Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

Advances in Clinical Radiology 4 (2022) 25–35
ADVANCES IN CLINICAL RADIOLOGY

Coronavirus Disease in the Abdomen

Mark A. Anderson, MD*, Mark A. Khauli, MD, Reece J. Goiffon, MD, PhD, Avinash Kambadakone, MD
Department of Radiology, Massachusetts General Hospital, 55 Fruit Street, White Building, Room 270, Boston, MA 02114, USA

KEYWORDS
• Coronavirus disease 2019 • COVID-19 • SARS-CoV-2 • Enteritis • Ischemic bowel

KEY 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 volts and iodine overlay reconstructions.

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.

*Corresponding author, *E-mail address:* mark.anderson@mgh.harvard.edu

https://doi.org/10.1016/j.yacr.2022.04.003
2589-8701/22/

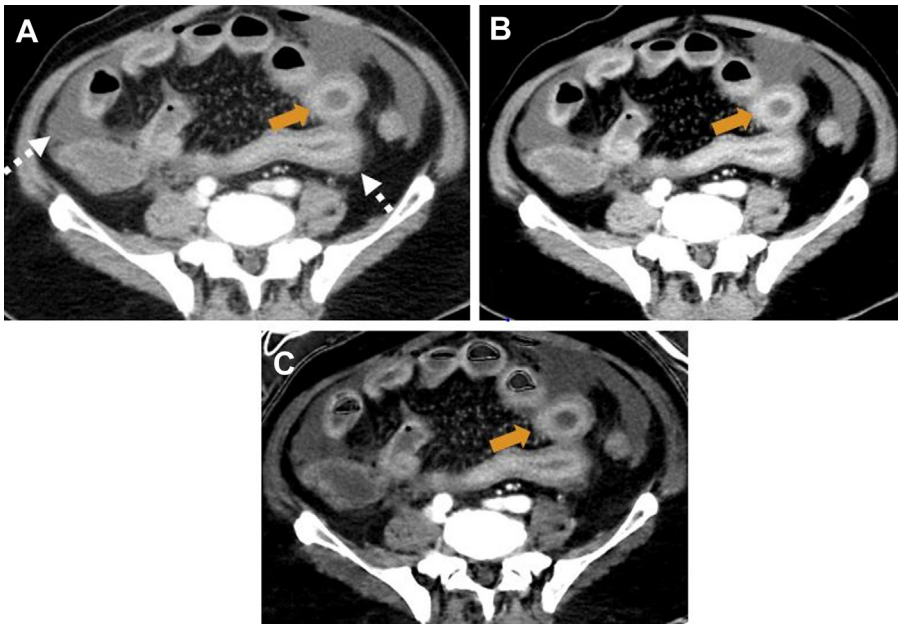

FIG. 1 Enteritis: A 60-year-old man presented with hypoxemic respiratory failure due to COVID-19 developed diarrhea. (**A**) Conventional blended 140 KeV, (**B**) virtual monoenergetic 65 KeV, and (**C**) iodine overlay axial images from dual-energy contrast-enhanced CT through the lower abdomen demonstrate long-segment circumferential small bowel wall thickening and stratified hyperenhancement (*solid arrows*) with perienteric stranding and free fluid (*dashed arrows*), consistent with enteritis. Mural hyperenhancement is more conspicuous on low-KeV and iodine overlay images.

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

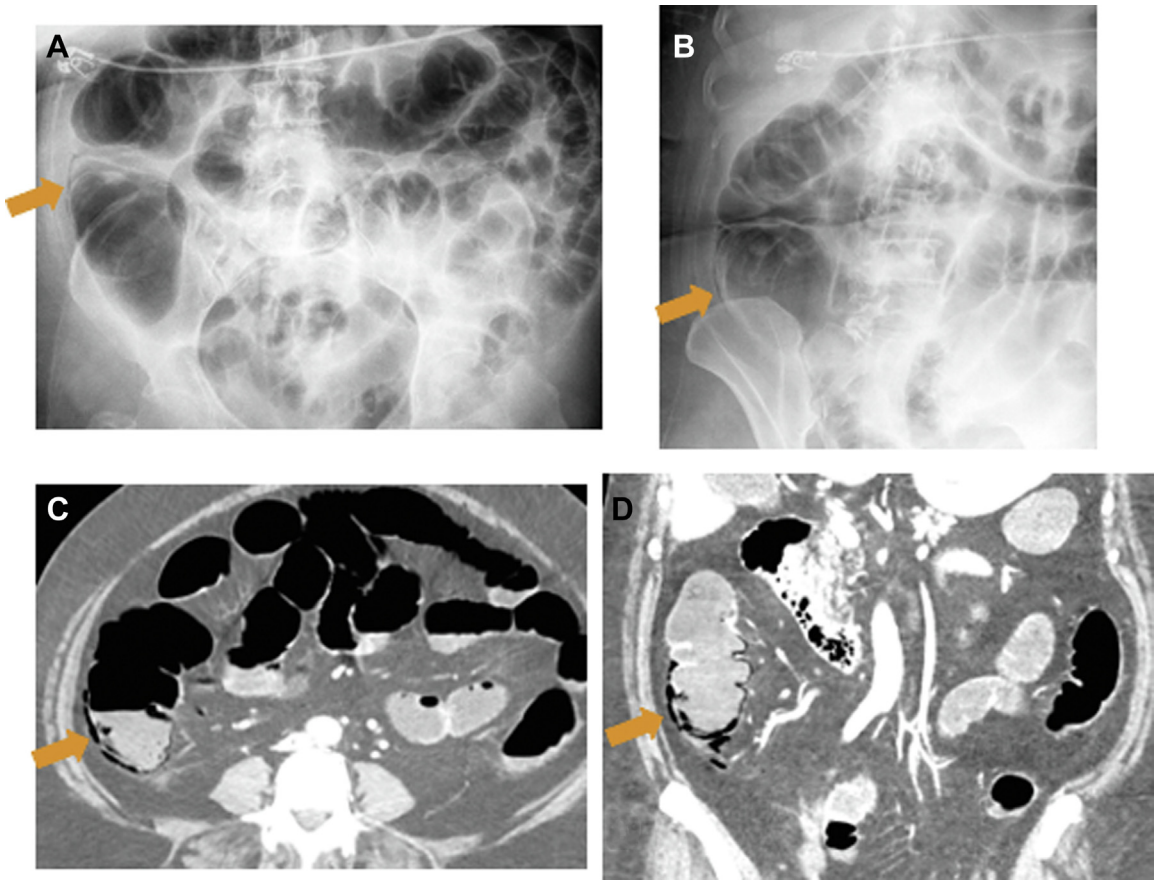

FIG. 2 Ischemic colitis: A 78-year-old woman with COVID-19 infection and respiratory failure incidentally was found to have right colonic pneumatosis on the abdominal radiograph (*solid arrows*, **A** and **B**) and CT (*solid arrows*, **C** and **D**). The same day laparotomy found that the entire bowel was pink and healthy, with no evidence of ischemic compromise. Second-look laparotomy 24 hours later found necrotic cecum, ascending colon, and ileum requiring right hemicolectomy and small bowel resection. Pathology reported microvascular fibrin thrombi supporting the diagnosis of ischemic necrosis.

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

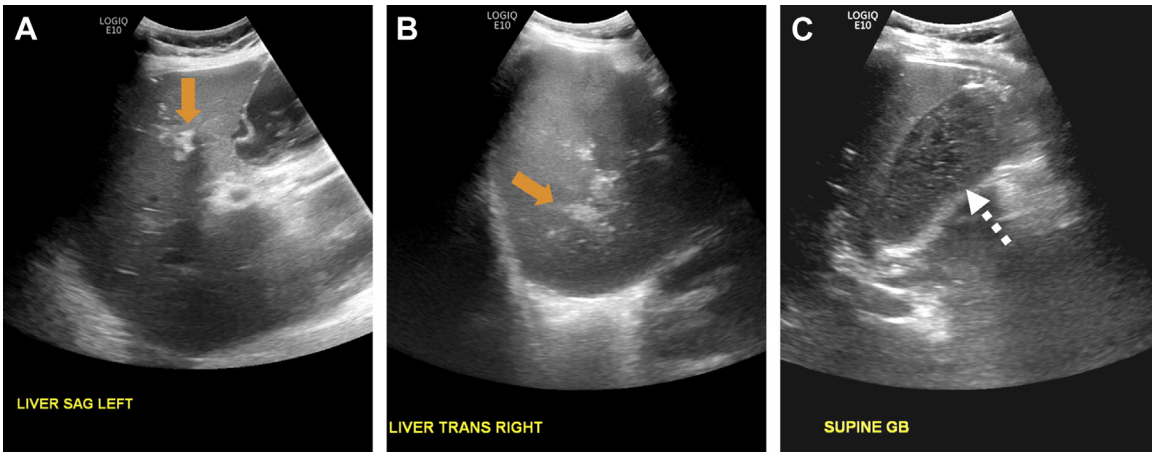

FIG. 3 Bowel ischemia: A 53-year-old critically ill patient with COVID-19 requiring extracorporeal membrane oxygenation (ECMO) had a rising pressor requirement, and a right upper quadrant ultrasound obtained for hyperbilirubinemia and transaminitis had branching echogenic foci along the portal vein branches 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**).

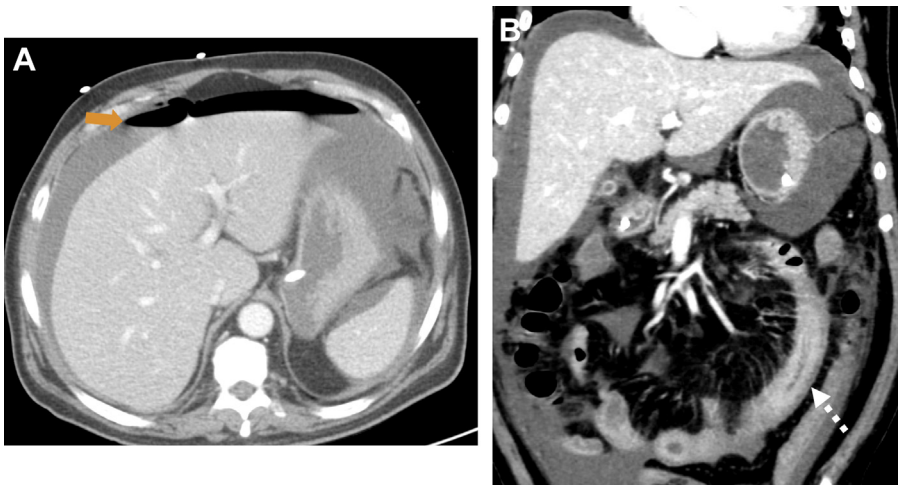

FIG. 4 Bowel perforation: A 60-year-old man presented with hypoxemic respiratory failure due to COVID-19 and during hospitalization developed diarrhea. He was diagnosed with enteritis. Worsening abdominal distension prompted repeat C. (**A**) Axial and (**B**) coronal images from contrast-enhanced CT 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 ultrasounds 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 nonenhancement 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

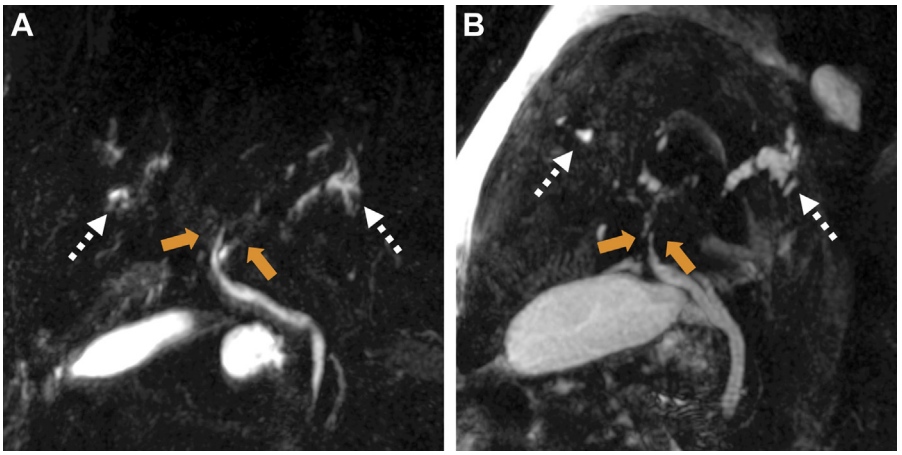

FIG. 5 Secondary sclerosing cholangitis: A 76-year-old man developed new, progressive bilirubin and alkaline phosphate elevation during hospitalization for COVID-19 pneumonia that was normalizing at discharge before markedly increasing 3 months after presentation. Coronal 1.5 maximum intensity projection (MIP) images of MR cholangiopancreatography (MCRP) obtained 103 days after initial hospital presentation (**A**) and showing multifocal stricturing of the central intrahepatic ducts (*solid arrow*, A) and upstream dilation (dashed *arrows*, A). At the time, total bilirubin was 13.6 mg/dL. Coronal 1.5 cm MIP image from MCRP of the same patient performed 456 days after admission (*solid arrow*, B) showing increased intrahepatic biliary dilation (*dashed arrows*, B) upstream from severe multifocal stricturing. Total bilirubin at the time was 3.2 mg/dL.

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

Splenic 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

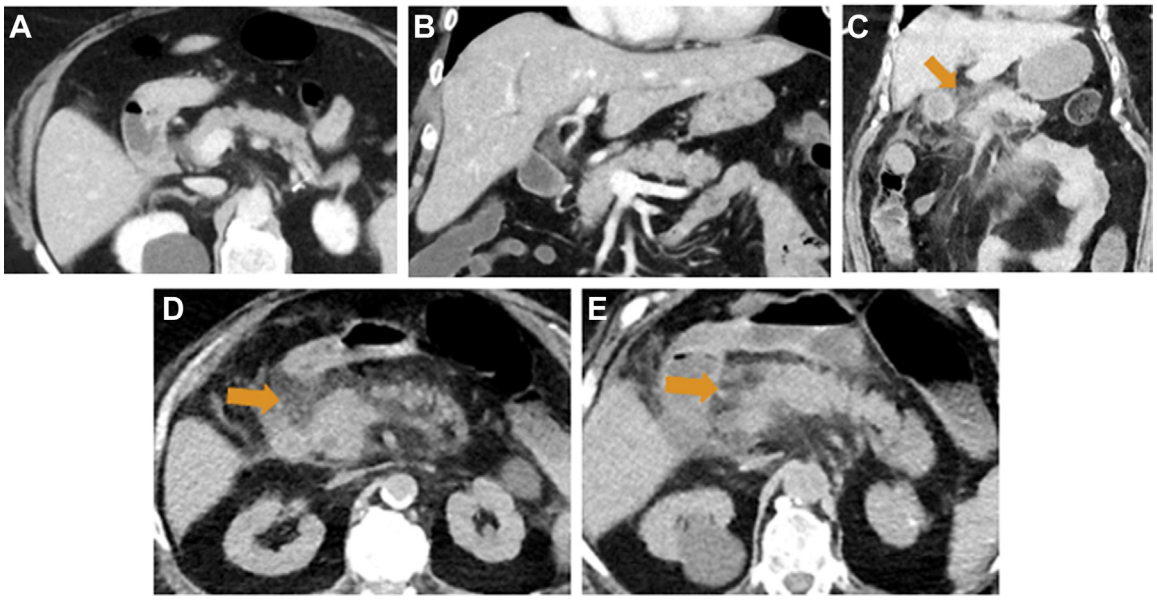

FIG. 6 Pancreatitis: A 77-year-old woman with no history of pancreatitis or predisposing factors presented with fevers and fatigue in the setting of COVID-19 and developed respiratory failure. Presentation abdominal CT showed a normal pancreas (**A** and **B**). During hospitalization, she developed 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.

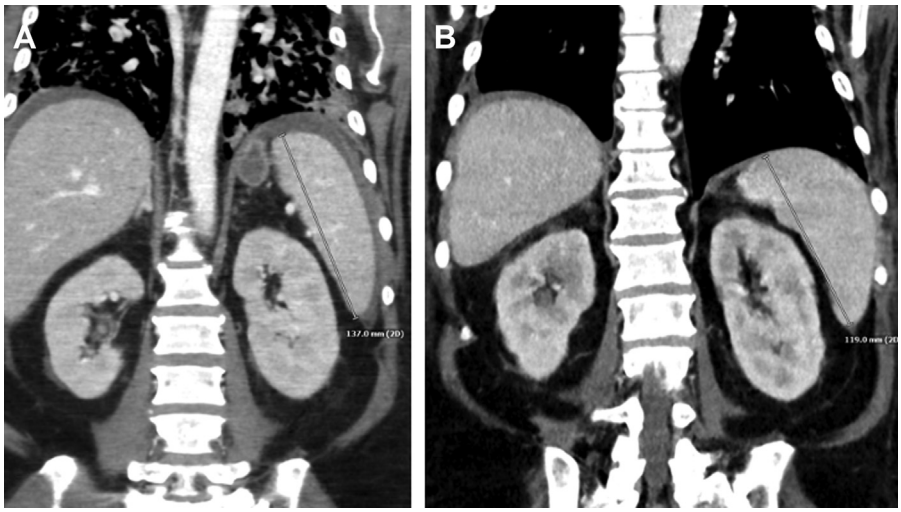

FIG. 7 Splenomegaly: A 60-year-old man with no history of splenomegaly or known predisposing conditions presented with hypoxemic respiratory failure due to COVID-19 and was found to have splenomegaly (**A**). After recovery from COVID-19 infection, the spleen was normal size (**B**), 2 months following his presentation with COVID-19.

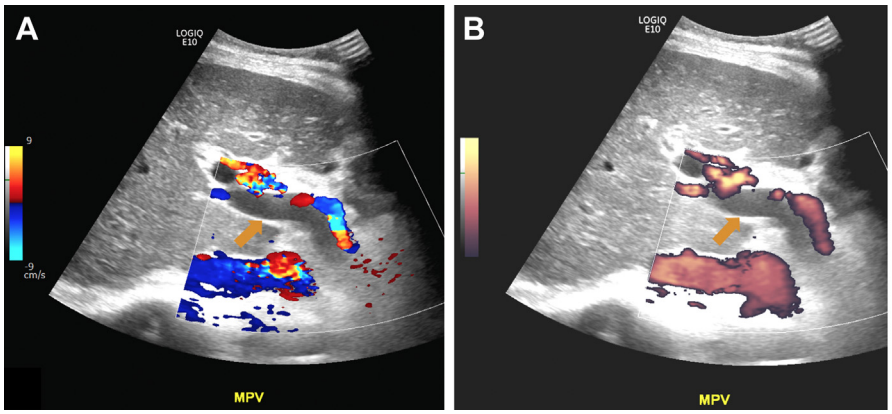

FIG. 8 Venous thrombus: A 65-year-old woman with COVID-19 infection developed nonocclusive portal vein thrombus characterized by lack of color and power Doppler flow and echogenic filling defect on ultrasound (*solid arrows*, **A** and **B**).

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.

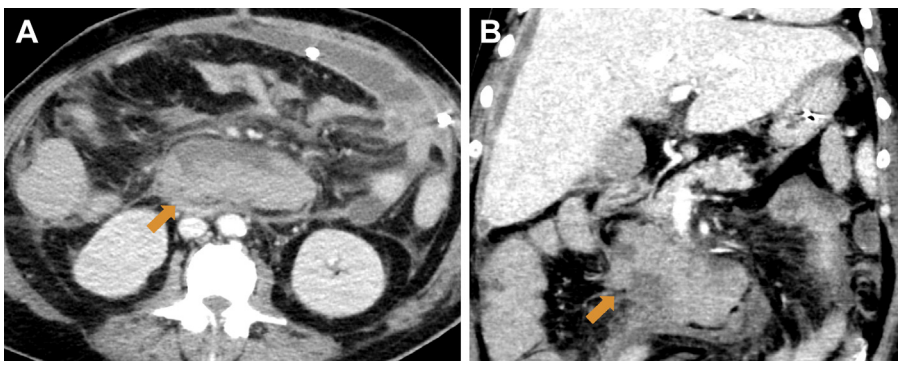

FIG. 9 Hemorrhage: A 60-year-old man presented with hypoxemic respiratory failure due to COVID-19 and developed diarrhea. His course was complicated by multiple intra-abdominal collections and spontaneous intra-abdominal/retroperitoneal hematomas with hemodynamic instability requiring transfusion and 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.

DISCLOSURE

All authors report that no conflicts of interest exist. Avinash Kambadakone discloses Research Grants from GE Healthcare, Philips, and PanCAN.

REFERENCES

[1] Bhayana R, Som A, Li MD, et al. Abdominal imaging findings in COVID-19: preliminary observations. Radiology 2020;297(1):E207–15.

[2] Moheb ME, Naar L, Christensen MA, et al. Gastrointestinal complications in critically ill patients with and without COVID-19. JAMA 2020. https://doi.org/10.1001/jama.2020.19400.

[3] Kaafarani HMA, El Moheb M, Hwabejire JO, et al. Gastrointestinal complications in critically ill patients with COVID-19. Ann Surg 2020;272(2):e61–2.

[4] Bertolini A, van de Peppel IP, Bodewes FAJA, et al. Abnormal liver function tests in COVID-19 patients: relevance and potential pathogenesis. *Hepatology*N/a(n/a). Doi:10.1002/hep.31480

[5] Melquist S, Estepp K, Aleksandrovich Y, et al. COVID-19 presenting as fulminant hepatic failure. Medicine (Baltimore) 2020;99(43). https://doi.org/10.1097/MD.0000000000022818.

[6] Mauro A, De Grazia F, Lenti MV, et al. Upper gastrointestinal bleeding in COVID-19 inpatients: incidence and management in a multicenter experience from Northern Italy. Clin Res Hepatol Gastroenterol 2020. https://doi.org/10.1016/j.clinre.2020.07.025.

[7] Phipps MM, Barraza LH, lasota ED, et al. Acute liver injury in COVID-19: prevalence and association with clinical outcomes in a large US cohort. Hepatology 2020. https://doi.org/10.1002/hep.31404.

[8] Weber S, Mayerle J, Irlbeck M, et al. Severe liver failure during SARS-Cov-2 infection. Gut 2020;69(7):1365–7.

[9] Wander P, Epstein M, Bernstein D. COVID-19 presenting as acute hepatitis. Am J Gastroenterol 2020;115(6):941–2.

[10] Zhai LL, Xiang F, Wang W, et al. Atypical presentations of coronavirus disease 2019 in a patient with acute obstructive suppurative cholangitis. Clin Res Hepatol Gastroenterol 2020. https://doi.org/10.1016/j.clinre.2020.05.003.

[11] Cheung S, Fuentes AD, Fetterman AD. Recurrent acute pancreatitis in a patient with COVID-19 infection. Am J Case Rep 2020;21:e927076-1–927076-3.

[12] Wang F, Wang H, Fan J, et al. Pancreatic injury patterns in patients with coronavirus disease 19 pneumonia. Gastroenterology 2020;159(1):367–70.

[13] Aloysius MM, Thatti A, Gupta A, et al. COVID-19 presenting as acute pancreatitis. Pancreatology 2020;20(5):1026–7.

[14] Afshar-Oromieh A, Prosch H, Schaefer-Prokop C, et al. A comprehensive review of imaging findings in COVID-19 - status in early 2021. Eur J Nucl Med Mol Imaging 2021. https://doi.org/10.1007/s00259-021-05375-3.

[15] Bütikofer S, Lenggenhager D, Garcia PDW, et al. Secondary sclerosing cholangitis as cause of persistent jaundice in patients with severe COVID-19. Liver Int 2021. https://doi.org/10.1111/liv.14971.

[16] Chiu CY, Sarwal A, Mon AM, et al. Gastrointestinal: COVID-19 related ischemic bowel disease. J Gastroenterol Hepatol 2021;36(4):850. https://doi.org/10.1111/jgh.15254.

[17] Keshavarz P, Rafiee F, Kavandi H, et al. Ischemic gastrointestinal complications Of COVID-19: A systematic review on imaging presentation. Clin Imaging 2020;73:86–95.

[18] Palacios S, Schiappacasse G, Valdes R, et al. COVID-19: Abdominal and pelvic imaging findings: a primer for radiologists. J Comput Assist Tomogr 2021. https://doi.org/10.1097/RCT.0000000000001152.

[19] Tahtabasi M, Hosbul T, Karaman E, et al. Does COVID-19 cause an increase in spleen dimensions? possible effects of immune activation, hematopoietic suppression and microthrombosis. Clin Imaging 2021;79:104–9.

[20] Taya M, Paroder V, Redelman-Sidi G, et al. Abdominal imaging findings on computed tomography in patients acutely infected with SARS-Cov-2: what are the findings?

Emerg Radiol 2021. https://doi.org/10.1007/s10140-021-01986-3.
[21] Koç ES, Çiçek B. COVID-19 induced haemobilia: a novel entity. J Gastrointest Liver Dis JGLD 2021;30(4):528–30.
[22] Parasa S, Desai M, Thoguluva Chandrasekar V, et al. Prevalence of gastrointestinal symptoms and fecal viral shedding in patients with coronavirus disease 2019: a systematic review and meta-analysis. JAMA Netw Open 2020;3(6):e2011335.
[23] Sultan S, Altayar O, Siddique SM, et al. AGA institute rapid review of the gastrointestinal and liver manifestations of COVID-19, meta-analysis of international data, and recommendations for the consultative management of patients with COVID-19. Gastroenterology 2020;159(1):320–34.e27.
[24] Khader M, Al Bishawi A, Kambal A, et al. SARS-Cov-2 infection presenting as colitis with chest and abdomen CT findings. Radiol Case Rep 2020;15(11):2427–32.
[25] Cheung KS, Hung IFN, Chan PPY, et al. Gastrointestinal manifestations of SARS-cov-2 infection and virus load in fecal samples from a hong kong cohort: systematic review and meta-analysis. Gastroenterology 2020;159(1):81–95.
[26] Qi F, Qian S, Zhang S, et al. Single cell RNA sequencing of 13 human tissues identify cell types and receptors of human coronaviruses. Biochem Biophys Res Commun 2020;526(1):135–40.
[27] Yang JK, Lin SS, Ji XJ, et al. Binding of SARS Coronavirus to its receptor damages islets and causes acute diabetes. Acta Diabetol 2010;47(3):193–9.
[28] Funt SA, Cohen SL, Wang JJ, et al. Abdominal pelvic CT findings compared between COVID-19 positive and COVID-19 negative patients in the emergency department setting. Abdom Radiol N Y 2020. https://doi.org/10.1007/s00261-020-02796-w.
[29] Zhang ML, Jacobsen F, Pepe-Mooney BJ, et al. Clinicopathologic findings in COVID-19-associated ischemic enterocolitis. Histopathology 2021. https://doi.org/10.1111/his.14457.
[30] Al Argan RJ, Alqatari SG, Al Said AH, et al. Gastrointestinal perforation secondary to COVID-19: Case reports and literature review. Medicine (Baltimore). 2021 May 14;100(19):e25771.
[31] Chauhan D, Kilic Y, Segal JP, et al. An unusual cause of gastrointestinal perforation in an adolescent patient with beta-thalassemia on deferasirox and SARS-cov-2 infection. J Hematol 2021;10(2):76–9.
[32] Iqbal U, Anwar H, Siddiqui HU, et al. Acute gastrointestinal bleeding in COVID-19 patients: a systematic review and meta-analysis. Clin Endosc 2021. https://doi.org/10.5946/ce.2021.071.
[33] Marasco G, Maida M, Morreale GC, et al. Gastrointestinal bleeding in COVID-19 patients: a systematic review with meta-analysis. Can J Gastroenterol Hepatol 2021;2021:2534975.
[34] Guglielmo FF, Wells ML, Bruining DH, et al. Gastrointestinal bleeding at CT angiography and CT enterography: imaging atlas and glossary of terms. Radiographics 2021;41(6):1632–56.
[35] Fulwadhva UP, Wortman JR, Sodickson AD. Use of dual-energy CT and iodine maps in evaluation of bowel disease. Radiographics 2016;36(2):393–406.
[36] Chen N, Zhou M, Dong X, et al. Epidemiological and clinical characteristics of 99 cases of 2019 novel coronavirus pneumonia in Wuhan, China: a descriptive study. Lancet Lond Engl 2020;395(10223):507–13.
[37] Bloom PP, Meyerowitz EA, Reinus Z, et al. Liver biochemistries in hospitalized patients with COVID-19. Hepatology 2021;73(3):890–900.
[38] Edwards K, Allison M, Ghuman S. Secondary sclerosing cholangitis in critically ill patients: a rare disease precipitated by severe SARS-Cov-2 infection. BMJ Case Rep 2020;13(11):e237984. https://doi.org/10.1136/bcr-2020-237984.
[39] Goldberg-Stein S, Fink A, Paroder V, et al. Abdominopelvic CT findings in patients with novel coronavirus disease 2019 (COVID-19). Abdom Radiol N Y 2020;1–11. https://doi.org/10.1007/s00261-020-02669-2.
[40] Beswick DM, Miraglia R, Caruso S, et al. The role of ultrasound and magnetic resonance cholangiopancreatography for the diagnosis of biliary stricture after liver transplantation. Eur J Radiol 2012;81(9):2089–92.
[41] Scarabelli A, Zilocchi M, Casiraghi E, et al. Abdominal computed tomography imaging findings in hospitalized COVID-19 patients: a year-long experience and associations revealed by explainable artificial intelligence. J Imaging 2021;7(12):258.
[42] Singh P, Singh SP, Verma AK, et al. A systematic review of abdominal imaging findings in COVID-19 patients. Visc Med 2021;26:1–12. https://doi.org/10.1159/000518473.
[43] Murray N, Darras KE, Walstra FE, et al. Dual-energy CT in evaluation of the acute abdomen. Radiographics 2019;39(1):264–86.
[44] Gelbmann CM, Rümmele P, Wimmer M, et al. Ischemic-like cholangiopathy with secondary sclerosing cholangitis in critically ill patients. Am J Gastroenterol 2007;102(6):1221–9.
[45] Laurent L, Lemaitre C, Minello A, et al. Cholangiopathy in critically ill patients surviving beyond the intensive care period: a multicentre survey in liver units. Aliment Pharmacol Ther 2017;46(11–12):1070–6.
[46] Jackson CB, Farzan M, Chen B, et al. Mechanisms of SARS-Cov-2 entry into cells. Nat Rev Mol Cell Biol 2022;23(1):3–20.
[47] Zhou L, Niu Z, Jiang X, et al. SARS-Cov-2 Targets by The Pscrna Profiling of ACE2, TMPRSS2 And Furin Proteases. Iscience 2020;23(11):101744.
[48] Ihlow J, Seelhoff A, Corman VM, et al. COVID-19: a fatal case of acute liver failure associated with SARS-Cov-2 infection in pre-existing liver cirrhosis. BMC Infect Dis 2021;21(1):901.
[49] Pirisi M, Rigamonti C, D'Alfonso S, et al. Liver Infection and COVID-19: the electron microscopy proof and

revision of the literature. Eur Rev Med Pharmacol Sci 2021;25(4):2146–51.
[50] Faruqui S, Okoli FC, Olsen SK, et al. Cholangiopathy after severe COVID-19: clinical features and prognostic implications. Am J Gastroenterol 2021;116(7):1414–25.
[51] Tafreshi S, Whiteside I, Levine I, et al. A case of secondary sclerosing cholangitis due To COVID-19. Clin Imaging 2021;80:239–42.
[52] Klindt C, Jensen BE, Brandenburger T, et al. Secondary sclerosing cholangitis as a complication of severe COVID-19: a case report and review of the literature. Clin Case Rep 2021;9(5):e04068.
[53] Roth NC, Kim A, Vitkovski T, et al. Post-COVID-19 cholangiopathy: a novel entity. Am J Gastroenterol 2021; 116(5):1077–82.
[54] Anderson MA, Goiffon RJ, Lennartz S, et al. Abdominal imaging utilization during the first COVID-19 surge and utility of abdominal MRI. Tomogr Ann Arbor Mich 2021;7(4):972–9.
[55] Faqeeh S, Madkhali R. Acute reversible renal failure requiring temporary dialysis in a patient with COVID-19. Radiol Case Rep 2020;15(11):2449–52.
[56] Lushina N, Kuo JS, Shaikh HA. Pulmonary, cerebral, and renal thromboembolic disease in a patient with COVID-19. Radiology 2020;296(3):E181–3.
[57] Knefati M, Ganim I, Schmidt J, et al. COVID-19 with an initial presentation of intraperitoneal hemorrhage secondary to spontaneous splenic rupture. Cureus 2021; 13(5):e15310.
[58] Dane B, Smereka P, Wain R, et al. Hypercoagulability in COVID-19: identification of arterial and venous thromboembolism in the abdomen, pelvis, and lower extremities. Am J Roentgenol 2020. https://doi.org/10.2214/AJR.20.23617 AJR.20.23617.
[59] Musoke N, Lo KB, Albano J, et al. Anticoagulation and bleeding risk in patients with COVID-19. Thromb Res 2020;196:227–30.
[60] Qiu C, Li T, Wei G, et al. hemorrhage and venous thromboembolism in critically ill patients with COVID-19. SAGE Open Med 2021;9. https://doi.org/10.1177/20503121211020167.
[61] Levi M, Thachil J, Iba T, et al. Coagulation abnormalities and thrombosis in patients with COVID-19. Lancet Haematol 2020;7(6):e438–40.
[62] Kolli S, Oza VM. SARS-cov-2 and portal vein thrombosis: a rare gastrointestinal manifestation of COVID-19. Cureus 2021;13(4):e14340.