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Case Series

Radiology Section

Imaging in Multisystem Complications of COVID-19 associated Coagulopathy: An Interesting Case Series

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ABSTRACT

The ongoing global threat of Coronavirus Disease 2019 (COVID-19) has been an issue of escalating concern due to its propensity to cause increased morbidity and mortality. Extensive reports have emphasised chest imaging findings in patients with Severe Acute Respiratory Syndrome Coronavirus-2 (SARS-CoV-2) infection leading to a greater understanding of pathogenesis in the lung. There is also an added lurking pernicious propensity of the virus to cause a distinct coagulopathy attributed to the increased virulence. Further, radiologists should be aware of thromboembolic imaging findings in patients with SARS-CoV-2 infection. In this case series, the authors present six cases of SARS-CoV-2 infection confirmed on nasopharyngeal Reverse Transcription Polymerase Chain Reaction (RT-PCR) tests, presenting with various systemic thromboembolic complications. The purpose of this case series is to explore diverse imaging findings in patients with COVID-19 coagulopathy.

Keywords: Coronavirus disease, Ischaemia, Prognosis, Thromboembolism, Thrombosis

INTRODUCTION

Coronavirus disease 2019 (COVID-19), first identified in Wuhan, China in December 2019, has become a worldwide pandemic causing extensive ailment and mortality causing an overwhelming impact on the global economy [1]. Although respiratory compromise is the fundamental trait of the disease, several extrapulmonary complications are identified, causing increased morbidity and mortality [2].

Thromboembolic complications of SARS-CoV-2 infection are a crucial issue and have a reported occurrence of about 31% in patients admitted in the Intensive Care Unit (ICU) [3].

In this case series, authors present six cases of SARS-CoV-2 infection confirmed on nasopharyngeal Reverse Transcription Polymerase Chain Reaction (RT-PCR) tests presenting with diverse systemic thromboembolic complications.

CASE SERIES

Case 1

A 65-year-old male patient on admission with a positive result for SARS-CoV-2 infection on the nasopharyngeal swab (RT-PCR) complained of right upper abdominal pain one week postadmission. He was afebrile with otherwise stable vital signs. On physical examination, the patient was anxious, breathing comfortably on nasal cannula and an abdominal examination showed diffuse tenderness on palpation, worst in the right upper quadrant. There was no abdominal distention, rigidity or guarding.

Laboratory work-up showed two-fold elevation in D-dimer and mild elevation of C-Reactive Protein (CRP) and Serum Ferritin (SF). Laboratory parameters of the patient on admission are mentioned in [Table/Fig-1].

Non Enhanced Computed Tomography (NECT) and Contrast Enhanced Computed Tomography (CECT) of the abdomen in arterial phase [Table/Fig-2a,b] showed a well-defined non enhancing, heterogeneous soft tissue attenuation mass-like lesion measuring 5.8×3.1×3.1 cm [AP- anteroposterior; TR-transverse; CC-craniocaudal (CC×TR×AP)] in the subhepatic region anteromedial to the ascending colon. The lesion showed a hypodense fat halo with adjacent fat stranding. A mildly hyperdense non enhancing serpiginous structure, suggestive of thrombosed vessel was noted coursing towards the lesion. Bowel loops adjacent to the above mentioned lesion showed no significant abnormality.

Parameters	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6
D-dimer (ng/mL FEU)	960	424**	2164	1255	1554	2254
Normal value <500 ng/mL FEU						
C-reactive protein (mg/L)	9.1	7.2	60.5	14.2	20.1	70.2
Normal value <5 mg/L						
Prothrombin time (sec)	13.0	12.5	17.0	15.5	16.0	18.5
Normal range 11-13.5 sec						
Ferritin (ng/mL)	455	154	668	885	455	743
Normal range- • Females-10-200 ng/mL • Males- 30-300 ng/mL						

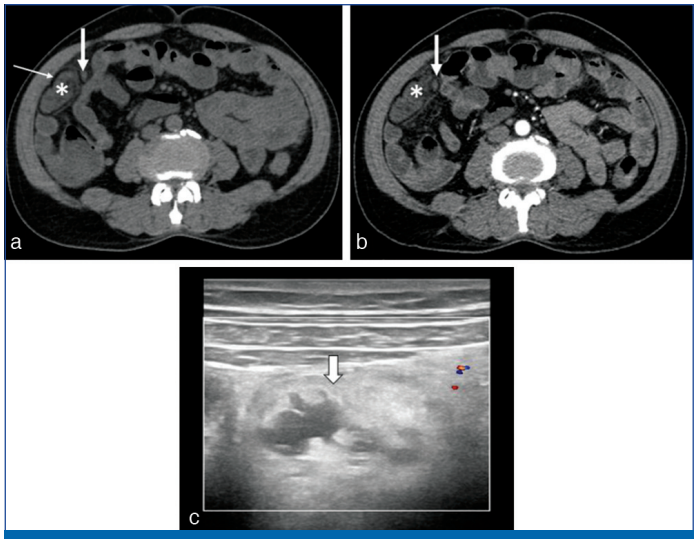
[Table/Fig-1]: Laboratory parameters of the patients on admission.
FEU: Fibrinogen equivalent units
**The patient presented with respiratory complaints and on admission her D-Dimer was 424 ng/mL FEU. On follow-up D-Dimer study done 48 hours later, which showed an elevated value of 752 ng/mL FEU. However, since the table was made based on admission values of laboratory parameters, the initial value has been mentioned

On colour Doppler ultrasound [Table/Fig-2c], the lesion appeared heteroechoic with a peripheral echogenic fat halo and showed no evidence of internal vascularity. The features on imaging were suggestive of intraperitoneal focal fat infarction, possibly omental infarction.

The patient was primarily admitted for the management of his COVID-19 pneumonia and had completed a 5 day course of remdesivir and was receiving Low Molecular Weight Heparin (LMWH) and dexamethasone. He was started on piperacillin-tazobactam injection for a week and completed the course. The patient was eventually discharged after three weeks of admission following clinical improvement.

Case 2

A 48-year-old female patient with a positive result for SARS-CoV-2 infection on the nasopharyngeal swab (RT-PCR) was admitted

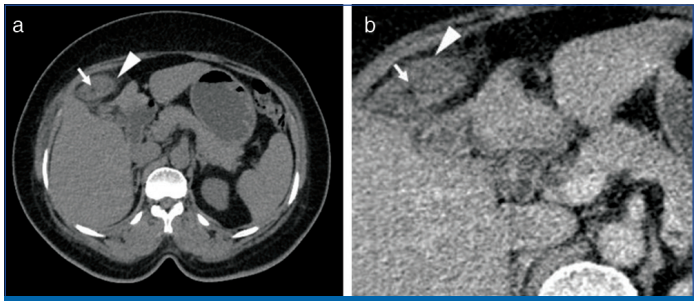


[Table/Fig-2]: a) Non enhanced; and b) CECT of the abdomen in arterial phase demonstrated a well-defined non enhancing, heterogeneous soft-tissue attenuation mass-like lesion (*) with fat halo (thin arrow) in the subhepatic region along with a hyperdense non enhancing serpiginous structure coursing towards the lesion (thick arrow) (suggestive of a thrombosed vessel). Ultrasound colour Doppler study; c) of the lesion showed a heterogeneous lesion (block arrow) with a peripheral hyperechoic fat halo without evidence of internal vascularity.

for respiratory complaints and was discharged after symptomatic improvement after seven days. She was advised to continue tablet rivaroxaban 10 mg for a week. She had no existing co-morbidities and presented to the Emergency Room (ER) 10 days postdischarge with complaints of fever and acute abdominal pain.

On abdominal examination, tenderness was noted in the right upper quadrant with no abdominal rigidity or guarding. Routine laboratory work-up was unremarkable, except for mild elevation of CRP. Laboratory parameters of the patient on admission are mentioned in [Table/Fig-1].

NECT and CECT of the abdomen in portovenous phase [Table/Fig-3a,b] showed an intraperitoneal, non enhancing, heterogeneous soft tissue density lesion with few small central hypodense areas of fat attenuation measuring 3.4×3.2×1.9 cm (CC×TR×AP). The lesion was seen anteroinferior to the liver and closely abutting the anterior abdominal wall with adjacent fat stranding. The features on imaging were suggestive of intraperitoneal focal fat infarction i.e., epiploic appendagitis.



[Table/Fig-3]: a) Non-enhanced; and b) Contrast-enhanced CT of the abdomen in portal-venous phase demonstrated an intraperitoneal non-enhancing soft-tissue attenuation lesion (arrowhead) with few small central hypodense areas of fat attenuation (arrow) anterior to the liver.

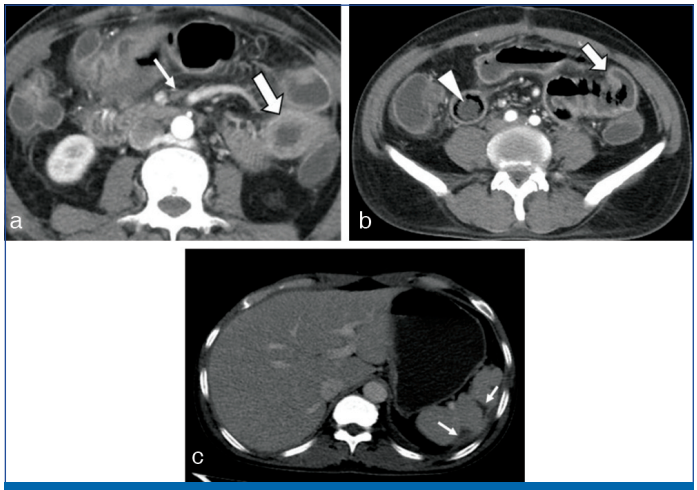
The patient was admitted to the inpatient department and was started on intravenous antibiotics and analgesics along with oral rivaroxaban. The abdominal symptoms improved on day 7 of admission and she remained in good health after completing her treatment course. She was discharged on day 10 after admission.

Case 3

A 45-year-old male patient on admission for respiratory complaints with a positive result for SARS-CoV-2 infection on the nasopharyngeal swab (RT-PCR) presented with complaint of acute onset of abdominal pain with abdominal guarding and rigidity 6 days postadmission. The patient had co-existing diabetes and dyslipidemia diagnosed on admission.

Laboratory work-up showed significant elevation of D-Dimer by four-fold along with elevated CRP, Prothrombin Time (PT), and SF. Laboratory parameters of the patient are mentioned in [Table/Fig-1].

A CECT of the abdomen [Table/Fig-4a-c] was suggested due to the abdominal guarding and rigidity. The findings of arterial phase [Table/Fig-4a,b] showed a hypodense intraluminal filling defect (thrombus) with non contrast opacification of the Superior Mesenteric Artery (SMA) just distal to the branching of ileocolic-right colic trunk till its termination. The distal jejunal loops and proximal ileal loops were dilated with mucosal hyperenhancement and showed small bowel faeces sign with no obvious transition point.



[Table/Fig-4]: (a, b) shows axial CECT of the abdomen- an intraluminal filling defect (thrombus) with non contrast opacification of the Superior Mesenteric Artery (SMA) (thin arrow), mucosal hyperenhancement (block arrow) involving small bowel loops, and small bowel faeces sign (arrowhead) (c) shows non enhancing hypodense splenic infarcts (arrows).

CECT of the abdomen in the portovenous phase [Table/Fig-4c] showed multiple wedge-shaped non enhancing hypodense areas within the splenic parenchyma possibly multiple splenic infarcts.

After initial resuscitation, the patient was taken up for emergency laparotomy and resection of 15 cm gangrenous distal jejunal loops, followed by anastomosis. The patient was shifted to the intensive postoperative care unit and succumbed to sepsis after 5 days.

Case 4

An otherwise healthy 41-year-old male, recently discharged from an outside hospital following a short and uneventful admission for COVID-19 a week back, presented to our ER with confusion, right hemiparesis, and dysarthria.

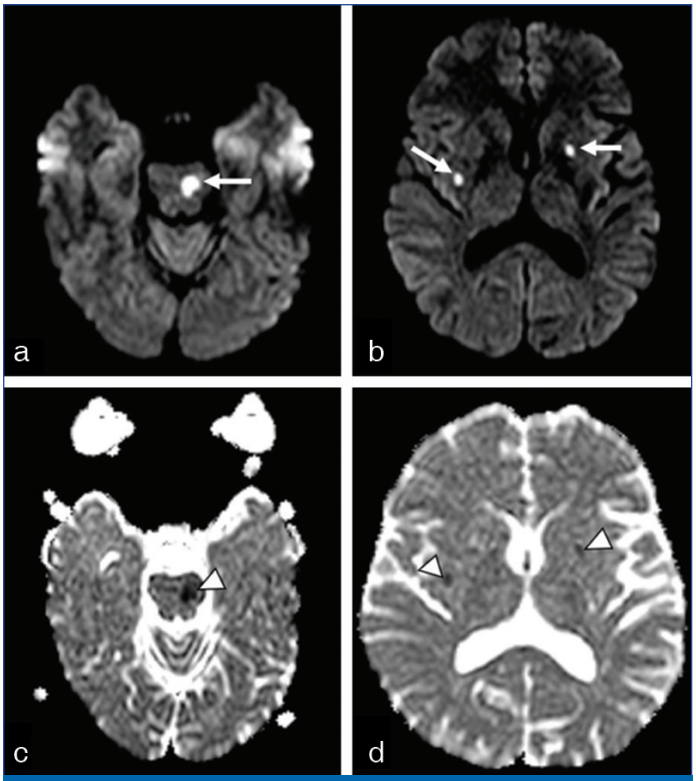
Laboratory work-up showed a three-fold elevation of D-Dimer along with elevated CRP, PT, SF. Laboratory parameters of the patient are mentioned in [Table/Fig-1].

Magnetic Resonance Imaging (MRI) study of the brain [Table/Fig-5a-d] revealed multifocal areas of restricted diffusion on Diffusion-Weighted Imaging (DWI) involving left hemipons and bilateral lentiform nuclei demonstrative of multifocal acute infarcts. The patient was treated with LMWH, clopidogrel along with dexamethasone after which he improved symptomatically and was discharged after 1 week.

Case 5

A 33-year-old male with no known medical history presented to the ER with vomiting and diarrhoea for 7 days, headache for the past 4 days, and altered mental status for the past 2 days with sudden onset of seizures.

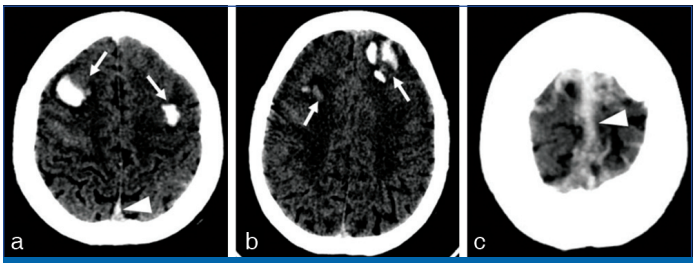
On examination, he exhibited signs of dehydration but there was no pyrexia or associated co-morbidities. As a part of routine



[Table/Fig-5]: (a and b) shows DWI of brain demonstrating restricted diffusion involving the left hemipons and bilateral lentiform nuclei (arrows). (c and d) shows the corresponding Apparent Diffusion Coefficient (ADC) mapping low signals (arrowheads).

admission screening on a visit to ER, the patient had a rapid SARS-CoV-2 nasopharyngeal swab test done which showed positive result. Laboratory parameters of the patient were as mentioned in [Table/Fig-1].

A NECT study of the head on admission [Table/Fig-6a,b] showed multiple intraparenchymal haemorrhage, involving the cortical and subcortical regions of the bilateral frontal lobes with adjacent oedema. NECT study of the head [Table/Fig-6c] also showed hyperdensity within the superior sagittal sinus and in cortical veins adjacent to the superior sagittal sinus, draining bilateral high frontoparietal lobes consistent with Cerebral Venous Thrombosis (CVT).



[Table/Fig-6]: Non contrast axial CT scan images of the brain [a, b, c] showed multifocal haemorrhagic foci with surrounding oedema involving the subcortical white matter of bilateral frontal lobes (arrows) and hyperdensity within the superior sagittal sinus corresponding to the thrombus (arrowheads).

The patient was started on anticoagulant therapy with intravenous LMWH along with antiepileptics and osmotic diuretics. During the following days, the patient's neurological state gradually worsened and he developed acute respiratory failure with pneumonia. He was transferred to ICU and was intubated on ventilator support. He was also started on antibiotic treatment with intravenous meropenem and dexamethasone along with anticoagulants and antiepileptics. The patient succumbed on day 14 after admission due to cardiorespiratory failure.

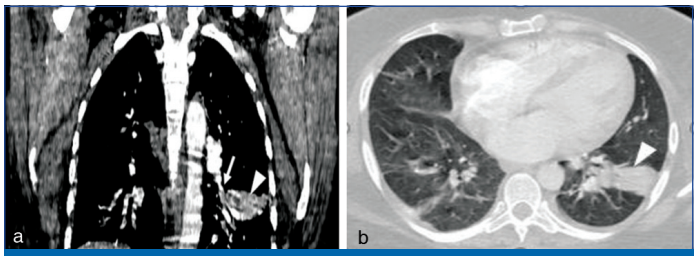
Case 6

A 29-year-old female tested positive for SARS-CoV-2 infection on the nasopharyngeal swab (RT-PCR) and was with respiratory support on admission. The patient had no existing co-morbidities and developed sudden excruciating chest pain and desaturation on day 2 of admission.

Laboratory work-up showed significant elevation of D-Dimer by four-fold along with elevated CRP, PT and SF. Laboratory parameters of the patient are mentioned in [Table/Fig-1].

CT pulmonary angiogram [Table/Fig-7a] in mediastinal window showed partial luminal filling defects in subsegmental arteries supplying basal segments of left lower lobe.

Axial CT thorax in lung window [Table/Fig-7b] demonstrated wedge-shaped infarct in the lateral basal segment of left lower lobe along with few linear atelectatic bands in the posterior basal segment of the right lower lobe. Upper and lower extremity Doppler ultrasounds were obtained but showed no evidence of deep venous thrombosis.



[Table/Fig-7]: a) Coronal reformatted and b) Axial pulmonary CT angiography images demonstrated thrombus as a filling defect in the left lower lobar segmental pulmonary artery (arrow) and a wedge-shaped pulmonary infarct (arrowhead).

The patient was on a 5 day course of remdesivir, along with dexamethasone and anticoagulant therapy with intravenous LMWH. She was extubated 3 days later. Following extubation, she showed improvement and was discharged home on oral rivaroxaban for 3 months.

DISCUSSION

SARS-CoV-2 infection is characterised by an exaggerated pro-inflammatory state, leading to the development of abnormal haemostasis [4]. These events precede the occurrence of localised and systemic coagulopathies and this consequential evolution has been termed COVID-19 associated coagulopathy which is characterised by elevated levels of D-dimer and fibrinogen, with minor abnormalities in PT and platelet counts [5].

In a study by Tang N et al., abnormal coagulation parameters were seen to be associated with 71.4% of non survivors in comparison to 0.6% survivors with SARS-CoV-2 infection, substantiating the increased mortality due to abnormal coagulopathy during the course of disease progression [6].

From various studies, it is now evident that a multitude of coagulopathy manifestations of SARS-CoV-2 infection in several systems are being increasingly identified and reported [7-9]. The purpose of our case series was to present the imaging characteristics of varied manifestations of COVID-19 associated coagulopathy with diverse clinical entities.

Intraperitoneal Focal Fat Infarction (IFFI): IFFI is a term encompassing an array of conditions with the common underlying pathology being infarction and necrosis of intra-abdominal fatty tissue [10].

Epiploic appendagitis and omental infarction are two distinct types of fat necrosis that commonly mimic acute abdomen and accurate identification of these conditions is crucial to avoid unnecessary surgical intervention [11]. Ahmed AO et al., reported the first case of omental infarction in a COVID-19 patient who presented with acute abdominal pain and the patient was managed conservatively without the necessity of any surgical intervention [12]. Few other case reports of epiploic appendagitis presenting in COVID-19 patients have been identified [13,14]. Primary omental infarcts are related to reduced end-organ perfusion as in hypercoagulable

states, congestive heart failure, and vasculitis, whereas secondary infarcts may be related to traumatic injury. Omental infarction on CT appears as an encapsulated fatty mass usually larger than 5 cm with soft-tissue stranding adjacent to the ascending colon [15].

Typical CT features of epiploic appendagitis include a small, oval area of fat attenuation with a ring of soft tissue and adjacent inflammation located anterior or anterolateral to the adjacent colon along with a central dot often representing a thrombosed vein [16].

Ultrasound with Doppler usually demonstrates an ovoid area of avascular echogenic fat in both conditions with a marginal hypoechoic ring in epiploic appendagitis [17].

Acute mesenteric arterial ischaemia: Diverse gastrointestinal manifestations due to cytopathic, ischaemic, thromboembolic, motility disorders have been seen in association with COVID-19 infection [18].

Angiotensin Converting Enzyme-2 (ACE-2) is most abundant in the lung alveolar epithelium, enterocytes of the small intestine and vascular endothelium, suggesting that the small bowel and vasculature may also be increasingly susceptible to SARS-CoV-2 infection [19]. The sources of bowel ischaemia in patients with COVID-19 include direct viral infection, small-vessel thrombosis, or non occlusive mesenteric ischaemia [20].

Acute arterial thrombus appears as low attenuation filling defects in the SMA or other major mesenteric arteries. Ancillary clues of altered bowel wall enhancement further increase the diagnostic accuracy [21]. Abnormal intestinal mural enhancement in mesenteric ischaemia contrasts from diminished enhancement in the early phase to hyperenhancement of the bowel in the reperfusion stage. The bowel wall thickness in bowel infarction varies from paper-thin wall in transmural infarction to mild-moderate thickened wall due to intramural haemorrhage/haematoma [22]. The concurrence of pneumatosis intestinalis along with porto-mesenteric gas increases the diagnostic accuracy of ischaemic bowel. Since mesenteric ischaemia can clinically mimic a multitude of causes manifesting as acute abdomen, cross-sectional imaging plays a critical role in the prompt diagnosis and management of patients with bowel ischaemia [23].

Cerebrovascular diseases- Acute ischaemic stroke and Cerebral Venous sinus Thrombosis (CVT): Acute ischaemic stroke, intracerebral haemorrhage, and CVT have been identified in case reports and cohorts of COVID-19 patients with a prevalence ranging between 0.5% and 5% [24]. The primary cohort study about neurological manifestations of COVID-19 in 214 patients in China showed an incidence of 2.8% acute cerebrovascular diseases in patients [25]. Another retrospective study of 277 patients with acute stroke showed COVID-19 positive patients were more likely to have a cryptogenic stroke (51.8% versus 22.3%) compared with COVID-19 negative patients [26]. Cerebrovascular disease seems to be as much as two-fold common in patients with severe COVID-19 infection presenting with neurological complaints [27].

Hughes C et al., published the first case of CVT in a COVID-19 patient in April 2020 [28]. Multiple other case studies also substantiate the evidence of an underlying prothrombotic state in SARS-CoV-2 infection to be a postulated risk factor for CVT [29-31]. In an acute ischaemic stroke, the role of unenhanced CT is indispensable, having the advantages of accessibility and rapidity and it facilitates not just in identifying haemorrhage but can also help detect early stage acute ischaemia by showing features such as the hyperdense vessel sign, the insular ribbon sign, and obscuration of the lentiform nucleus [32].

MRI further aids to perceive the relative age of a cerebral infarct and classify them as early hyperacute, late hyperacute, acute, subacute, or chronic with the findings seen at ADC and DWI, Fluid-Attenuated Inversion Recovery (FLAIR), and unenhanced T1- and T2-weighted, susceptibility-weighted MRI sequences [33]. Further, CT and MRI perfusion imaging can also identify possible areas of salvageable brain tissue in the ischaemic penumbra [32].

Cerebral Venous Thrombosis (CVT) is one of the rare causes of cerebral haemorrhage that warrants the initiation of anticoagulation therapy as anticoagulation is contraindicated in most cases of cerebral bleeding and this, in turn, makes the diagnostic task challenging.

As NECT of the head is usually the first-line imaging in patients who present at the emergency department with neurologic symptoms. The direct visualisation of CVT as hyperattenuation in the occluded sinus or vessel (cord sign) is present in only 25-56% of cases [34]. CECT venography may demonstrate an empty delta sign where a triangular area of contrast enhancement surrounds a hypoattenuating thrombus [35].

Non enhanced MRI is more sensitive than non enhanced CT for detecting CVT in the venous sinuses where the loss of flow void/altered signal intensity cautions the possibility of cerebral venous thrombosis [36]. Time of flight Magnetic Resonance venography (TOF MR) is the method most commonly used for the diagnosis of CVT [36].

Pulmonary thromboembolism: Pulmonary embolism is documented as a frequent and fatal complication in severe COVID-19 patients and was seen with a reported frequency of 16.5% to as high as 30% in severe cases admitted to the Intensive Care Unit (ICU) [37-39]. D-Dimer blood levels showed a significant correlation both with the incidence and distribution with the levels being significantly increased in cases of main pulmonary arterial involvement and with an increasing number of lobes involved [40].

Computed Tomographic Pulmonary Angiography (CTPA) is a crucial requisite in patients with suspected pulmonary embolism. Pulmonary thromboembolism causes partial and complete intraluminal filling defects having a sharp interface with intravascular contrast material with the increased caliber of the vessel in acute conditions [41]. Peripheral wedge-shaped areas of increased attenuation may represent infarcts and along with linear atelectatic bands are significant supplementary findings associated with acute pulmonary embolism.

Other auxiliary features of right ventricular failure identified as right ventricular dilatation, deviation of the interventricular septum to the left side may also increase the diagnostic accuracy [42]. Considering the increasing mortality with COVID-19 coagulopathy, it is imperative to consider the usage of antithrombotic agents to alleviate the thrombotic embolic events in high-risk patients with COVID-19 [43,44].

CONCLUSION(S)

Radiologists must recognise and acquaint themselves with diverse complications of COVID-19 associated coagulopathy to aid in accurate diagnosis and prompt clinical management. The increasingly identified manifestations in COVID-19 associated coagulopathy also warrant the rationale of initiation of early anticoagulation in patients with abnormal coagulation parameters.

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