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Imaging of Coronavirus Disease 2019 Infection From Head to Toe: A Primer for the Radiologist

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A B S T R A C T

Coronavirus Disease 2019 (COVID-19) disease has rapidly spread around the world after initial identification in Wuhan, China, in December 2019. Most common presentation is mild or asymptomatic disease, followed by pneumonia, and rarely- multiorgan failure and Acute Respiratory Distress Syndrome (ARDS). Knowledge about the pathophysiology, imaging and treatment of this novel virus is rapidly evolving due to ongoing worldwide research. Most common imaging modalities utilized during this pandemic are chest radiography and HRCT with findings of bilateral peripheral, mid and lower zone GGO and/or consolidation, vascular enlargement and crazy paving. HRCT is also useful for prognostication and follow-up of severely ill COVID-19 patients. Portable radiography allows follow-up of ICU patients & obviates the need of shifting critically ill patients and disinfection of CT room. As the pandemic has progressed, numerous neurologic manifestations have been described in COVID-19 including stroke, white matter hyperintensities and demyelination on MRI. Varying abdominal presentations have been described, which on imaging either show evidence of COVID-19 pneumonia in lung bases or show abdominal findings including bowel thickening and vascular thrombosis. Numerous thrombo-embolic and cardiovascular complications have also been described in COVID-19 including arterial and venous thrombosis, pulmonary embolism and myocarditis. It is imperative for radiologists to be aware of all the varied faces of this disease on imaging, as they may well be the first physician to suspect the disease. This article aims to review the multimodality imaging manifestations of COVID-19 disease in various organ systems from head to toe.

Introduction

First reported in Wuhan province of China in December 2019, the novel Coronavirus 2019 (COVID-19), also known as severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) rapidly spread worldwide leading to a substantial public health crisis. The worst affected countries are the USA, Brazil, and India, the total number of cases in the world being over 167 million as of May 24, 2021.[1] Common COVID-19 symptoms are fever, cough, dyspnea, myalgia, fatigue, and anosmia. As the pandemic has progressed, abdominal symptoms like loss of appetite, nausea, vomiting, diarrhea, and abdominal pain have also been noted[1]. An early study reported that approximately 81% of patients of COVID-19 had mild symptoms, 14% had severe disease, including pneumonia and dyspnea, and 5% had critical illness including multiorgan failure and respiratory failure.[2] It has been reported that COVID-19 disease predisposes to a prothrombotic state marked by elevated D-dimer and thrombocytopenia, potentially resulting in thromboembolic complications. In addition, an immune-mediated cytokine storm in the second week of this viral illness may set into motion severe complications like ARDS and multiorgan disease, which may be fatal. The gold standard for the diagnosis of COVID-19 is real time polymerase chain reaction (RT-PCR) on nasopharyngeal and/or oropharyngeal swab; however, its sensitivity is reported to be only 30-60 % due to limitations of sample collection, transportation, and kit performance.[3] Radiography is insensitive in identifying early disease but has a valuable role in the follow-up and prognostication of diagnosed patients. Typical chest CT findings of COVID-19 pneumonia are peripheral, multifocal GGO, and consolidative opacities.

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Chen et al[4] reported 12 of 33 COVID patients testing negative on RT-PCR initially, showing CT features of COVID-19 disease and being confirmed on subsequent repeat RT-PCR. A study by Meng et al[5] described abnormal CT findings in 58 asymptomatic COVID-19 patients at presentation, although 16 of them later developed symptoms. COVID-19 infection is increasingly being described as a multiorgan disease rather than just viral pneumonia. There have been numerous reports of the disease causing thromboembolic, gastrointestinal, renal, cardiovascular, and neurologic complications. Prior literature has focused chiefly on imaging of pneumonia with CT and radiography. In this review, we wish to describe the imaging manifestations of COVID-19 disease in the various organ systems of the human body.

Pathogenesis of COVID-19

The inhaled virus SARS-CoV-2 binds to the epithelial cells of the nasal cavity and starts multiplying, following which it enters alveolar epithelial cells in the lung by binding to angiotensin-converting enzyme 2 (ACE-2) receptors. Cell entry is via binding of the spike protein of SARS-CoV-2 virus to the ACE-2 receptor.[5] ACE-2 receptor is expressed in various sites apart from the lung epithelium, including cardiovascular endothelium, small bowel, liver, biliary epithelial cells, pancreatic islet cells, kidney, and brain, making these organs prone to injury. It has been postulated that receptor expression may potentiate cytotoxic effects by aiding local viral infection and replication.

Due to the destruction of cells expressing ACE-2, there is relative ACE-2 deficiency leading to derangement of Renin-Angiotensin system, elevated Angiotensin-II, and reduced Angiotensin1-7, which potentiates endothelial inflammation, thrombosis, and hypertension.[6] This may also play a role in multiorgan injury to the heart, kidney, and liver.

COVID-19 is recognized as a multisystem disease due to its affinity for ACE-2 receptor and cytokine-mediated inflammation. Early investigations suggest that the most common laboratory findings are elevated D-dimer levels, mild thrombocytopenia, and PT prolongation, which are associated with increased mortality.[6,7] Decreased lymphocyte count due to direct cytotoxic action, leukocytosis, and neutrophilia due to hyperinflammatory response is also seen in COVID-19 patients.[7] Patients with COVID-19 show an increased risk of vascular thrombosis, manifesting as deep vein thrombosis, pulmonary embolism, arterial stroke, and visceral or peripheral artery thrombosis. Above hemostatic abnormalities are primarily related to an inflammatory syndrome characterized by elevated cytokines, including IL-1 and IL-6, referred to as thromboinflammation.[8] The various system involvement is described below with its imaging features.

CNS Involvement

The most common neurologic symptoms reported in COVID-19 are headache, altered consciousness, pathologic wakefulness after sedation, agitation, and confusion. Jain *et al.*[7] and Mahammedi *et al.*[9]reported that the most common positive neuroimaging studies revealed stroke and the former described that finding of either large ischemic or hemorrhagic stroke was associated with high mortality Figure 1 and Figure 3 Chougar *et al.*[10] found several cerebrovascular complications such as ischemic stroke, microhemorrhages, deep vein thrombosis, cytotoxic lesions of corpus callosum, multifocal white matter enhancing lesions, and basal ganglia abnormalities in COVID-19 patients.

Kandemirli *et al.*[11] found increased cortical signal on FLAIR (Fluid-attenuated inversion recovery), cortical diffusion restriction, cortical blooming, leptomeningeal enhancement, and subcortical and deep white matter abnormality. Radmanesh *et al.*[12] described 2 patterns of MRI findings, one being deep and subcortical white matter hyperintensity with mild diffusion restriction and the other one being diffuse subcortical and callosal microhemorrhages Figure 2. Kremer *et al.*[13] described mainly 3 patterns of MRI findings after excluding ischemic infarcts-medial temporal lobe hyperintensity, non−confluent white matter hyperintensities with diffusion restriction with or without enhancement with or without microhemorrhages and isolated widespread microhemorrhages.

The various proposed mechanisms for neurologic implications include the following - Direct central nervous system spread, and neurotropism is supported by evidence of earlier SARS-CoV strains gaining access to the CNS via olfactory pathways or the bloodstream and known to cause meningitis and encephalitis.[14] Another proposed etiology is indirect neurologic involvement due to an excessive systemic proinflammatory response, which may cause widespread dysregulation of homeostasis with coagulopathy and increase the risk of acute cerebrovascular diseases.

COVID-19 may also lead to parainfectious autoimmune-based neurologic complications such as acute disseminated encephalomyelitis (ADEM) and Guillain Barré syndromes, which are recognized complications of microbial infections.[15,16] Lastly, since most patients were admitted to ICUs forARDS, more general assumptions may also be considered, such as delayed post-hypoxic leukoencephalopathy, metabolic or toxic encephalopathy, and posterior reversible encephalopathy syndrome (PRES).[17,18] Various patterns of neurologic abnormalities in COVID-19 patients are summarized in Table 1.

Pulmonary Involvement

Common clinical symptoms of COVID19 pneumonia include fever, cough, and expectoration, while few present with vomiting, dyspnea, and low oxygen saturation levels.[19] Fleishner society recommends imaging in symptomatic COVID-19 patients with worsening respiratory status or those with moderate and/or severe disease. Imaging is not recommended in asymptomatic and/or mild patients or for primary diagnosis or screening of patients.[20] Chest radiographs were found to be insensitive in diagnosing COVID-19 pneumonia; however, findings of multifocal haziness and consolidations have been described.[21] With the progression of pneumonia, consolidations may become more confluent with an ARDS type diffuse involvement in later stages (Fig 4).

Point of care ultrasound is a useful diagnostic and prognostic tool for COVID-19, particularly in ICU and isolation wards.[22] Lung ultrasound findings in COVID-19 include thickened pleural lines(shred sign), vertically oriented echogenic B lines, multifocal consolidations with air bronchograms, and A-lines during the recovery phase.[23,24]

Literature of COVID-19 pneumonia has been focused on chest CT due to its high sensitivity to diagnose lung abnormalities. Tao et al[3] described a high sensitivity of CT to diagnose COVID-19 with RT-PCR as the gold standard. They also found positive CT findings in 75 % of patients in a group of 413 suspected COVID patients with a negative RT-PCR. Similar findings were reported by Korkmaz *et al.*[25] This underscores the need to utilize CT as a problem-solving tool in the present scenario. CT findings of COVID-19 have been described as multifocal bilateral GGO and/or mixed GGO- consolidation with rounded morphology and bilateral, multifocal, peripheral, and lower lobe distribution.[26,27] (Fig 5) Other common features of interlobular septal thickening, air bronchogram, and vascular enlargement and less common findings of reverse halo, nodule with halo, subpleural line sign, subpleural transparent line sign, fibrotic streaks, bronchiectasis, and bronchus deformation were also described. (Fig 6A-B). A variant of reverse halo sign – bull's eye sign has been described in COVID-19, which consists of a central ground-glass nodule surrounded by an inner ring of air and an outer ring of ground glass. These likely represent areas of organizing pneumonia.[28] Pleural effusion, pericardial effusion, lymphadenopathy, cavitation, and pneumothorax are uncommon findings that may be seen on disease progression.[29,30]

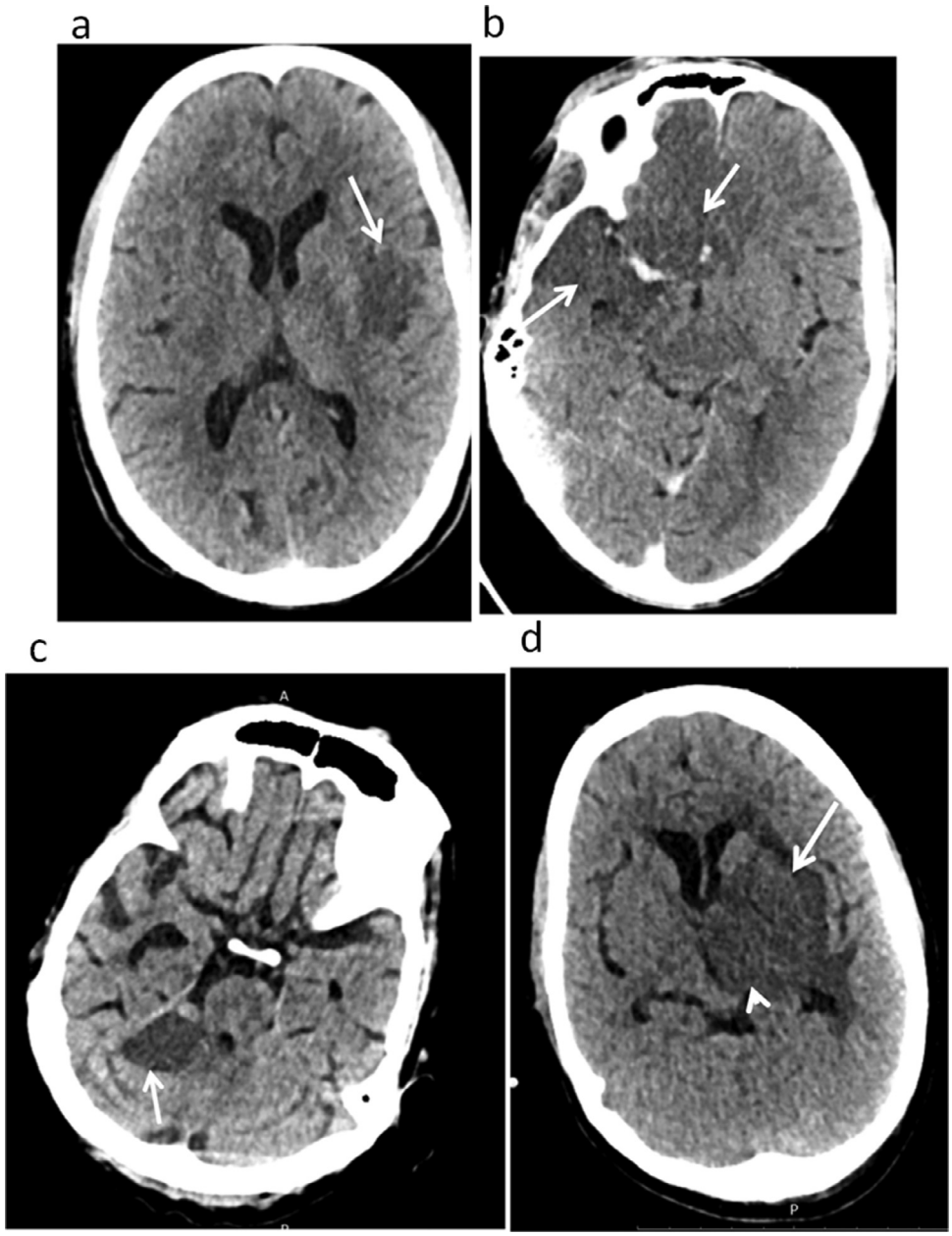

FIG 1. Ischemic infarction in 4 different COVID-19 patients (A) Axial NCCT in a 33 years old male reveals hypodensity in left basal ganglia and external capsule region suggesting acute MCA territory infarction. (B) Axial NCCT in a 50 years old man reveals extensive hypodensity in right basifrontal and temporal lobes possibly involving both ACA and MCA territory. (C) Axial NCCT at the level of posterior fossa in an 84 years old male reveals hypodensity in right superior cerebellum suggesting acute infarct. (D) Axial NCCT in a 27 years old woman reveals hypodensity in left basal ganglia (arrow) and thalamus (arrowhead) suggesting infarction.

Temporal follow-up of CT findings has revealed initial unifocal GGO followed by an increase in number and size of GGO, progressing to mixed GGO-consolidation with or without crazy paving pattern reaching a peak around ten day's, thereafter showing slow resolution with linear subpleural consolidative opacities or reverse halo sign which is typical to organizing pneumonia (Fig 7).[29,31] Pulmonary fibrosis may develop with time, which manifests as mild to severe subpleural and peribronchovascular fibrosis, macrocystic lung changes, and bronchiectasis. CT helps evaluate post-COVID fibrosis, especially in those patients who present with persistent cough and dyspnea (Fig 6C-F).[32,33]

Vascular and perfusion abnormalities, including dilatation, tortuosity of pulmonary vessels not limited to the area of GGO, mosaic perfusion, focal oligemia, and focal hyperemia have been identified in CT images of COVID-19 patients. The typical response to pneumonia is hypoxic vasoconstriction in the consolidated lung. In COVID-19, there is inflammation-mediated paradoxical vascular dilatation in the consolidated lung. This leads to shunting of blood to poorly ventilated areas of lung parenchyma, which further exacerbates hypoxia due to ventilation-perfusion mismatch.[34]

The various pulmonary findings of COVID-19 are summarized in Table 2.

Cardiovascular Involvement

Various reports of coagulopathy and acute pulmonary embolism (PE) have been published in recent times, indicating a higher risk of thromboembolic events in COVID-19 pneumonia. An elevated risk of developing PE was associated with these factors- obesity, elevated D-dimer, elevated CRP, and rising D-dimer values over time (Fig 8).[35]

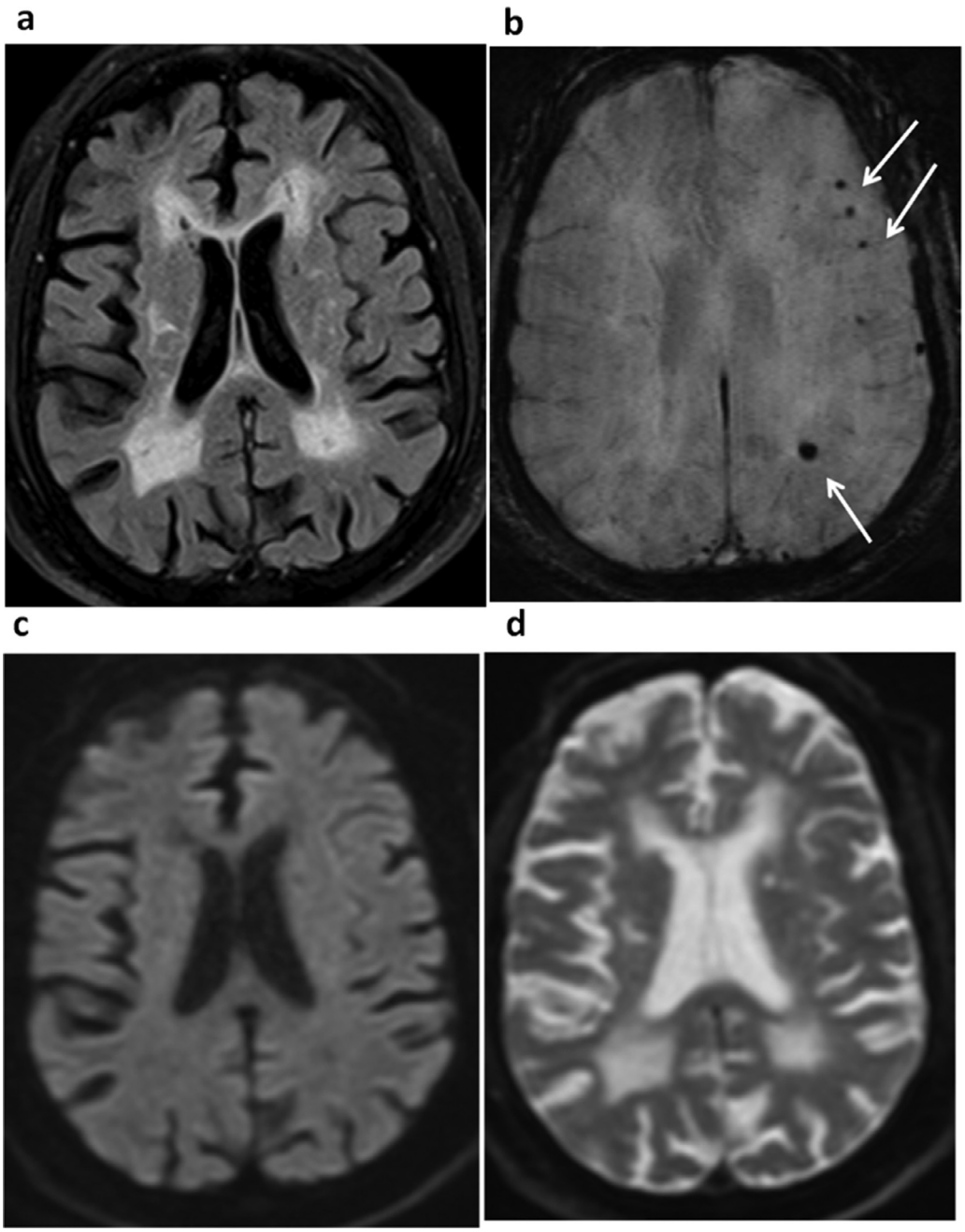

FIG 2. Diffuse leukoencephalopathy in a 64 years old male admitted in ICU. (A) Axial FLAIR MR reveals diffuse confluent white matter hyperintensity in bilateral corona radiata and centrum semiovale (not shown). (B) Axial GRE (Gradient echo) reveals multiple microhemorrhages in left gray-white matter junction (arrows). (C) Diffusion weighted imaging (DWI) B = 1000 and (D) Apparent diffusion coefficient (ADC) map reveals no diffusion restriction.

In a study comprising 100 COVID-19 patients, a 23% prevalence of acute pulmonary thromboembolism was found.[36] Another study showed that patients with COVID-19 infection undergoing lower extremity CT angiography (CTA) had a higher propensity for lower extremity clots when compared to the controls.[37] A study on 81 critically ill patients found the incidence of venous thromboembolism (VTE) to be 25%.[38]

COVID-19 predisposes to arterial and venous thrombosis, the imaging features of which are not specific and appear similar to those associated with other diseases (Fig 9). However, if radiologists are familiar with its prevalence and pathogenesis in these patients, it can aid in early detection and a more favorable outcome. Since these patients undergo aggressive anticoagulation therapy, there is a possibility of increased incidence of bleeding complications which can be diagnosed on imaging.

COVID-19 can affect the cardiovascular system through direct and indirect mechanisms, such as myocardial injury, acute coronary syndromes (ACS), right ventricular dysfunction and/or acute cor pulmonale, arrhythmias, and cardiogenic shock.[39] There have been reports of clinically diagnosed myocarditis with supplemental imaging findings.[40,41] Figures 10 and 11 Abnormalities of the cardiac rhythm including new-onset atrial fibrillation, sinus node dysfunction, complete heart block, and ventricular arrhythmias have also been described.[19]

Puntmann *et al.*[42] found contrast-enhanced MRI (CEMRI) evidence of cardiovascular involvement and myocardial inflammation in 78 % and 60% respectively of recently recovered COVID-19 patients without any relation to severity and course of acute illness or preexisting conditions. The CEMRI abnormalities detected were elevated native

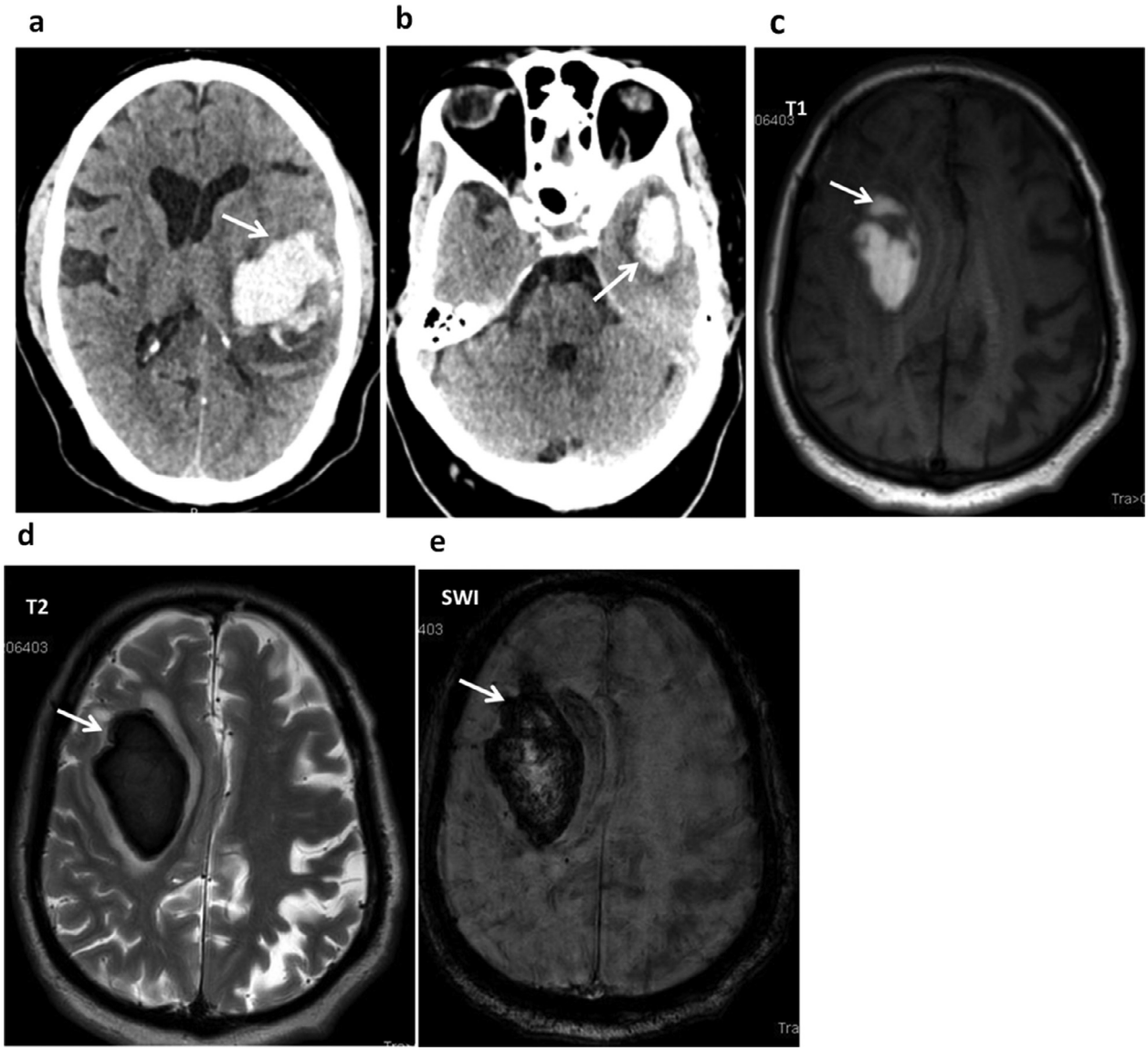

FIG 3. Intracranial hemorrhage. (A,B) Axial NCCT in a 30 years old man reveals a large hyperdense hematoma in left parietotemporal lobe (arrows in A, B) with perilesional edema and midline shift. (C,D,E) Early subacute hematoma in a 72 years old man. (C)Axial T1W image reveals T1 hyperintense lesion in right frontal lobe (arrow) with surrounding mass effect. (D) Axial T2 W image reveals corresponding hypointense signal and (E) SWI image reveals blooming consistent with blood products.

TABLE 1
Neurologic complications of COVID-19 infection.

	Complications and MRI manifestations	Proposed etiology
1	Cerebrovascular complications including ischemic, hemorrhagic stroke and deep vein thrombosis	Virus associated coagulopathy
2	Basal ganglia abnormalities	Immune-mediated
3	Cytotoxic lesion of the corpus callosum	Inflammatory response to COVID-19[75]
4	FLAIR hyperintensity in medial temporal lobes	Neuroinvasion [76]
5	Multifocal bilateral non-confluent white matter hyperintensities +/-diffusion restriction+/- enhancement+/- hemorrhage.	• Post-infective demyelination due to the immunologic process. • Supported by imaging similarities to ADEM. • Also reinforced by a recent neuropathologic study that described ADEM-like lesions in the subcortical WM in a patient with severe COVID-19.
6	Isolated bilateral microhemorrhages	Disseminated intravascular coagulation(DIC), hypoxia or small vessel vasculitis
7	Cortical FLAIR hyperintensity with diffusion restriction+/- cortical blooming +/-leptomeningeal enhancement[11]	Neurotropism, cytokine storm, hypoxia
8	Diffuse confluent white matter hyperintensity	Delayed post hypoxic leukoencephalopathy or post-infectious demyelination
9	Acute necrotizing hemorrhagic encephalopathy [77] The case report revealed bilateral thalamic, medial temporal, and subinsular regions with hemorrhagic change & rim enhancement.	Breach in the blood-brain barrier due to cytokine storm rather than demyelination. [78]

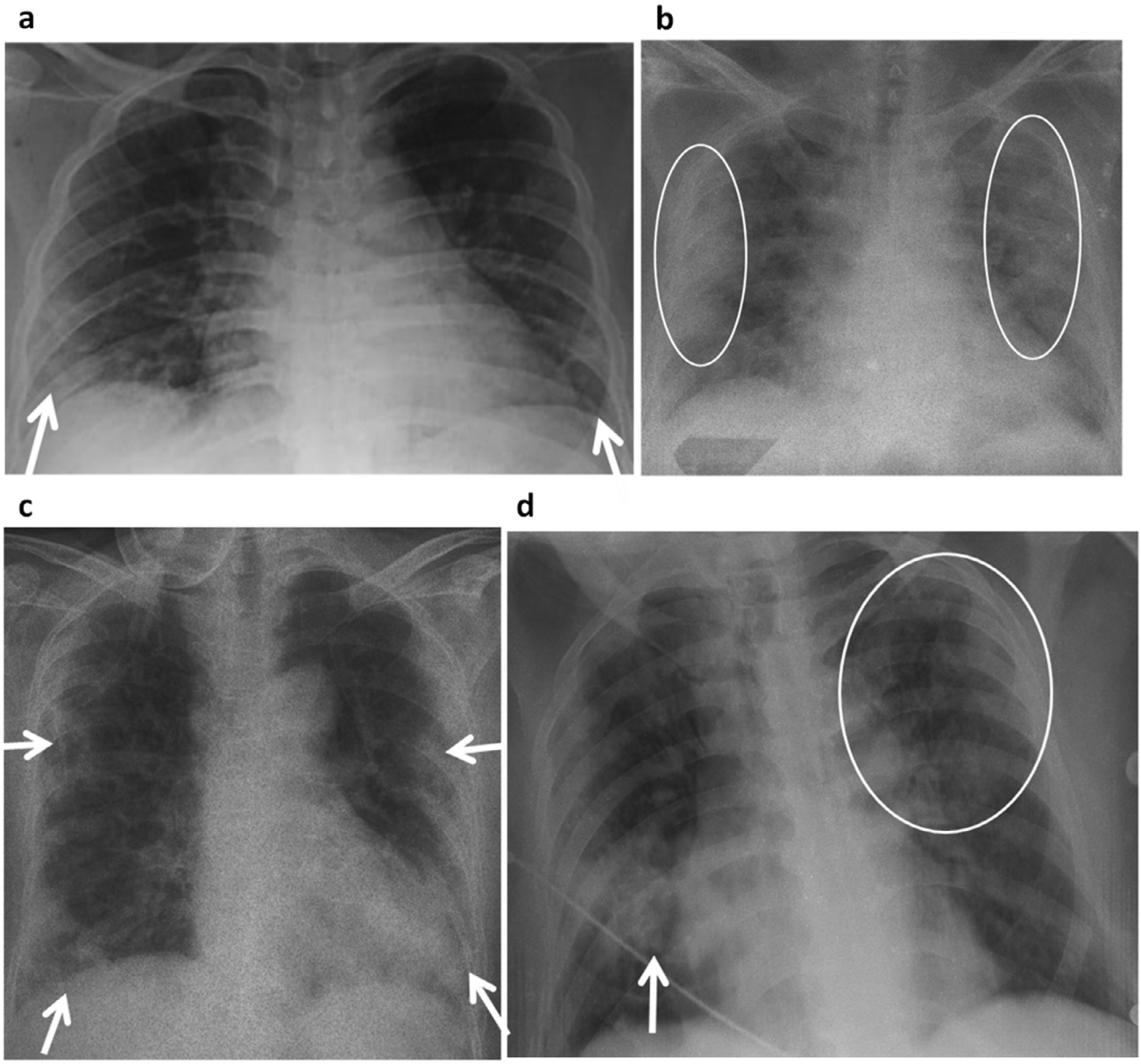

FIG 4. Radiographic manifestations of COVID-19 pneumonia. (A) 26-year-old male reveals bilateral peripheral GGO in lower zones (arrows). (B) 57-year-old female reveals bilateral peripheral predominant consolidations (encircled) in all the zones. (C) 60-year-old male shows bilateral peripheral consolidations (arrows) in all the zones with scattered reticular opacities and background emphysema (D) 72-year-old male reveals right mid-zone rounded consolidation (arrow) and left mid-zone haziness (encircled).

T1, elevated native T2, delayed myocardial enhancement, and pericardial enhancement.[42]

A multitude of factors has been recognized in the pathophysiology of cardiovascular manifestation. ACE-2 receptor is widely present in the cardiovascular tissue – in cardiac myocytes, fibroblasts, and endothelial cells, suggesting a role in a direct viral injury mechanism.[43,44] It has been proposed that patients who have preexisting cardiovascular disease have increased ACE-2 levels, predisposing them to complications.[45,46] Myocarditis has been suggested as an acute cause of cardiac dysfunction, and it has been postulated that the viral load correlates with the severity of myocardial injury. Another mechanism is cytokine storm or systemic inflammatory response syndrome, which can cause myocardial injury due to demand ischemia.[47] Troponin levels are often raised primarily in severe cases and are associated with poor prognosis. Increased pulmonary vascular pressure occurring secondary to ARDS and pulmonary thromboembolism can cause right ventricular dysfunction.[48] While various viral infections are a risk factor for myocardial infarction (MI),[49] the risk is higher with COVID-19, which could be related to the increased coagulability causing an increase in thrombotically mediated MI.

The various cardiovascular complications of COVID-19 are summarized in Table 3.

Multisystem Inflammatory Syndrome in Children (MIS-C)

Multisystem inflammatory syndrome in children (MIS-C was first described as a Kawasaki-like syndrome in patients with a history of prior contact with a COVID-19 patient. It has been proposed that MIS-C is the result of a post infectious phenomenon due to abnormal IgG antibody mediated enhancement of the disease. An alternative hypothesis suggests that coronaviruses due to their known ability to block interferon responses can result in a delayed cytokine storm leading to MIS-C.[50]

Case series and studies have described a broad spectrum of multisystem imaging findings in this syndrome, including perihilar bronchial wall thickening, perihilar or lower lobe consolidations, rapid development of pulmonary edema, ARDS, pulmonary embolism,

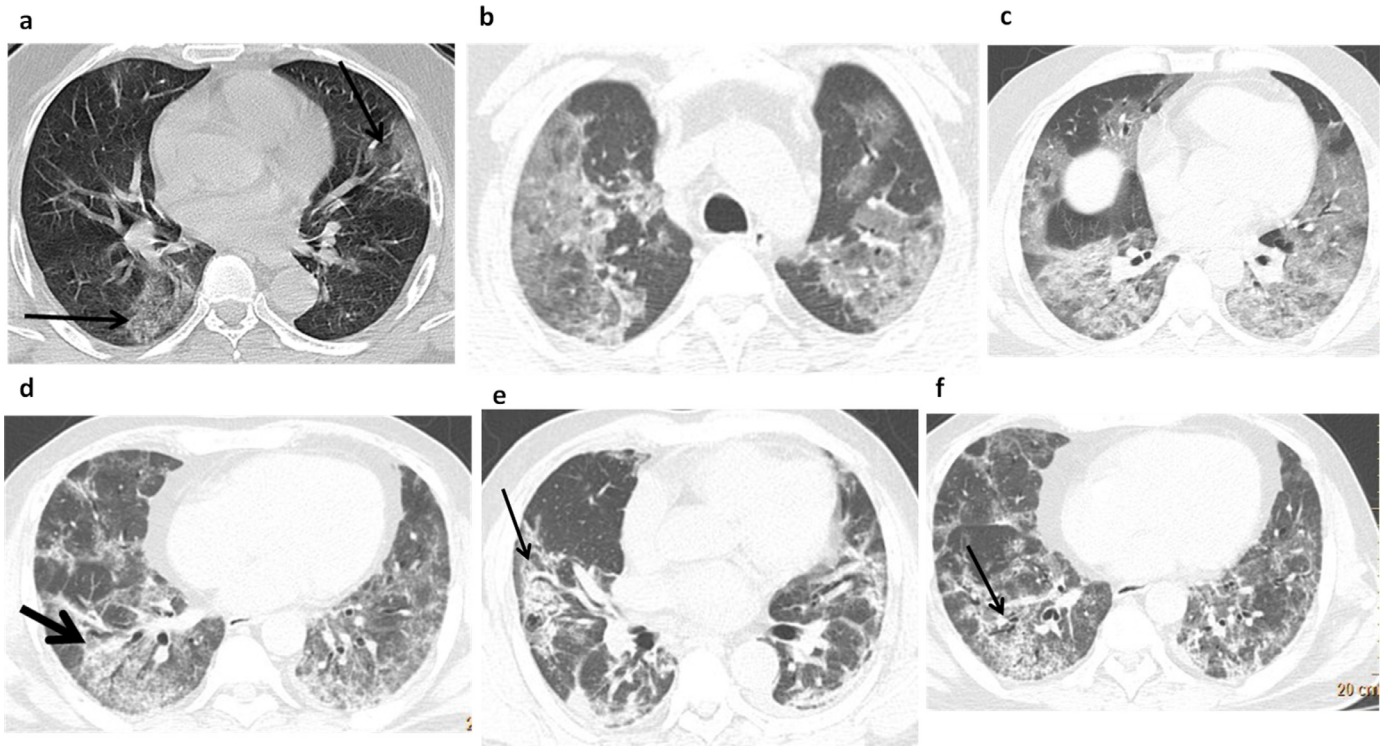

FIG 5. Typical chest CT findings of COVID-19 pneumonia in different patients. Axial CT images reveal (A) Early findings of peripheral rounded GGO (B, C) bilateral peripheral and lower lobe confluent GGO. Other commonly noted signs are (D) air bronchogram (arrow), (E) vascular enlargement (arrow), and (F) crazy paving (arrow).

cardiomegaly, coronary aneurysms, pericardial effusion, gall bladder wall thickening, splenic infarcts, echogenic kidneys, right iliac fossa bowel thickening, and mesenteric lymphadenopathy.[51,52] MIS-C related myocarditis imaging findings have been described on echocardiography to show transient systolic dysfunction and on cardiac MRI to show myocardial edema without enhancement.[53]

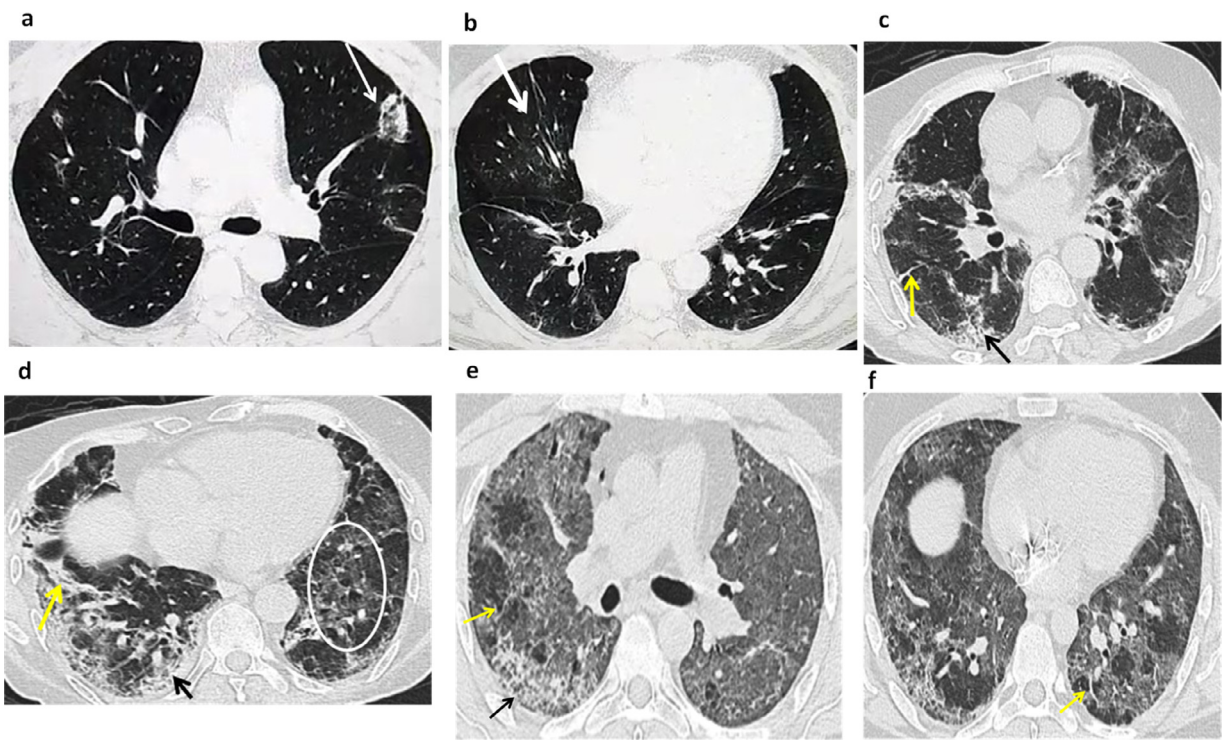

FIG 6. Residual CT findings in recovered patients. (A,B) Axial CT chest in a 59-year-old Post COVID-19 patient with residual symptoms reveals (A) reverse halo sign (arrow) and (B) bronchiectasis (arrow). (C,D) Axial CT chest in a 75-year-old Post COVID-19 patient with residual symptoms reveals resolving bilateral subpleural GGO (encircled D), reticular opacities, fibrotic bands (yellow arrows C,D), and subpleural honeycombing(black arrow C,D) in bilateral lower lobes, representing post-COVID fibrosis (E,F) Axial CT chest in a 34-year-old COVID-19 recovered patient with residual symptoms reveals bilateral widespread residual GGO, peripheral reticular opacities, fibrotic bands (yellow arrows E, F), subpleural honeycombing in right lower lobe (black arrow E) compatible with post COVID fibrosis (Color version of the figure is available online.).

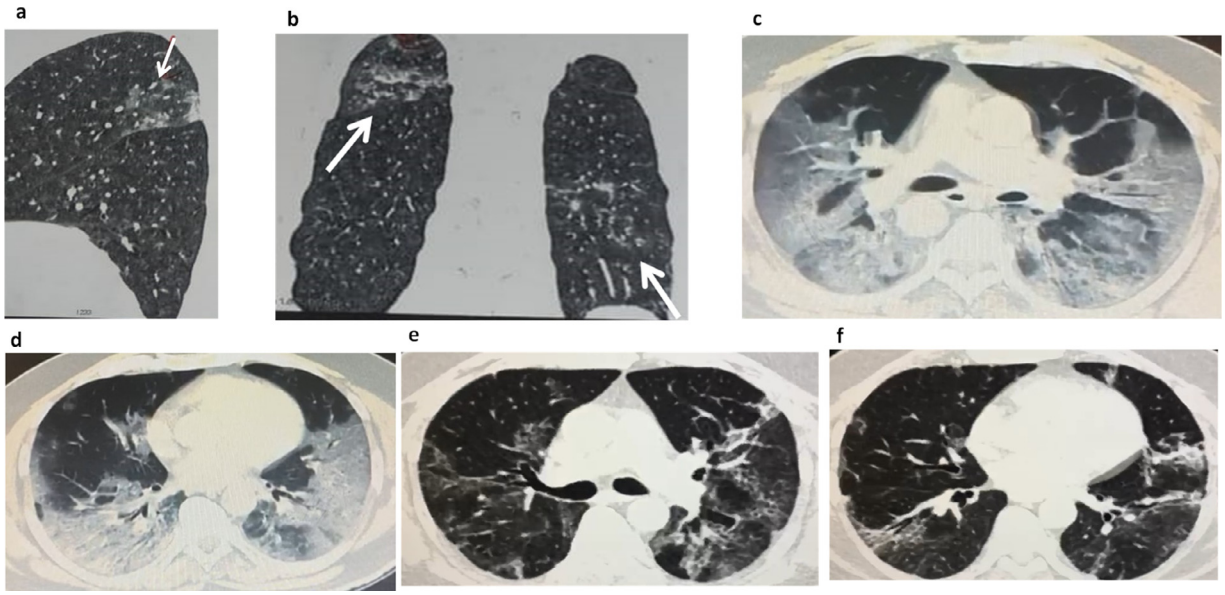

FIG 7. Temporal changes in COVID-19 pneumonia. (A,B) Sagittal and coronal reformatted CT chest in a 63-year-old patient who presented with cough and fever reveal mild patchy ground-glass opacities in bilateral lungs (arrows). (C,D) Repeat CT was done in the same patient after clinical worsening ten day's later. Axial images reveal radiological progression and confluent ground-glass opacities in bilateral lungs predominantly in dependent zones. Remdesevir was started at this point. (E,F) After 4 weeks from testing positive, follow-up axial CT reveals significant improvement with residual GGO, interlobular septal thickening and reticular opacities. (Color version of figure is available online).

TABLE 2
Pulmonary complications of COVID-19 infection.

	Complication and imaging manifestations	Proposed etiology
1	Pneumonia	Progressive pulmonary infiltration
	Round ground-glass opacity, consolidation in multifocal peripheral distribution	Organizing pneumonia pattern
	Common features of interlobular septal thickening, air bronchogram	
	Uncommon features of reverse halo, nodule with halo	
2	ARDS	Cytokine storm
3	Vascular enlargement	Increased blood supply due to inflammation
4	Post-COVID-fibrosis	Residual fibrosis after resorption of pneumonitis
	Mild to severe subpleural and peribronchovascular fibrosis, macrocystic lung changes, bronchiectasis.	
	Affects lung in previously involved sites.	
	Severe cases, diffuse lung involvement with potential candidate for lung transplant.	

GIT and Hepatobiliary Involvement

Tian et al[19] reported the most common gastrointestinal symptoms to be anorexia and diarrhea in adults and children respectively. Other gastrointestinal symptoms in COVID-19 patients included nausea, vomiting, gastrointestinal bleeding, and abdominal pain. A multicenter study by Pan *et al.*[54] reported that over 18% of COVID-positive patients presented with abdominal symptoms, and approximately 5% had isolated gastrointestinal symptoms without respiratory complaints.

Studies have reported findings of COVID-19 pneumonia in lung bases on abdominal imaging for non–respiratory clinical complaints, most commonly abdominal pain. This emphasizes that radiologists must pay attention to lung bases, especially during this pandemic, for early diagnosis, even when not clinically suspected.[55,56]

It was reported in a recent study that 57% of acutely ill COVID positive patients were found to have positive findings on abdominopelvic CT, most common of which were small bowel thickening, solid organ infarctions, and vascular thrombosis Figure 12A-D).[57]

Other related findings of bowel wall hyper-enhancement, pneumatosis, portal venous gas, and fluid-filled colon have been described in literature. On surgery and histopathology, some of these have correlated with yellow discoloration of bowel, ischemic bowel, and fibrin thrombi within the necrotic bowel.[58,59]

ACE-2 receptors are found throughout the gastrointestinal tract, with the most significant functional role in the small bowel and colon. SARS-CoV-2 binding to bowel epithelial cells leads to mucosal inflammation (e.g., enteritis). Viral load causes alterations in intestinal microbial flora and cellular regulation of fluid and electrolytes, leading to diarrhea. There have been few case reports of COVID-19 patients presenting with abdominal pain, being diagnosed with epiploic appendagitis. Possible etiology of this condition is inflammation and venous thrombosis, both known associations of COVID-19[60,61] (Fig 12E-F).

Almost 40% of COVID-positive patients have abnormal liver function tests (LFT) on admission, and LFT abnormalities are associated with higher fevers, higher levels of C-reactive protein, and longer hospital stay.[62] Liver dysfunction is likely secondary to the virus's binding to hepatic ACE-2 receptors causing an immune cytokine storm, leading to hepatocellular inflammation and damage of hepatocytes. The imaging correlate of liver dysfunction is hepatic steatosis which has been described on ultrasound in COVID-19 patients. A study has demonstrated that COVID19 patients with high body mass index and non–alcoholic fatty liver disease (NAFLD) has a greater risk for both progression in liver damage and COVID-19. Progression of liver damage in chronic liver diseases in the setting of COVID-19 may be complicated by hepatic failure, hepatic encephalopathy, and gastrointestinal bleeding to diagnose which imaging would be helpful.[63]

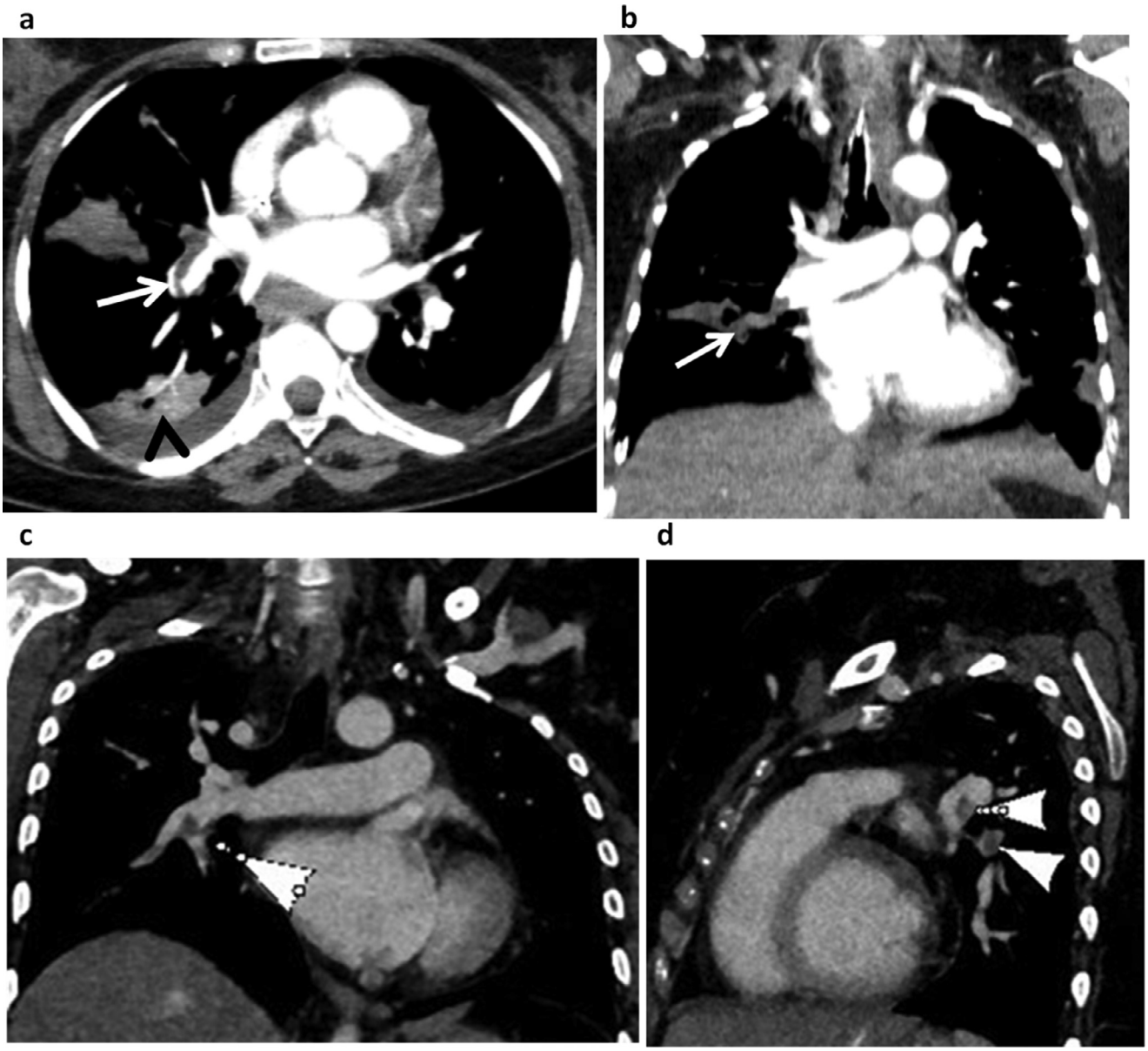

FIG 8. Pulmonary embolism. (A,B) Axial (A) and coronal reformatted (B) CT Pulmonary angiography (CTPA) images reveal a hypodense filling defect in the right interlobar pulmonary artery (arrow A,B). Bilateral mild pleural effusion and right basal atelectasis (arrowhead A) also noted. (C,D) Coronal (C) and Sagittal (D) reformatted CTPA images in a different patient shows filling defects in the right interlobar pulmonary artery and its segmental branches (arrowheads).

TABLE 3
Cardiovascular complications of COVID-19 infection.

	Complications and imaging manifestations	Proposed etiology
1	Acute pulmonary embolism, venous thromboembolism, Arterial thrombosis —role of CTPA and CT Angiography	Increased coagulability
2	Acute coronary syndromes	Increased coagulability
3	Cardiomyopathy	Direct myocardial injury or immune-mediated reaction
4	Acute cor pulmonale	Secondary to ARDS and increased pulmonary vascular pressure, Pulmonary thromboembolism
5	Arrhythmia	Hypoxic state due to ARDS or myocarditis
6	Cardiogenic shock	Myocarditis and/or myocardial infarction
7	Myocarditis Myocardial edema and delayed enhancement on CE-MRI	Direct injury (myocardial inflammation) or cytokine storm
8	MIS-C related myocarditis	• Transient systolic dysfunction on echocardiography • Diffuse myocardial edema on STIR-MRI without delayed enhancement

An upper abdominal ultrasound-based study on 37 COVID-19 positive patients showed gallbladder sludge in 60%, wall thickening in 3%, and pericholecystic fluid in 3% patients.[59] Another study showed gallbladder distension and wall edema in 25% of COVID-19 patients reported as possible or definite cholecystitis (Fig 13A-D).[57] SARS-CoV-2 binding to gallbladder epithelial cells leads to mucosal inflammation and hyperemia. Gallbladder wall edema is also a common finding in acute hepatitis and is an independent predictor of a more

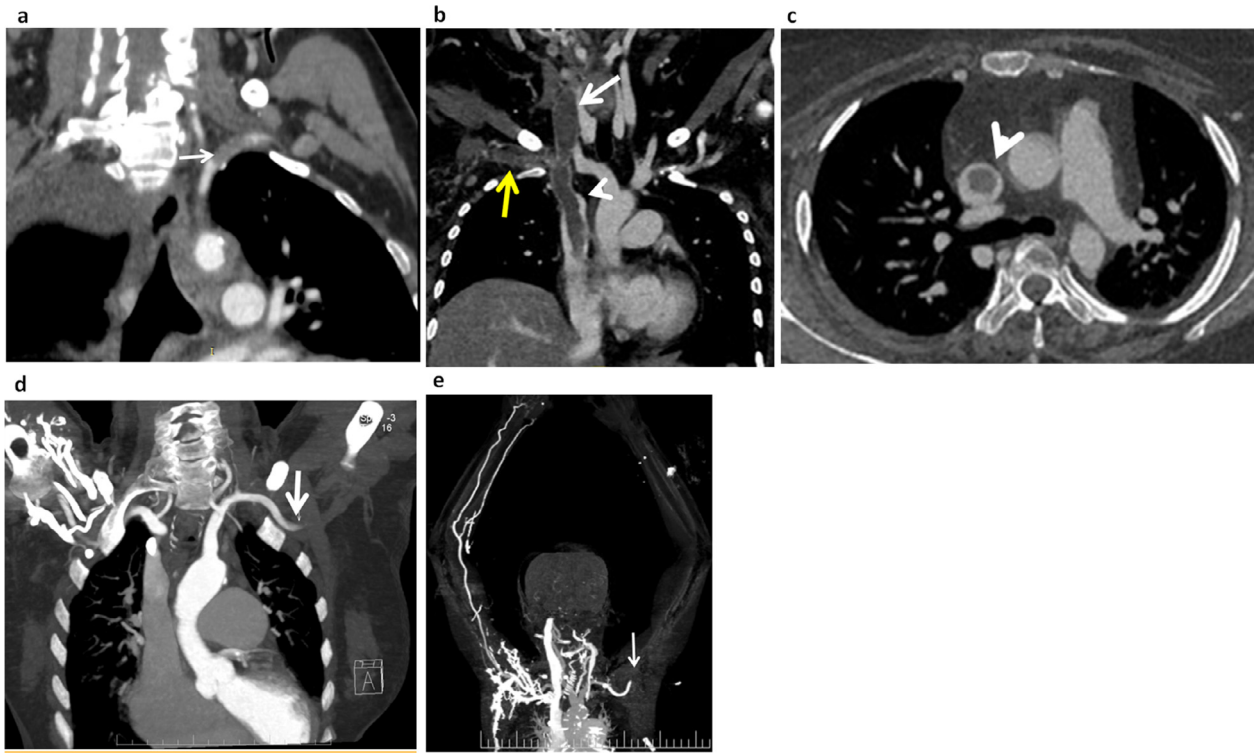

FIG 9. Thromboembolic complications in different COVID positive patients. (A) Subclavian artery thrombosis. Coronal reformatted CECT reveals a hypodense filling defect in the left subclavian artery (B, C) Superior vena cava (SVC) thrombosis. Coronal reformatted (B) and axial (C) CECT reveals large hypodense filling defect in SVC (white arrowhead B,C) extending to right internal jugular vein (white arrow b) and right subclavian vein (yellow arrow B). (D,E) Axillary artery thrombosis. Coronal maximum intensity projection (MIP) images of CT angiography revealed sudden cut-off of left axillary artery suggesting thrombotic occlusion (white arrow) (Color version of the figure is available online.).

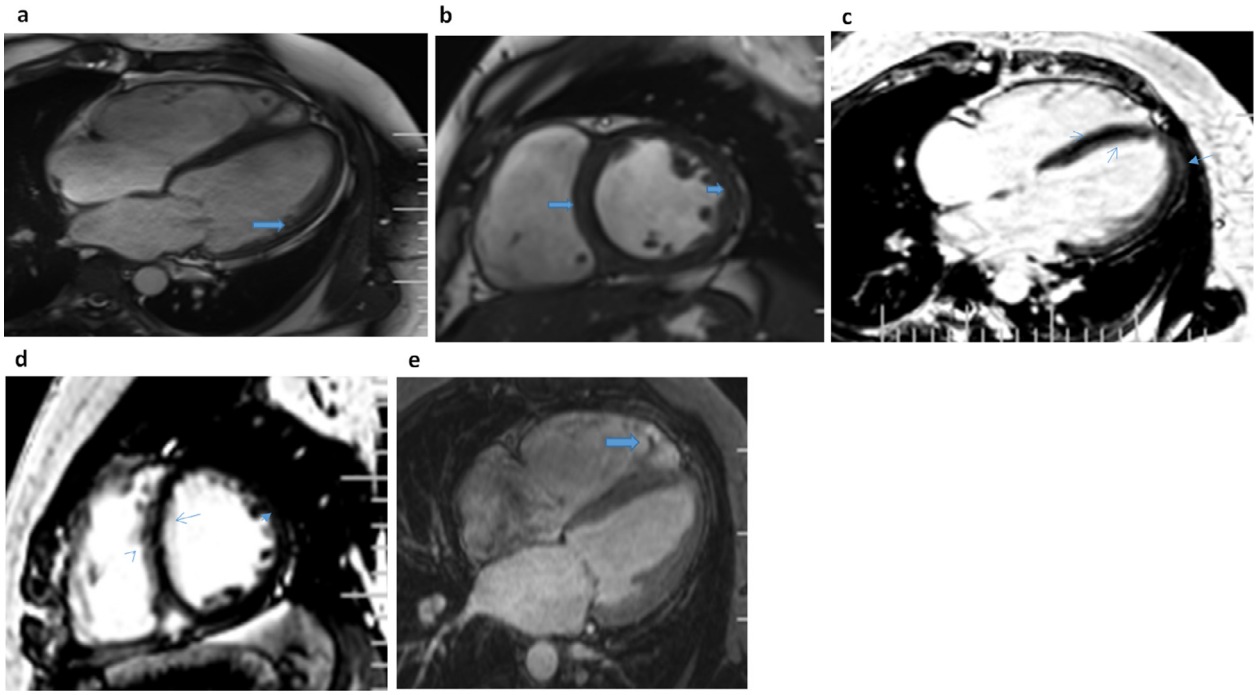

FIG 10. Post-COVID myocarditis. 34-year male, diagnosed with COVID-19 2 months' back. Persistent fatigue and breathlessness after recovery from COVID. RT-PCR negative. On 2D echo- RVand LV were dilated. Cardiac MRI was performed. (A,B):Fast spin echo(FSE) T2-W images (A) four chamber, (B)two chamber short axis view reveal dilated LV, RV and myocardial edema (arrows) (C,D) LGE (Late gadolinium enhancement) PSIR (phase sensitive inversion recovery) images, (C) four chamber, (D) two chamber short axis view: showing diffuse epimyocardial and midmyocardial linear enhancement predominantly along the septum and lateral wall (thin arrows in C& D). (E) Inversion recovery (IR) thrombus imaging: small thrombus in the RV apex (thick arrow). Global systolic dysfunction with LVEF of 43 % was also noted. (Color version of figure is available online).

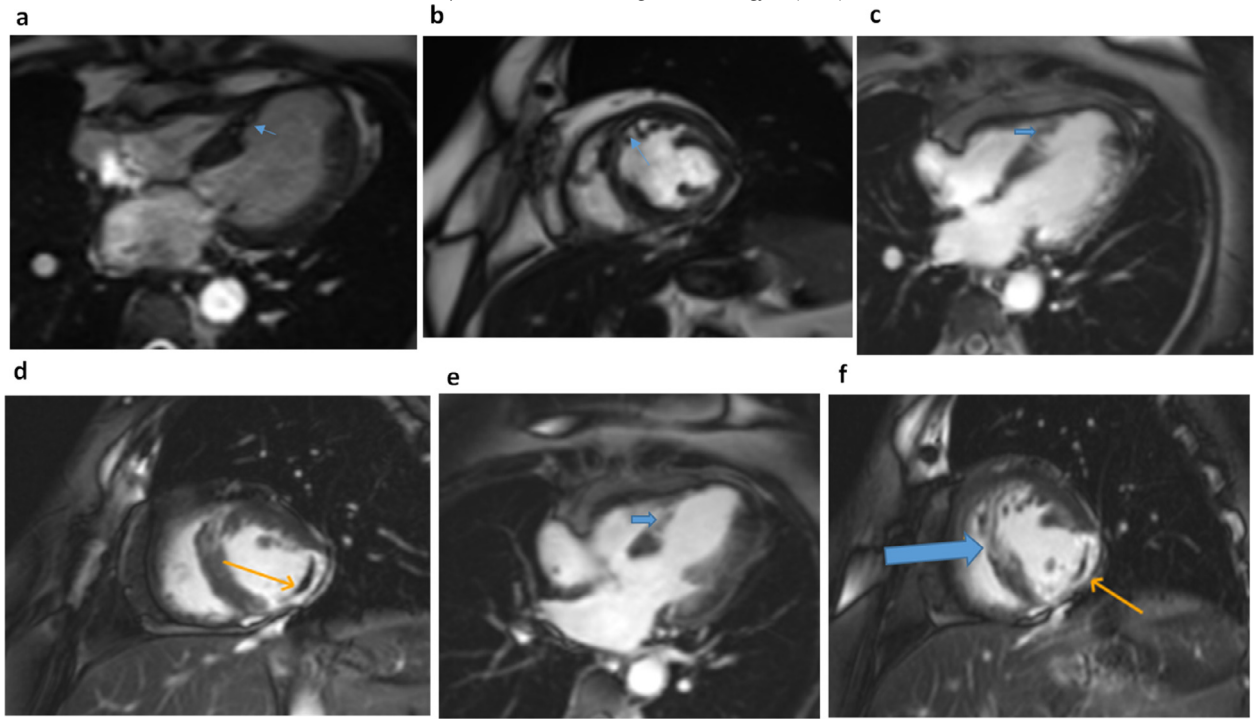

FIG 11. Acute coronary syndrome. 47 years female diagnosed with COVID-19 disease, acute coronary syndrome. (A,B) Fast spin echo(FSE) T2-W images (A) four chamber view, (B) two chamber short axis view reveals significant myocardial thinning of the mid and distal LV anteroseptal wall and LV apex with mild intramyocardial linear high signal intensity (short thin arrow A, B) . (C-F) LGE-PSIR images, (C,E) Four chamber long axis, & (D,F) 2 chamber short axis reveal marked subendocardial transmural late gadolinium enhancement involving more than 75% of myocardial thickness in mid and distal LV anteroseptal wall in the left anterior descending artery distribution and along the mid and distal LV inferolateral wall in left circumflex artery (LCX) distribution (thick arrow C,E,F). 'No reflow zone' in infero-lateral wall due to MVO(microvascular obstruction) in LCX distribution (long thin arrow in D, F). (Color version of figure is available online).

TABLE 4
Abdominal Involvement of COVID-19 infection.

	Complication and imaging manifestations	Proposed etiology
1	Small bowel thickening, solid organ infarctions and vascular thrombosis	SARS-CoV-2 binding to bowel epithelial cells leads to mucosal inflammation, prothrombotic state
2	Fluid-filled bowel loops identified on CT were consistent with the clinical diarrhea	Alterations in intestinal microbial flora and cellular regulation of fluid and electrolytes
3	Liver dysfunction and hepatocytes- USG altered echotexture, steatosis	Binding of virus to hepatic ACE-2 receptors or immune cytokine storm
4	Gallbladder sludge in 60%, wall thickening in 3% and pericholecystic fluid	Viral binding to ACE-2 or secondary to hepatocyte damage
5	Acute pancreatitis	Inflammation due to viral binding of ACE-2 receptor
6	Epiploic appendagitis	Inflammation and venous thrombosis

severe clinical course.[45] Gallbladder wall edema seen in COVID-19 may reflect hepatocellular damage, either directly by SARS-CoV-2 or by a reactive inflammatory response.

A study showed that 9 out of 72 patients presented with abdominal pain, tested positive for COVID-19, and showed findings similar to acute pancreatitis.[64] Liu *et al.*[65] reported high ACE-2 expression in pancreas, which lead to 2% and 17% evidence of pancreatic injury in non–severe and severe patients, respectively (Figure 13 E,F). The various abdominal findings of COVID-19 are summarized in Table 4.

Renal Involvement

There is significant co-expression of ACE-2 and Transmembrane protease, serine (TMPRSS) in cells of proximal convoluted tubules, which places them at high risk of injury due to SARS-CoV-2.[66] Cytokine storm may also have a role in renal injury. A review by Zaim *et al.* described that 0.5%-19% of COVID-19 patients may show varying degrees of renal injury. Possible renal manifestations of COVID-19 include acute kidney injury, electrolyte abnormalities, proteinuria, and hematuria.[67] Imaging-wise, this may be seen on ultrasound as increased renal cortical echogenicity and altered corticomedullary differentiation. This knowledge is relevant for radiologists as they must exercise caution while advising contrast-enhanced CT, which may be replaced by ultrasonography or non–contrast MRI.

Musculoskeletal Involvement

Myalgia, arthralgia and fatigue have been commonly reported symptoms in COVID-19.[68] During the SARS-CoV-1 epidemic, late complications including reduced bone mineral density (BMD), osteoporosis, and osteonecrosis were reported related to prolonged ICU stay, corticosteroid use, systemic inflammation, and prothrombotic state. Similar complications may be anticipated during the current SARS-CoV-2 pandemic.[69] There have been case reports of myositis and rhabdomyolysis related to COVID-19 effects. MRI findings of myositis include swelling, T2 hyperintensity, and enhancement

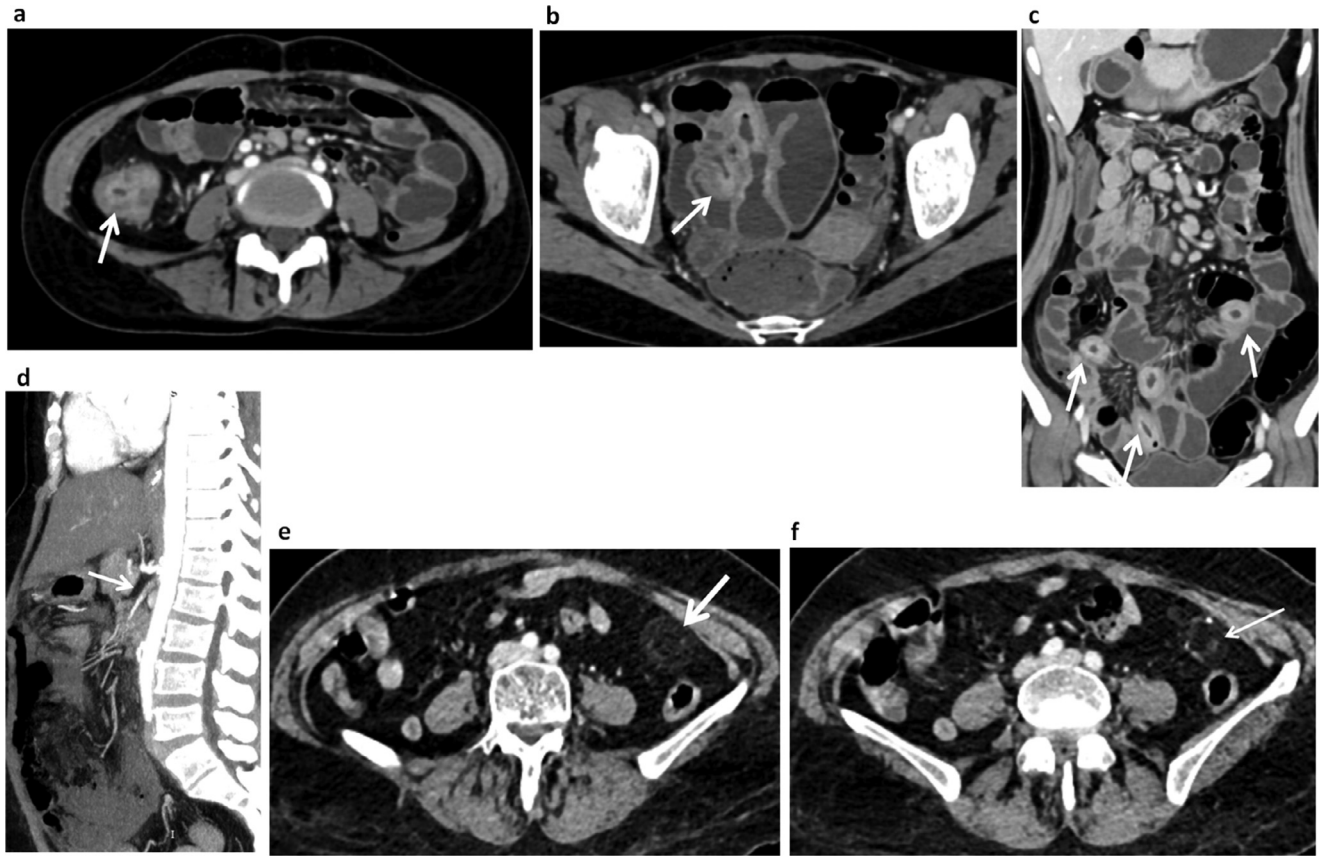

FIG 12. (A, B) Bowel thickening. Axial CECT abdomen reveals multifocal bowel wall thickening and hyperenhancement in ascending colon (arrow A) and distal ileum (arrow B). (C) Small bowel thickening. Coronal reformatted CECT in another patient reveals multifocal small bowel thickening and hyperenhancement (arrows) (D) Superior mesenteric artery (SMA) thrombosis. Sagittal maximum intensity projection (MIP) shows a hypodense filling defect in the SMA suggestive of thrombosis. (E,F) Epiploic appendigitis. 63 years female with abdominal pain. Axial CECT abdomen reveals fat density lesion with surrounding inflammation and central hyperdense dot in left hypochondrium suggestive of epiploic appendigitis. Lung bases revealed patchy ground-glass opacities (not shown), later diagnosed as COVID-19.

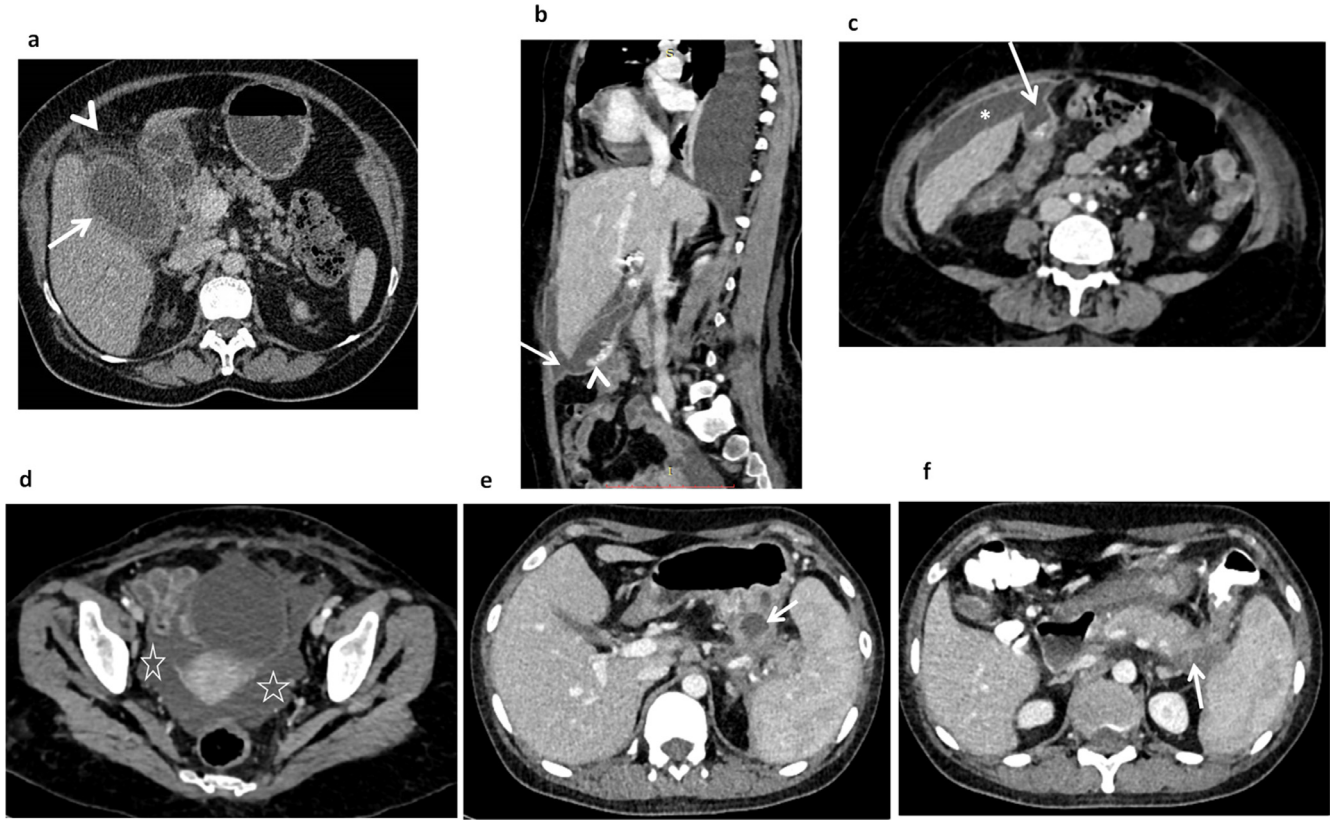

FIG 13. Gall bladder manifestations (A) Cholecystitis. Axial CECT abdomen reveals an overdistended gall bladder with circumferential wall thickening (arrow) and pericholecystic fat stranding (arrowhead), representing acute acalculous cholecystitis. (B,C,D) Cholelithiasis with gall bladder perforation. Sagittal reformatted (B) and axial (C) CECT images reveal cholelithiasis(arrowhead) with a focal perforation in gall bladder fundus (arrow) and adjacent perihepatic collection (*). Pleural effusion and moderate ascites (star in D) were also noted. (E,F) Acute Pancreatitis. 35 years male with pain abdomen. Axial CECT abdomen reveals bulky pancreas with peripancreatic fat stranding and free fluid. Small pseudocyst was also noted in lesser sac (arrow). He later tested positive for COVID-19.

within the muscles. Myonecrosis is identified by the stipple sign showing foci of enhancement in a rim enhancing area of non–enhancement.[70]

Post-infectious neuropathies have also been rarely reported in COVID-19, and their imaging findings include nerve enlargement, hypoechogenicity, loss of architecture, and T2 hyperintensity on ultrasound and MRI, respectively.[70,71] Soft tissue hematomas may develop in COVID-19 due to virus-induced bleeding complications or anticoagulation prophylaxis of thrombotic coagulopathy.[72] Arthritis in COVID-19 may be viral related or more commonly due to the triggering of chronic rheumatologic diseases such as Rheumatoid or Psoriatic arthritis. Viral induced arthritis shows non–specific findings of synovial enhancement and thickening on MRI, while inflammatory arthritis may reveal polyarticular arthritis and erosions.[70,73,74]

Conclusion

The novel Coronavirus brought the whole world to a standstill in 2020. Being a new disease, its clinical manifestations, radiological imaging, and treatment have rapidly evolved owing to ongoing research. It has been realized that the effects of COVID-19 are not limited to the lung and involve several organ systems in the body. The role of imaging is ancillary to the RTPCR test for diagnosis but indispensable for prognosticating and following up COVID-19 pneumonia. Imaging is also imperative in diagnosing thromboembolism, complications in brain, cardiovascular system, gastrointestinal, and hepatobiliary system.

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Declaration of Competing Interest

None.

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