



Acute Ischemic Colitis in a Covid-19 Patient

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Received date: 08 January 2021; **Accepted date:** 16 February 2021; **Published date:** 24 February 2021

Citation: Urciuoli L, Guerriero E. Acute Ischemic Colitis in a Covid-19 Patient. Asp Biomed Clin Case Rep. 2021 Feb 24;4(1):60-65.

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Abstract

Coronavirus disease 2019 (COVID-19) is caused by a novel strain of coronavirus called severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), which was first discovered in Wuhan, China, in December 2019. The virus has quickly spread around the globe and has been declared a global pandemic by the World Health Organization.

Although in COVID-19 respiratory symptoms predominate, there is a relevant body of evidence that initial symptoms and complications of COVID-19 are not limited to the pulmonary system, but can involve also the gastrointestinal tract.

We report a case that presented with ischemic colitis in the setting of PCR-confirmed SARS-CoV-2 infection.

Keywords

COVID-19, Computed Tomography, Pneumonia, Acute Ischemic Colitis

Case Report

A 75-year-old woman, with a past medical history of hyperlipidemia and diabetes mellitus type 2, without previous usage of antibiotic/probiotic/natural products, presented to the emergency department (ED) with abdominal pain, diarrhea and rectal bleeding. She also had a 3-day history of fever, dry cough, and dyspnea.

At the initial presentation, she presented fever (39°C), tachycardia (110 bpm), and oxygen saturation of 89% at rest in room air.

Physical examination was remarkable for tenderness over the lower quadrant regions bilaterally but otherwise, there was a non-distended abdomen, no guarding, and no rebound tenderness. Coarse breath

sounds at the bases of both lungs were heard on auscultation.

The laboratory tests revealed low hemoglobin 10 g/dL (13.5-17.5 g/dL), leucopenia $4 \times 10^3/\mu\text{L}$ ($4.4-11 \times 10^3/\mu\text{L}$), lymphopenia with an absolute lymphocyte count of $700/\mu\text{L}$ ($900-2900/\mu\text{L}$), normal level of lactate dehydrogenase (LDH) 180 U/L (122-222 U/L) and elevated inflammatory markers, such as C-reactive protein (CRP) 8 mg/dL (0-0.8 mg/dL), procalcitonin 2 ng/ml (0-0.5 ng/mL), D-dimer 3.000 ng/mL (0-500 ng/mL) and ferritin 600 ng/mL (24-336 ng/mL); the prothrombin time was 14 s (9.9-13 s), and the INR was 1.3 (0.9-1.1). Stool test (stool culture) was negative for bacterial infections.

Because of her respiratory condition, real-time

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reverse transcription polymerase chain reaction (RT-PCR) was performed on the patient's nasal swab sample and it was positive for SARS-CoV-2. An enhanced Computed Tomography (CT) was performed.

Chest CT showed bilateral ground-glass opacities

(GGOs) with superimposed interlobular thickening (crazy-paving pattern) with both peripheral and central distribution, especially located in the left upper lobe, in the middle lobe, and in lower lobes (**Fig-1a**, **Fig-1b** and **Fig-1c**). No pleural and pericardiac effusions were found.

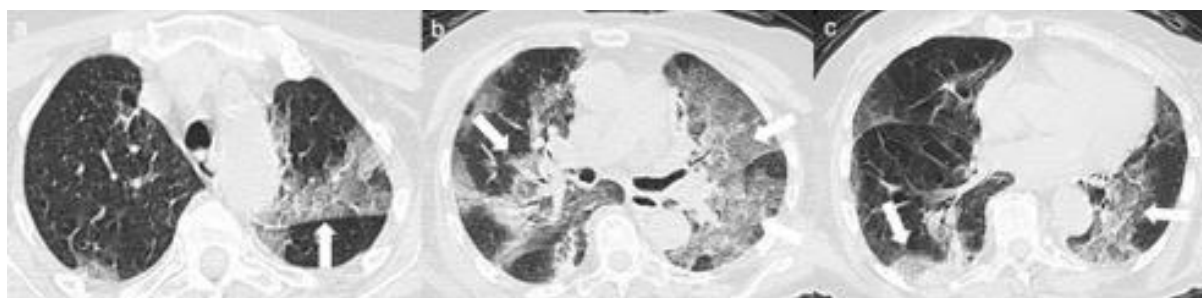


Fig-1:

CT shows bilateral ground glass opacities with superimposed interlobular thickening (crazy paving pattern) with both peripheral and central distribution, especially located in left upper lobe (arrow in a), in middle lobe and in lower lobes (arrows in b, c).

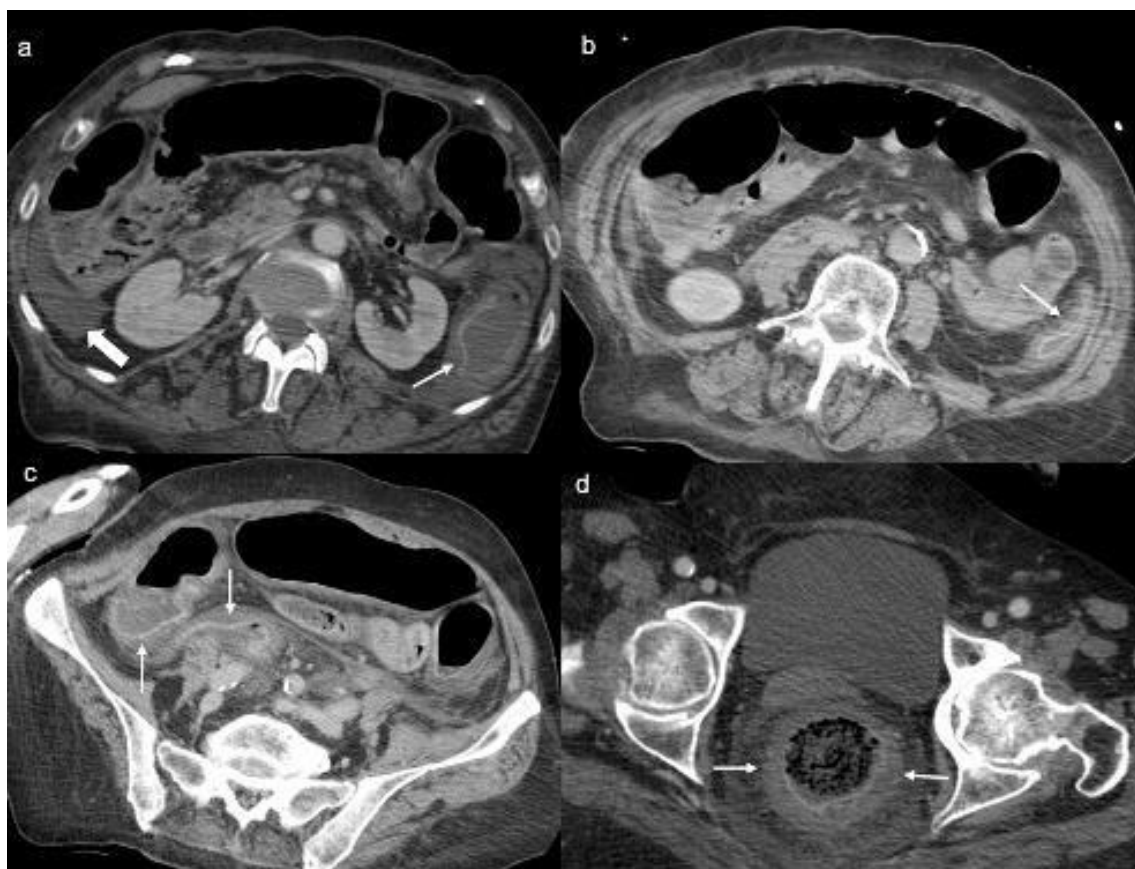


Fig-2:

CT shows a segmental colonic wall thickening, with low-density ring of submucosal edema between enhancing mucosa and serosa (target sign), involving the left colon, the cecum and the recto-sigmoid junction (thin arrows in a, b, c, d). A slight pericolic inflammation is detected in the high rectum (thin arrow in d). There is free peritoneal fluid was in the right lateroconal space (arrow in a).

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Abdominal scans showed a segmental colonic wall thickening, with a low-density ring of submucosal edema between enhancing mucosa and serosa (target sign), involving the splenic flexure, the left colon, the cecum the recto-sigmoid junction (Fig-2a, Fig-2b, Fig-2c, Fig-2d and Fig-4). A slight pericolic inflammation was detected, especially in the high rectum (Fig-2d). The superior and inferior mesenteric

arteries were patent (Fig-3a and Fig-3b). Free peritoneal fluid was detected, especially in the right lateroconal space (Fig-2a, Fig-3a). Infarction of the spleen's inferior pole was also present (Fig-4). No other parenchymal infarctions or extraluminal gas or abnormalities in the small bowel were detected. The radiological findings were consistent with non-occlusive acute ischemic colitis.

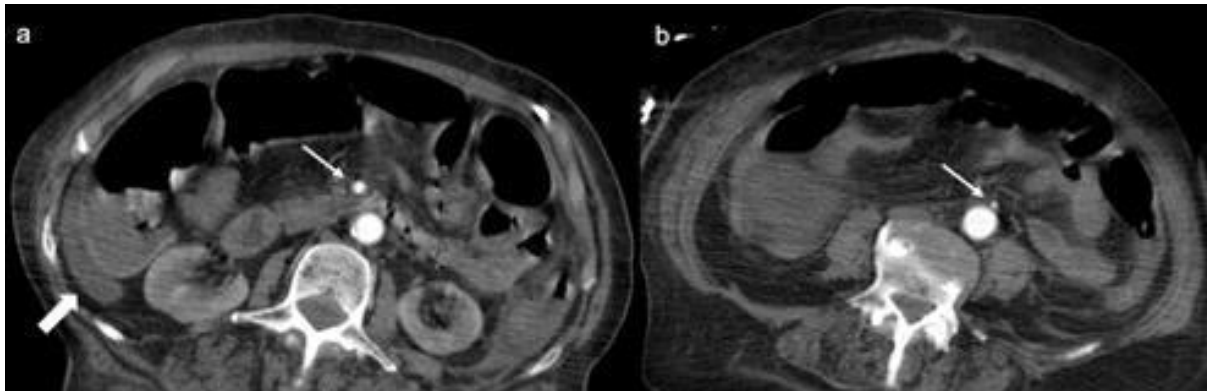


Fig-3:

CT shows patency of the superior and inferior mesenteric arteries (thin arrows in a, b). There is free peritoneal fluid in the right lateroconal space (arrow in a).

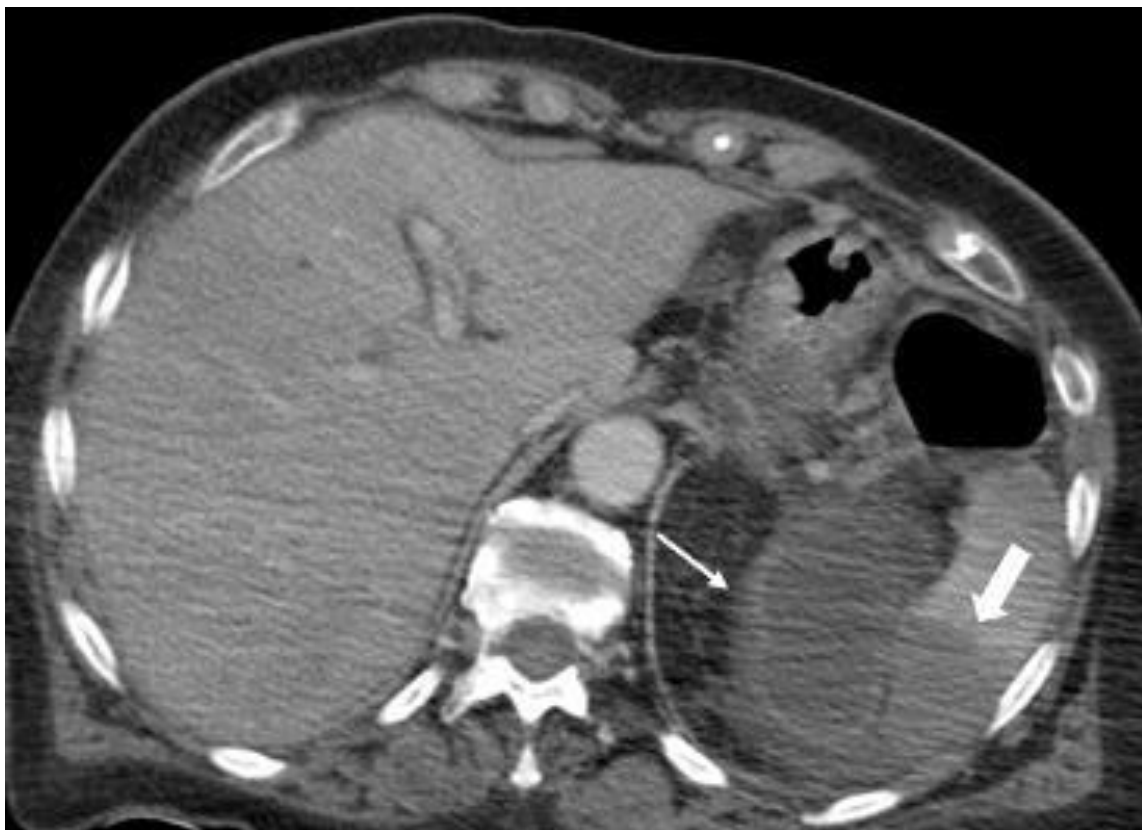


Fig-4:

CT shows infarction of the spleen's inferior pole (arrow). There is CT a colonic wall thickening, with low-density ring of submucosal edema between enhancing mucosa and serosa (target sign), involving the splenic flexure (thin arrow).

The patient was initially managed conservatively with bowel rest and intravenous fluids. Intravenous antibiotics and therapeutic Low-molecular-weight heparin (LMWH) anticoagulation were started. She died following cardiorespiratory deterioration due to COVID-19 pneumonia.

Discussion

Coronavirus disease 2019 (COVID-19) is caused by a novel strain of coronavirus called severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) [1,2].

COVID-19 has a wide range of clinical presentations, with symptoms ranging from asymptomatic to rapid multiple organ dysfunction syndromes, and has a high mortality.

Although in COVID-19 respiratory symptoms predominate, there is a relevant body of evidence that initial symptoms of COVID-19 are not limited to the pulmonary system. Gastrointestinal symptoms such as abdominal pain, nausea, vomiting, non-bloody diarrhea, and transaminitis have been identified as part of the disease spectrum. Some case series have noted that 3% to 10% of COVID-19 patients initially present with isolated gastrointestinal symptoms [3,4].

SARS-CoV-2 has been linked to the development of coagulopathy and thromboembolic complications in severe COVID-19 patients. Both arterial and venous thrombosis can occur with COVID-19. Arterial thrombosis reported so far include stroke, acute limb ischemia, acute coronary syndrome, and rarely ischemic colitis [5-7].

The exact pathological mechanism leading to the complication of acute ischemic colitis in COVID-19 is not well understood at present.

SARS-CoV-2 has a great affinity for the membrane receptors of the angiotensin-converting enzyme 2 (ACE-2) present in type-II alveolar cells of the lung (AT2), the enterocytes of the ileum and colon, and the cholangiocytes and B-cells of the pancreas.

Given that the intestine is the largest immune organ

and expresses abundant ACE-2 receptors for SARS-CoV-2, it is not at all surprising that a severe inflammatory response occurs with damage to the mucosa and necrosis of the intestinal wall [8].

Furthermore, the systemic inflammatory response and sepsis are associated with thrombotic phenomena and disseminated intravascular coagulation, characterized by the massive presence of fibrin deposits, a marked elevation of D-dimer levels, and moderately low platelet counts.

It remains still unclear if these coagulation disorders are directly induced by SARS-CoV-2 or are secondary to the systemic inflammatory response [9,10].

Ischemic colitis, although the most common ischemic pathology of the Gastrointestinal (GI) tract, is relatively rare. It can be classified as non-occlusive (most common) or occlusive. Given the patent vasculature, non-occlusive ischemic colitis is favored, which is most commonly due to a low flow state resulting in transient ischemia, often due to other medical conditions such as heart disease, hypotensive episodes, surgery, myocardial infarction, arrhythmia, vasculitis, and thrombophilia. Some common but less specific symptoms include painless diarrhea, nausea, or vomiting.

On physical examination, varying degree of tenderness is elicited over the affected region, depending on the extent of colonocyte damage; even signs of peritonitis, Systemic Inflammatory Response Syndrome (sepsis, tachypnea, and tachycardia) may be evident, along with augmenting laboratory findings, such as leukocytosis, metabolic acidosis, elevation of lactate. Computed tomography and endoscopic finding are crucial for a prompt diagnosis [11,12].

To date, this vascular disorder of the GI tract has been poorly described in the setting of COVID-19 infection.

Chan et al [13] were the first to report a case of SARS-CoV-2 infection causing ischemic colitis with high D-dimer levels and inflammatory markers, together with clinical and radiographic findings of

ischemic colitis.

Similarly, Paul et al. [14] reported a case of severe COVID-19 pneumonia, developing ischemic colitis.

Almeida et al. [15] described the cases of three males that presented with respiratory distress and colonic ischemia.

Lazaro et al. [16] reported a case of a 53-year-old man with a medical history of type 2 diabetes, and hypercholesterolemia, with ischemic colitis as first manifestation of infection of COVID 19.

Singh et al. [17] described a case of an 82-year-old female presenting with fever and cough and who was diagnosed with COVID-19 and subsequently developed abdominal distention, tenderness, and underwent emergent laparotomy and was found to have a gangrenous ascending colon and markedly distended colon from the cecum to rectosigmoid junction.

Considering the above, our case confirms that we should be wary of the fact that COVID-19 patients can present with acute ischemic colitis.

We report a case of coincident presentation of COVID-19 pneumonia, acute ischemic colitis and splenic infarction. Our CT findings were very suggestive for a severe form of Covid-19 pneumonia [18,19], while abdominal scans showed an unsuspected scenario: non-occlusive mesenteric ischemia involving the large bowel.

The present case adds to the literature regarding the extrapulmonary complications of COVID-9 and underlines the need for further reports regarding under-recognized and life-threatening complications in this infection.

Author Contributions

L.U. and E.G. contributed equally to the design of the research, to the analysis and interpretation of the patient data, and to the writing of the manuscript.

Funding

This research received no external funding.

Conflicts of Interest

All authors have read and approved the final version of the manuscript. The authors have no conflicts of interest to declare.

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