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INTRODUCTION
Numerous abdominal manifestations have been reported in patients with coronavirus disease 2019 (COVID-19), including involvement of the luminal gastrointestinal (GI) tract, hepatobiliary system, pancreas, kidneys, spleen, and blood vessels. Specific COVID-19 related pathologic entities described in the literature within these abdominal organ systems include ileus, Ogilvie syndrome, GI bleeding, bowel ischemia, gastric and bowel perforation, liver function abnormalities, acute hepatitis, fulminant liver failure, cholestasis, cholecystitis, secondary sclerosing cholangitis, hemobilia, pancreatitis, acute renal dysfunction, proteinuria, hematuria, splenomegaly, ascites, and arterial, portal venous, and systemic venous thrombosis [1–21].

Among the abdominal manifestations, GI symptoms are particularly common. A meta-analysis of COVID-19 patients showed a pooled incidence of any GI symptom in 12%, including abdominal pain in 4% to 5%, diarrhea in 7% to 8%, and nausea or vomiting in 5% to 8% [22,23]. At one institution, 45% of COVID-19 patients admitted to the surgical intensive care unit (ICU) experienced GI symptoms [3]. Some COVID-19 patients present with only GI manifestations in the absence of respiratory symptoms [5,9,11,13,24]. GI manifestations are more frequent among COVID-19 patients with a longer duration of illness and ICU submission but have not been definitively associated with increased mortality [14]. Pathophysiologic support of abdominal organ involvement includes fecal shedding of the causative SARS-CoV-2 RNA in 40% to 50% of COVID-19 patients and epithelial expression of its host cell entry point, the angiotensin-converting enzyme 2 (ACE-2) receptor, in the esophagus, stomach, small bowel, colon, rectum, hepatocytes, cholangiocytes, pancreatic islet cells, spleen, and kidneys [2,14,22,25–27].

Abdominal imaging manifestations of COVID-19 have been described. Although most of the radiological abnormalities are nonspecific without distinguishing imaging features to suggest COVID-19, unique presentations such as findings of bowel ischemia preceding gross findings of bowel necrosis have been reported.
Awareness of the spectrum of abdominal manifestations of COVID-19 allows radiologists to optimize their search pattern and to raise the possibility of this etiology when appropriate. This review provides a comprehensive overview with the illustrative imaging examples of COVID-19 in the abdomen.

**DISCUSSION**

**Luminal Gastrointestinal Tract**

Bowel inflammation, hypomotility, and ischemia are the major reported bowel imaging abnormalities in COVID-19 patients, reported in approximately 30% of patients imaged by computed tomography (CT) including findings of ischemia in 20% of ICU patients [1,3,20,28,29]. Bowel inflammation due to COVID-19 has a similar appearance to other causes of gastritis, enteritis, and colitis, including circumferential mural thickening, hyperenhancement, and surrounding edema and fat stranding (Fig. 1). The findings of ischemia are also not etiology-specific and include bowel wall nonenhancement, pneumatosis intestinalis, and portal or mesenteric venous gas (Figs. 2 and 3) [18]. The characteristic pathologic findings of necrotic, ischemic bowel in COVID-19 patients are a yellowing anti-mesenteric discoloration grossly with microvascular fibrin thrombi in the submucosal arterioles on histologic examination [1,3,29]. Importantly, imaging findings of bowel ischemia in COVID-19 patients may occur in asymptomatic patients and precede the clinical manifestation and gross appearance of bowel necrosis detected by physical examination and exploratory laparotomy, necessitating close follow-up and possible second-look laparotomy pending clinical evolution of patient status (see Fig. 2) [1].

Hypomotility of large and small bowel manifests as an ileus pattern with gas and fluid-filled, distended large, and small bowel [1,3]. Among COVID-19 patients in one institutional surgical ICU, 56% developed a hypomotility complication clinically or radiographically, two developed small bowel ischemia requiring resection, one of which developed Ogilvie syndrome-like paralytic colonic ileus requiring total colectomy [3]. When assessing this population of ICU patients who often have ileus pattern on abdominal radiographs, which are routine in many institutions, it is...
prudent for radiologists to maintain a high index of suspicion for identifying pneumatosis intestinalis and portal venous gas, which can be subtle (see Fig. 2).

Additional complications of bowel involvement in COVID-19 consist of gastric, small bowel, and colonic perforation (Fig. 4) as well as GI bleeding, including hematochezia and melena [16,30,31]. Upper GI bleeding, defined as arising proximal to the ligament of Treitz, is more common than lower GI bleeding and has been reported to respond to conservative management in most of the patients (~60%) [32,33]. The presence of active GI bleeding in COVID-19 patients and localization of the anatomic source may be diagnosed by radiologists using multiphase CT angiography for active GI bleeding and CT enterography for occult or suspected small bowel bleeding [34]. Dual-energy CT technique allows the reconstruction of virtual unenhanced iodine overlay and monochromatic low kiloelectron volt images which can increase the conspicuity of bowel pathology such as lack of wall enhancement in ischemia, hyperenhancement in inflammation, differentiate mural or luminal hemorrhage from enhancement or active bleeding, and reduce radiation dose (see Fig. 1) [35].

**Hepatobiliary**

Acute liver function test (LFT) abnormalities are common in patients with COVID-19; however, the imaging
FIG. 3 Bowel ischemia: A 53-year-old critically ill patient with COVID-19 requiring extracorporeal membrane oxygenation (ECMO) had a rising pressor requirement, and a right upper quadrant ultrasound obtained for hyperbilirubinemia and transaminitis had branching echogenic foci along the portal vein branches with associated dirty shadowing, consistent with portal venous gas (solid arrows, A and B) and raising suspicion for COVID-19 ischemic bowel. The abdomen was explored, and there was patchy necrosis noted on the transverse colon. Pathology showed subserosal organizing fibrin thrombus and focal submucosal edema in the ischemic colon. The patient also had a sludge-filled gallbladder consistent with cholestasis (dashed arrow, C).

FIG. 4 Bowel perforation: A 60-year-old man presented with hypoxemic respiratory failure due to COVID-19 and during hospitalization developed diarrhea. He was diagnosed with enteritis. Worsening abdominal distension prompted repeat CT. (A) Axial and (B) coronal images from contrast-enhanced CT through the upper abdomen demonstrated free intraperitoneal air and ascites, suggesting bowel perforation (solid arrow, A) and long-segment enteritis of small bowel with perienteric stranding and free fluid (dashed arrow, B), consistent with enteritis. A midline laparotomy was performed on entering the abdomen, 3L of enteric contents was evacuated from the abdomen, and a focal perforation in the inflamed proximal duodenum was identified and repaired.
manifestations are nonspecific [4,7]. Biochemical abnormalities may show a predominantly hepatocellular or cholestatic pattern [3,4,22,23]. Severe LFT abnormalities accompanying acute liver failure, liver ischemia and necrosis, and liver-related mortality in COVID-19 have been reported [3,8,9,36]. COVID-19 patients with severe LFT abnormalities have a worse prognosis than those without LFT abnormalities, including significantly higher rates of ICU admission, intubation, renal replacement therapy, and mortality [4,7,37]. Hepatobiliary abnormalities detected by imaging and attributed to COVID-19 include cholestasis, acute cholecystitis, hemobilia, periportal edema, hepatomegaly, and secondary sclerosing cholangitis [10,14,15,18,21,38,39]. As with bowel abnormalities, the imaging manifestations of gallbladder and liver inflammation are nonspecific and include findings such as gallbladder distension and wall thickening, hepatomegaly, and periportal edema [14,39]. Differentiation of liver function abnormalities due to COVID-19 from other causes is essential, both given the prognostic implications for COVID-19 patients and the need for recognizing other treatable causes, such as obstructive choledocholithiasis and portal vein thrombosis.

Sonographic findings of cholestasis, including gallbladder distension and sludge, have been reported in 54% of right upper quadrant ultrasonograms in COVID-19 inpatients (see Fig. 3). Although often visible on ultrasound (US) imaging, MRI may have a higher sensitivity for imaging detection of subtle biliary pathology, such as mild intrahepatic duct dilation and bile duct structuring [1,40]. CT may not be as sensitive as biliary ductal dilation has been reported in up to 10% of COVID patients by this modality [39,41,42]. Dual-energy CT with iodine overlay and monochromatic low-KeV reconstructions have utility in improving assessment of gallbladder wall enhancement to detect hyperemia in cholecystitis and nonenhancement in gangrenous cholecystitis [43].

Sclerosing cholangitis in critically ill patients requiring vasoactive medications and mechanical ventilation has been described in the literature, hallmarks of which include cholestasis and cast formation leading to acute secondary biliary infection and ischemia with possible development of chronic sclerosing cholangitis [44,45]. In contrast to causes of critical illness such as influenza or bacterial sepsis, SARS-CoV-2 has a potential mechanism for specific biliary injury via cholangiocyte co-expression of proteins involved in viral entry: ACE-2 receptor, transmembrane protease serine 2, and cathepsin L [26,46–48]. This is supported by a retrospective cohort study comparing biliary injury in patients with severe COVID-19 and influenza showing more frequent and severe cholestasis among the COVID-19 cohort as well as biopsy specimens demonstrating SARS-CoV-2 virions within cholangiocytes [15,49]. Sclerosing cholangitis is best imaged with MR cholangiopancreatography (MRCP), a challenge in patients with COVID-19 who most frequently require hospitalization for respiratory failure and may have difficulty with prolonged supination with intermittent breath holds for MRI. Reports of COVID-19-related cholangitis describe a similar spectrum of findings to another sclerosing cholangitis, including common bile duct wall thickening and hyperenhancement, intrahepatic ductal beading, stricturing, and maybe temporally dissociated from biochemical markers of hepatocyte injury (Fig. 5) [15,38,50–53]. The incidence of mild COVID-19-related sclerosing cholangitis may be underestimated, as MRI is rarely performed in the acute to subacute stages of COVID-19 and rarely results in clinical management changes [54].

Pancreas
Pancreatic inflammation associated with COVID-19 has been described with biochemical and imaging abnormalities (Fig. 6) [3,11–13,20]. Wang and colleagues [12] reported abnormalities in amylase or lipase levels indicating pancreatic injury in 17% of inpatients (n = 52) presenting with COVID-19 pneumonia. Pancreatitis has been described as an initial presentation of COVID-19, with later development of respiratory symptoms and eventually respiratory failure [13]. Recurrent acute pancreatitis during COVID-19 illness and necrotizing pancreatitis have also been described [11,18]. Pancreatic expression of ACE-2 receptors as the binding and entry site for SARS-CoV-2 supports COVID-19 as the causative agent in patients with acute pancreatitis [18,27]. Imaging manifestations on CT or MR include parenchymal swelling, edema, and peripancreatic inflammation, as well as parenchymal non-enhancement in cases of necrosis, which do not differ from other etiologies of pancreatitis [3,11–13,20]. Dual-energy CT iodine overlay reconstructions increase the conspicuity of non-enhancing parenchyma in necrotizing pancreatitis, and virtual unenhanced reconstructions can differentiate hemorrhagic pancreatitis from enhancing inflammation [43].

Kidneys
Acute kidney injury, proteinuria, and hematuria are known complications of COVID-19 and may require dialysis [14,18]. Renal imaging findings in COVID-19 patients with renal dysfunction are sparsely reported.
but include patchy bilateral hypoenhancing areas in the renal cortices as well as wedge-shaped non-enhancing cortical infarctions [18,55,56]. Areas of perfusional alteration manifesting on renal imaging may be secondary to visible large vessel thrombosis or microthrombotic/embolic phenomena without visible vessel occlusion [55]. Acute renal injury-associated CT imaging findings of cortical hypoperfusion are reported to be reversible with the recovery of renal function [55]. In cases of subtle hypoenhancement of parenchyma, findings will be more conspicuous on iodine overlay and monochromatic low-KeV reconstructions from dual-energy CT than with conventional blended CT images [43]. In addition to recognizing these imaging abnormalities in COVID-19 patients with renal dysfunction, it is important to rule out alternative causes such as mechanical urinary tract obstruction from urolithiasis or urothelial neoplasm.

**Spleen**

Spleen size has been found to increase in the acute phase of COVID-19 infection, and the degree of enlargement correlates with disease severity (Fig. 7) [19]. Splenomegaly is a nonspecific finding which also occurs in other infections associated with cytokine storm, including other viral infections and noninfectious causes, such as portal hypertension or lymphomatous infiltration [19]. A case of a woman without a history of trauma who presented with sudden onset abdominal pain was found to have hemoperitoneum and spontaneous splenic rupture, and to have COVID-19 infection, was determined to be the likely etiology for splenic rupture and bleeding as no alternative cause was found on extensive workup [57]. Authors hypothesized the mechanism to be microthrombotic organ congestion and laceration. Imaging recognition of splenic abnormalities, including changes in spleen size, infarcts, and bleeding complications, is important.

**Vasculature**

Patients with COVID-19 are hypercoagulable and at risk for arterial and venous thromboembolic events. In the abdomen, involvement of the arterial vasculature (aorta, iliac, celiac, common hepatic, and superior mesenteric arteries) as well as venous structures (portal and renal veins) has been described (Fig. 8) [20,58–60]. Visceral consequences of non-visualized microembolic events include renal infarcts, splenic infarcts, and bowel ischemia, as described above [1,58]. The reported incidence of solid organ infarcts or vascular thrombosis in COVID-19 patients ranges from 18% to 45% [39,61]. Although these are often complications that develop
FIG. 6 Pancreatitis: A 77-year-old woman with no history of pancreatitis or predisposing factors presented with fevers and fatigue in the setting of COVID-19 and developed respiratory failure. Presentation abdominal CT showed a normal pancreas (A and B). During hospitalization, she developed increased abdominal distension and a repeat CT 1 week after presentation showed new pancreatic swelling and peripancreatic fat stranding (solid arrows, C–E), consistent with acute pancreatitis. No alternative cause apart from active COVID-19 was found.

FIG. 7 Splenomegaly: A 60-year-old man with no history of splenomegaly or known predisposing conditions presented with hypoxemic respiratory failure due to COVID-19 and was found to have splenomegaly (A). After recovery from COVID-19 infection, the spleen was normal size (B), 2 months following his presentation with COVID-19.
in critically ill patients with known COVID-19 illness, they may also be the presenting manifestation of infection as in a case report of a young woman, without past medical history, who developed abdominal pain and bloating and was found to have portal vein thrombosis by CT and US as well as COVID-19 infection, without an alternative cause for the thrombus [62]. Hemorrhage in the abdomen of COVID-19 patients without an alternative cause (Fig. 9) has also been reported, including intraperitoneal hemorrhage from organ splenic thrombosis, hemobilia, and hematuria [18,21,57,60]. Dual-energy CT performed in the setting of vascular pathology or bleeding can provide radiation dose reduction by eliminating the need for separate unenhanced acquisition and instead of using virtual unenhanced reconstructions [43]. As many of these vascular complications are visualized on cross-sectional imaging examinations done for separate indications, radiologists must be keenly aware of the high incidence and scrutinize the blood vessels for patency.

FIG. 8 Venous thrombus: A 65-year-old woman with COVID-19 infection developed nonocclusive portal vein thrombus characterized by lack of color and power Doppler flow and echogenic filling defect on ultrasound (solid arrows, A and B).

FIG. 9 Hemorrhage: A 60-year-old man presented with hypoxemic respiratory failure due to COVID-19 and developed diarrhea. His course was complicated by multiple intra-abdominal collections and spontaneous intra-abdominal/retroperitoneal hematomas with hemodynamic instability requiring transfusion and vasopressor support. Axial (A) and coronal (B) contrast-enhanced CT through the abdomen demonstrates a heterogeneously hyperdense collection in the midline retroperitoneum displacing the mesenteric root (solid arrows, A, B), consistent with hemorrhage.
SUMMARY
Multiple abdominal manifestations of COVID-19 have imaging correlates, most of which are nonspecific and indistinguishable from other causes of organ inflammation. Awareness of the possible abdominal manifestations of COVID-19 should enhance detection by radiologists and improve patient care.

CLINICS CARE POINTS
- Coronavirus disease 2019 can affect the bowel, liver, bile ducts, gallbladder, pancreas, spleen, kidney, and blood vessels.
- Radiologists should scrutinize these organs in imaging studies, regardless of presenting symptomatology, for complications.
- Bowel ischemic imaging findings may precede gross findings of necrotic change on laparotomy and require to take back to the operating room for a second look.
- Dual-energy computed tomography can increase the conspicuity of abnormalities, such as enteritis using monoenergetic low kiloelectron volt and iodine overlay reconstructions.

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