

# COVID-19: Abdominal and Pelvic Imaging Findings: A Primer for Radiologists

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**Abstract:** The COVID-19 pandemic presents an ongoing global health threat. The SARS-CoV-2 is known to cause substantial pulmonary disease, and most of the current radiological publications are dedicated to describing and characterizing these findings. However, studies regarding imaging findings in the abdomen and pelvis of infected patients are still very limited. The aim of this review is to discuss the most frequent abdominal manifestations based on the current literature and representative images from our local experience.

**Key Words:** COVID-19, SARS-CoV-2, abdominal imaging, multidetector computed tomography, mesenteric ischemia, SIRS

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The coronavirus disease 2019 (COVID-19) pandemic, caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), remains a global health challenge. The rapid and extensive spread of the disease, combined with acute respiratory failure and associated multisystemic complications, accounts for a major cause of morbidity and mortality. As of August 2020, the World Health Organization has reported over 23 million confirmed cases and more than 800,000 deaths worldwide.<sup>1</sup>

Much research, effort, and resources has been dedicated to studying the different manifestations of the COVID-19 infection. This has allowed a better understanding of the pathophysiology and the injury mechanisms of the virus, as well as a detailed characterization of respiratory and pulmonary complications.

Common clinical features may vary according to geographical location, age, comorbidities, and other patient variables. However, the most frequently reported symptoms are cough (50% cases), fever (43%), myalgia (36%), and headache (34%).<sup>2,3</sup>

As testing capacity and case numbers have increased, there has also been a substantial increase in reports of extrapulmonary and multisystemic manifestations of COVID-19. However, there are still few studies describing abdominal complications in COVID-19 infection, and even fewer focused on the imaging findings of these complications. This is likely due to the fact that the link between SARS-CoV-2 and abdominal complications has only recently been recognized.<sup>3–5</sup>

Gastrointestinal symptoms have been described in 18% to 34% of patients with COVID-19,<sup>3–5</sup> the most common being diarrhea, nausea/vomiting, and abdominal pain.<sup>6–9</sup> Bhayana et al<sup>5</sup> described that the most frequent findings in abdominal multidetector computed tomography (MDCT) of patients with COVID-19 were

abnormalities in the intestinal wall (31%), solid organ infarctions (4.8%), and pancreatitis (2.4%). These extrapulmonary complications significantly increase the risk of morbidity and mortality. Furthermore, it is thought that the viral spread to other organs usually occurs in the second week of the disease course, which correlates with more severe clinical features and higher mortality.<sup>10,11</sup>

This article reviews the updated evidence on some of the most frequent abdominal and pelvic complications in COVID-19, based on the current literature and representative images from our local experience.

## Pathophysiology

The mechanisms involved in the development of thrombotic complications and multiorgan failure caused by SARS-CoV-2 remain under study. The virus has been shown to enter the cellular epithelium of the lung by binding to the angiotensin-converting enzyme 2 (ACE2) receptor.<sup>12,13</sup> This receptor is expressed at high levels in the bile epithelium,<sup>13</sup> pancreatic islet cells,<sup>10</sup> and cells of the gastrointestinal epithelium.<sup>6</sup>

Recent studies propose 4 key mechanisms that may play a role in the pathophysiology of multiorgan injury caused by infection with SARS-CoV-2. These include direct viral toxicity, endothelial cell damage and thromboinflammation, dysregulation of the immune response, and dysregulation of the renin-angiotensin-aldosterone system.<sup>14</sup> In addition, it is thought that the expression of ACE2 could stimulate a direct cytotoxic effect, allowing local infection and viral replication to take place in the gastrointestinal tract.<sup>6,15,16</sup> Furthermore, the virus has been found in human feces in significant proportions, which would support the theory of local infection.<sup>17,18</sup>

New evidence suggests that oxidative stress may also participate in the pathogenesis of COVID-19 by perpetuating the cytokine storm cycle, blood clotting mechanisms, and exacerbating hypoxia, sustaining and worsening direct tissue injury produced by the virus.<sup>19</sup>

Further studies are required for a better understanding of the implications of each of these mechanisms of injury in abdominal organs. However, in the next lines, we present the most common organ-specific manifestations of the disease and gather existing evidence on how some of the mentioned mechanisms of injury have been proven to affect abdominal organs.

## Intestinal Abnormalities

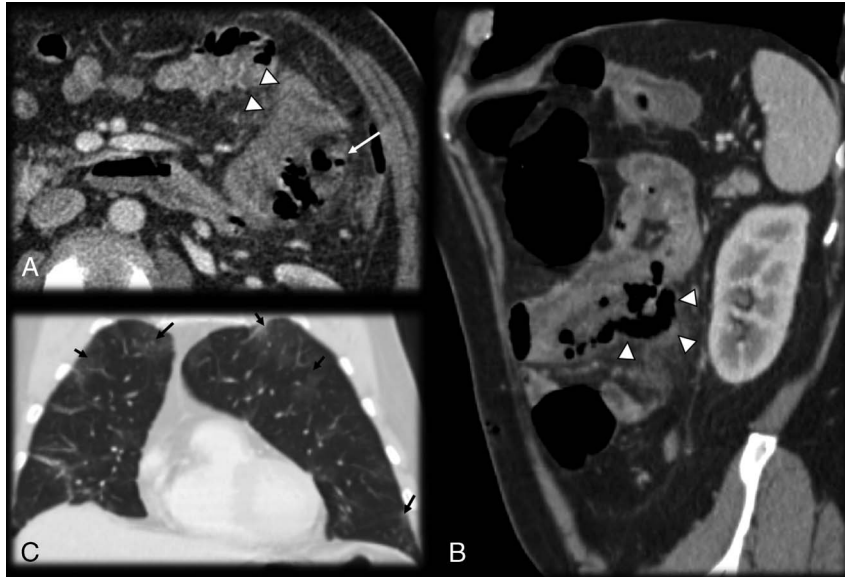
Intestinal abnormalities are not a frequently reported complication of COVID-19; however, some reports suggest an association between intensive care unit admission and bowel wall abnormalities.<sup>5</sup> The diagnosis and characterization of these abnormalities are possible through imaging studies. Some of the most frequently reported findings in MDCT studies include thickening and pneumatosis of the intestinal wall, gas in the portal-venous system (portal pneumatosis), and wall enhancement abnormalities (Figs. 1, 2).<sup>5,20,21</sup>

Some of these intestinal complications in patients with COVID-19 could be caused by the development of in situ thrombosis

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**FIGURE 1.** Mesenteric ischemia in a 69-year-old man with COVID-19. Axial (A) and sagittal (B) reconstructions of a contrast-enhanced CT. Segmental wall thickening and pneumatosis on a nonenhancing segment of the jejunum (white arrow heads). A defect in the lateral wall of the jejunum and adjacent localized collection of intraperitoneal free gas [white arrow in (A)] are in keeping with perforation. C, Multifocal peripheral and bilateral areas of ground-glass opacity and consolidations (black arrows).

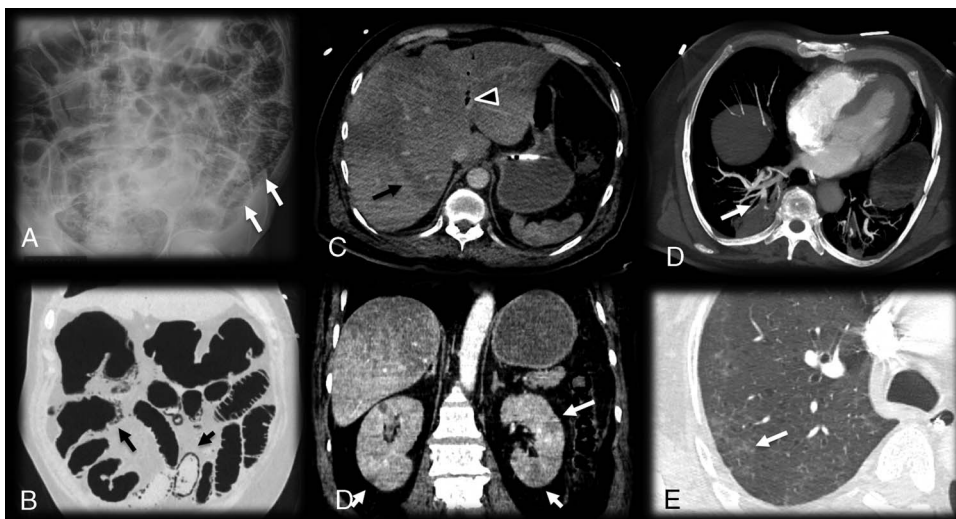
in small vessels, direct viral infection, or nonocclusive mesenteric ischemia (possibly due to systemic global hypoperfusion).<sup>14,15,22</sup> Systemic coagulopathy is also a frequent finding in patients with severe COVID-19 infection and could be the cause of some of the intestinal ischemic complications.<sup>10,20</sup>

Of the reported intestinal abnormalities in patients with COVID-19, acute intestinal ischemia is one of the most common and severe. Limited published data are available on this complication; however, it has been reported that up to 20% of MDCT images in intensive care unit patients could present findings of advanced (late) mesenteric ischemia, such as portal pneumatosis

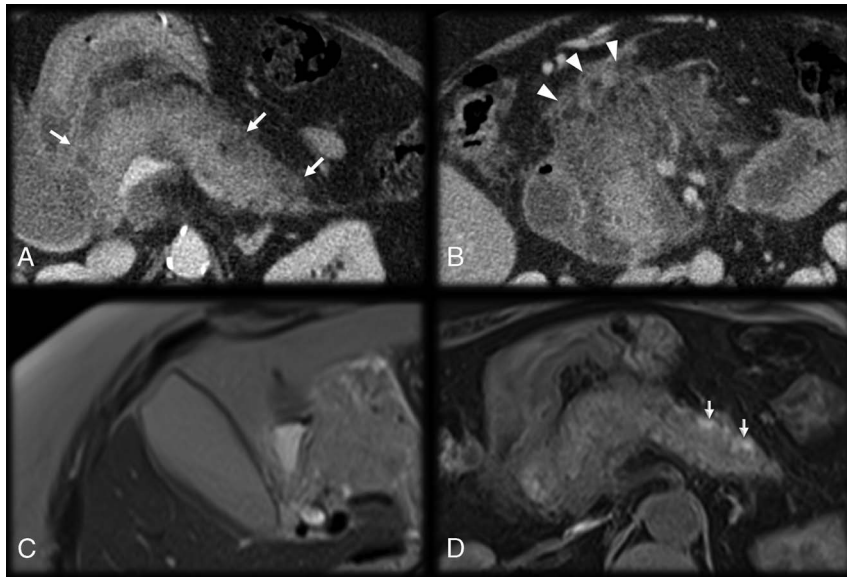
and bowel wall pneumatosis.<sup>6</sup> In some patients with intestinal ischemia, the bowel wall presented an atypical yellow appearance at laparotomy, in contrast to the usual purple or black color of necrotic bowel.<sup>5,20,21</sup> However, it is not possible to detect these findings through imaging.

### Acute Pancreatic Injury

Acute pancreatitis is a multifactorial pathology. Alcohol, gallstones, and hypertriglyceridemia represent the most common etiologies. Although infectious pancreatitis is infrequent,



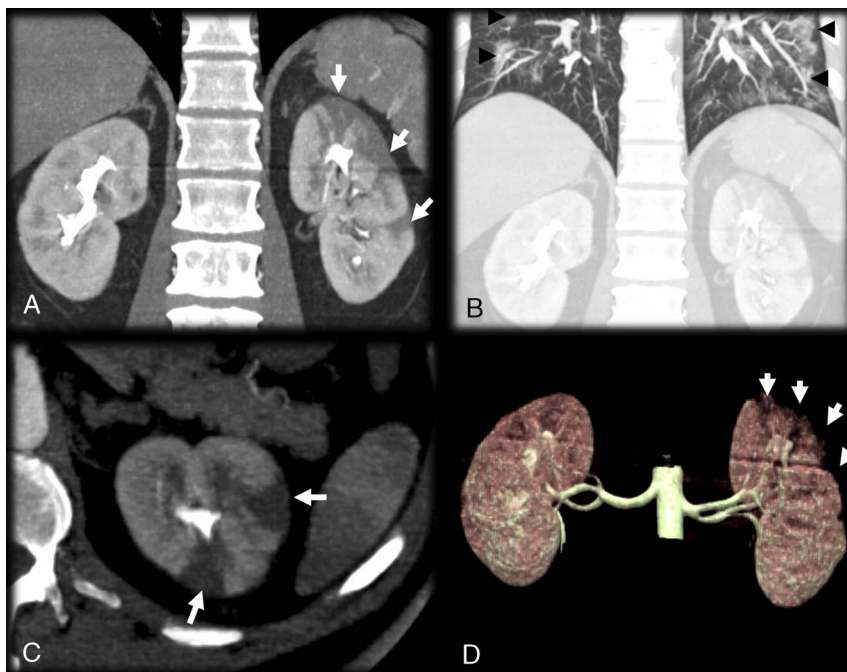
**FIGURE 2.** A, Plain abdominal x-ray with dilated loops of small bowel and intestinal wall pneumatoses (arrows) secondary to extensive abdominal ischemia, more evident on the coronally reconstructed images from a CT of the abdomen (lung window) (B) depicting intestinal wall pneumatoses and gas in mesoileocolic venous vessels (black arrows). Axial (C) and coronal (D) reconstruction from a CT angiography shows perfusion abnormalities in multiple organs (white arrows) with intrahepatic left portal pneumatosis [arrow head in (C)]. D, Pulmonary thromboembolism on the right lower pulmonary lobe (white arrow) with areas of pulmonary infarction and (E) peripheral areas of ground-glass opacity (arrows). Patient died in the next hours secondary to multisystemic organ failure.



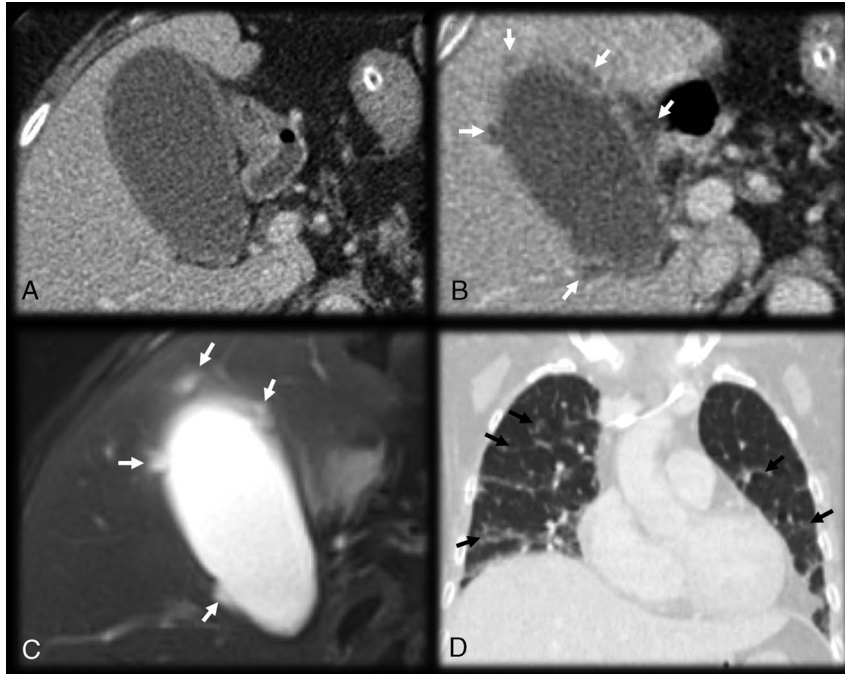
**FIGURE 3.** Acute necrotizing pancreatitis in a 64-year-old woman with COVID-19. A, Axial image of a contrast-enhanced CT of the abdomen shows an ill-defined hypoattenuating region in the body of the pancreas (white arrows), with lack of parenchymal enhancement corresponding with necrosis in less than 30% of the pancreas. B, Peripancreatic fluid with increased attenuation and a heterogeneous appearance of the peripancreatic fat around the head and body of the pancreas (white arrow heads). C, Axial T2-weighted MRCP performed 20 days later shows no gallstones or sludge in the gallbladder or bile ducts. D, Axial fat-saturated T1-weighted image reveals a hyperintense signal in the body and tail of the pancreas (white arrows), findings that are suggestive of hemorrhage, that corresponds to the areas of necrosis.

the largest number of incidents are caused by viruses, including hepatotropic viruses, mumps, cytomegalovirus, coxsackie B virus, HIV, varicella-zoster, and influenza A.<sup>23</sup> Recent analyses have revealed that ACE2 was expressed in both the pancreas and

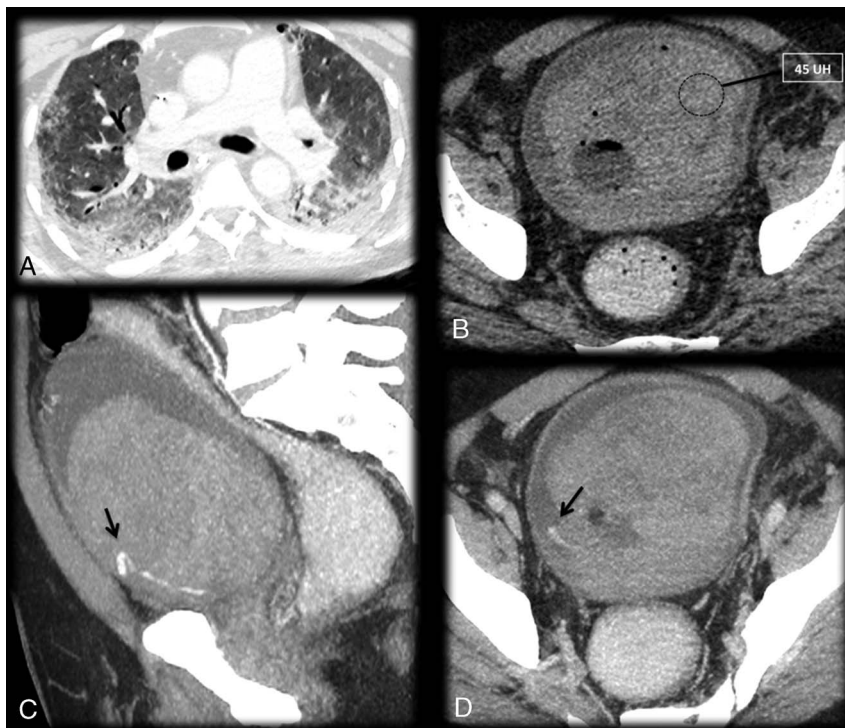
the lungs, with a slightly higher incidence in the pancreas. This indicates that it is likely that SARS-CoV-2 binds to ACE2 in the pancreas and causes pancreatic injury as a result of direct viral infection.<sup>24</sup>



**FIGURE 4.** Renal infarction and COVID-19 pneumonia in a 56-year-old man. Coronal reconstruction (A) and axial (C) images of a contrast-enhanced CT with wedge-shaped parenchymal infarcts involving the upper and lateral segments of the left kidney (white arrows). D, Volumetric 3D reconstructed images of the kidneys depicting areas of infarction (white arrows). B, Incidental finding of multiple ground glass opacifications in a bilateral and subpleural distribution (black arrows). Figure 4 can be viewed online in color at [www.jcat.org](http://www.jcat.org).



**FIGURE 5.** Acute acalculous cholecystitis in a 68-year-old woman COVID-19 pneumonia. A, Axial images of a contrast-enhanced CT that shows distension and mild wall thickening of the gallbladder. Axial image from a contrast-enhanced CT (B) and axial fat-saturated T2-weighted MRCP images (C) 7 days after the baseline, which shows a less distended gallbladder and several pericholecystic fluid collections (white arrows) that are in keeping with perforation. No gallstones or sludge was identified in the gallbladder in MRCP. D, Multifocal peripheral and bilateral areas of ground-glass consolidation and reticulation (black arrows).



**FIGURE 6.** Urinary bladder hematoma with active hemorrhage in a 57-year-old patient. A, Axial images from a CT of the thorax (lung window) show multifocal peripheral and bilateral areas of ground-glass opacity and consolidation in the lungs. B, Axial nonenhanced CT image demonstrates a hyperdense (45 Hounsfield units) heterogeneous hematoma located in the bladder lumen. C, Sagittal maximum intensity projection reconstruction and (D) axial images from a contrast-enhanced CT of the pelvis show 2 areas of contrast extravasation (black arrows) that are in keeping with active hemorrhage on arterial (C) and portal-venous phase (D) on the anterior and right anterolateral walls of the urinary bladder.

There have been a few case reports of acute pancreatic injury in COVID-19 infection. One of the earliest reported instances involved 2 cases in a Wuhan family with confirmed COVID-19 and pancreatitis, where other etiologies were ruled out.<sup>25</sup> A study conducted by Liu et al,<sup>26</sup> of 64 patients with severe COVID-19, reported that 17.9% had increased amylase and 16.4% increased lipase levels. However, of the 13 patients, only 5 (7.46%) showed pancreatic changes on computed tomography (CT) imaging.

Wang et al<sup>27</sup> also found that of 52 patients with COVID-19 pneumonia, 17% presented evidence of pancreatic injury. In a study conducted by Bhayana et al,<sup>5</sup> 42 patients with COVID-19 underwent abdominal MDCT for gastrointestinal symptoms, and only 1 (2.7%) patient presented findings compatible with pancreatitis. It is to be noted that most of these cases reported moderate or severe COVID-19. This seems to suggest that the pathophysiology of pancreatitis is more likely caused by a systemic inflammatory response rather than being a direct cytopathological effect of the virus.

Acute pancreatitis is a rare manifestation in COVID-19 (Fig. 3), and further studies are needed to determine a causal relationship between acute pancreatic injury and the virus. However, the temporal association between pancreatitis and COVID-19 is highly suggestive of SARS-CoV-2–induced damage.

### Kidney Injury

Several studies report that between 0.5% and 19% of patients with COVID-19 have some degree of acute renal failure, and it is described as the second most frequent fatal complication.<sup>2</sup> It is thought that acute renal failure is possibly related to the cytopathic effect of SARS-CoV-2.<sup>28</sup>

In a study conducted in Wuhan China, when histological renal samples from postmortem patients infected with COVID-19 were examined, it was demonstrated that the SARS-CoV-2 virus can directly infect the tubular epithelium and podocytes.<sup>29</sup> This suggests that direct viral infection could explain the acute renal failure and proteinuria.

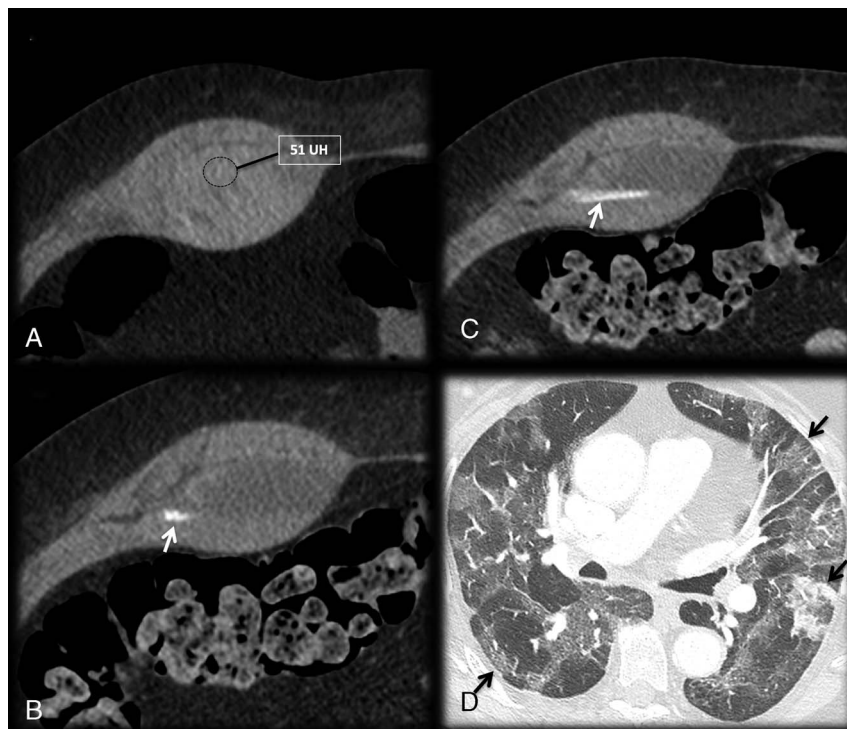
Regarding renal infarction (Figs. 2, 4), the most accepted mechanisms are the prothrombotic effects of the virus, small vessel thrombosis, and global systemic hypoperfusion.<sup>22</sup> The inflammatory effect on the vascular endothelium could also favor ischemic events.<sup>30,31</sup>

### Hepatocellular Dysfunction and Cholecystitis

Abnormal liver enzymes in patients with COVID-19 were first reported by Chen et al.<sup>31</sup> Since then, these alterations have been widely reported and could be present in more than 50% of patients with COVID-19.<sup>13,19,32</sup>

The ACE2 receptors in the liver are expressed especially in cholangiocytes (60%) and to a lesser extent in hepatocytes (3%). The direct viral damage, together with the effects produced by systemic inflammation, cytokine storm, and metabolic alterations associated with hypoxia, seems to be the main mechanisms involved in the liver damage produced by COVID-19.<sup>14,22,33</sup> An example of liver dysfunction is presented in Figure 2.

The expression of ACE2 in the gallbladder epithelium suggests direct viral infection, and it is thought to produce local inflammation on the gallbladder mucosa.<sup>32</sup> This could explain the imaging findings in cases of acalculous cholecystitis (Fig. 5), in which no other etiology, such as gallstones, biliary sludge, or pericholecystic fluid, has been found.<sup>34–36</sup>



**FIGURE 7.** Spontaneous rectus sheath hematoma on a 75-year-old woman with severe COVID-19. A, Axial unenhanced CT image shows a hyperattenuating mass (51 Hounsfield units) on the muscle sheath of the rectus abdominus, revealing a hematoma on this location. Axial images from a CT angiography obtained in the arterial (B) and venous (C) phase show active bleeding (white arrows) from a segmental branch of the right inferior epigastric artery. D, Bilateral and peripheral consolidations (black arrows) as a manifestation of a known COVID-19 pneumonia that showed the “atoll sign,” also known as “reverse halo sign,” located in the left inferior pulmonary lobule.

However, it is important to remember that abnormal liver enzymes and acalculous cholecystitis can occur in critically ill patients.<sup>35</sup>

The pathological mechanisms of the virus that result in injury to the gallbladder and liver have not yet been fully determined. More studies focused on the complications of SARS-CoV-2 on these organs and the underlying mechanisms of damage are needed.<sup>19</sup>

## Hemorrhagic Complications

An important concern in patients with COVID-19 is the high incidence of thromboembolic events. Prompt treatment with anticoagulants has proven to reduce mortality.<sup>20,34</sup> However, the anticoagulant treatment increases the risk of hemorrhagic complications, as observed in Figures 6 and 7. To our knowledge, studies regarding abdominal hemorrhagic complications have not yet been reported, but with the increase in anticoagulant therapy as part of the management and prevention of thromboembolic events in patients with COVID-19, awareness of the possibility of these events occurring is necessary.

## Systemic Inflammatory Response Syndrome

There are a very limited number of publications regarding systemic inflammatory response syndrome in adults infected with COVID-19. Some findings suggest that this syndrome could be a major contributor to COVID-19-associated coagulopathy, supporting the damage mechanism of thromboinflammation.<sup>37</sup> Furthermore, systemic coagulopathy is common in critically ill patients with COVID-19, and this observation has been supported by descriptions of complement-mediated microvascular injury and vascular imaging abnormalities.<sup>5</sup>

In children, COVID-19 infection can manifest as multisystem inflammatory syndrome in children, a serious condition that presents as a group of signs and symptoms similar to Kawasaki disease toxic shock syndrome. The constellation of findings includes airway inflammation with rapid development of pulmonary edema, coronary artery aneurysms, and extensive right iliac fossa inflammatory changes.<sup>38</sup>

In adults, some of the more severe multiorgan complications can present as a manifestation of the “hypotension complex,” which is sometimes observed in patients with severe hypotension due to shock,<sup>22,38,39</sup> as seen in Figure 2.

## CONCLUSION

SARS-CoV-2, initially considered a respiratory tract pathogen, can cause multiple-organ dysfunctions. Gastrointestinal symptoms have been reported frequently in patients with COVID-19, and increasing evidence has been able to link viral infection to multiple abdominal complications. Updated studies indicate the virus can produce similar complications in different organs, the most common of which seem to be of thrombotic or inflammatory nature.

As some of the images demonstrated, the abdominal manifestations can present as an isolated event in the abdomen (like pancreatitis or acalculous cholecystitis) or as part of a systemic response (like the inflammatory response seen in systemic inflammatory response syndrome and multisystem inflammatory syndrome in children or as perfusion abnormalities in multiple organs).

We hope that increased awareness of these abdominal manifestations, combined with a deeper understanding of their pathogenesis and an adequate interpretation of imaging findings, will increase the timely detection of these pathologies and allow their prompt treatment.

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