and abdominal pain. In this subgroup, imaging may reveal distended fluid-filled bowel loops with post-contrast enhancement and surrounding fat stranding, due to mesenteric inflammation. In cases with bowel ischemia, the most significant imaging findings include bowel wall unenhancenent, bowel wall thickening, pneumatosis intestinalis and portal venous gas. Macrovascular arterial or venous thrombosis is identified in nearly half of COVID-19 patients with bowel ischemia [28]. That means that almost half of patients with this devastating complication, as was our case, may present with patent main vessels in imaging preoperative investigation.

Conclusion

Intestinal ischemia may complicate the course of critically ill patients with a COVID-19 infection. Although this may be the result of major vessel occlusion there are cases, such as this presented herein, that happens although the main mesenteric vessels appear to be patent. This may cause subtle

symptoms in the initial phase, heralding the need of a high level of suspicion and alert from the surgeon that may be invited for a consultation.

Any critically ill patient with a suspicion of bowel ischemia and patent mesenteric vessels, may need early surgical intervention, as this complication is invariably fatal if left untreated during its initial phase.

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