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CHEST

Pulmonary Vein Thrombosis in COVID-19

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Research on COVID-19, the cause of a rapidly worsening pandemic, has led to the observation of laboratory derangements such as a propensity towards a hypercoagulable state. However, there are currently no reports on the incidence of pulmonary venous thrombosis in the setting of COVID-19. We report a case in which follow-up chest CT scans revealed an expansile filling defect in a branch of the right inferior pulmonary vein, which is consistent with pulmonary venous thrombosis. Our objective was to provide insight into an uncommon sequela of COVID-19 and consequently garner increased clinical suspicion for pulmonary VTE during hospitalization.

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Since September 2020, numerous COVID-19 cases have emerged with >43 million confirmed cases and 1,157,509 deaths worldwide.[1] Health care teams have generated substantial literature on clinical manifestations,[2] disease course, and notably, the radiologic and laboratory abnormalities commonly found in this patient population,[3] particularly the hypercoagulable state of patients with COVID-19.

Here, we present a case of pulmonary vein thrombosis (PVT) in the setting COVID-19 infection, which to our knowledge has not been reported. We hope to demonstrate the clinical and radiographic findings of PVT in a hospitalized patient with COVID-19 and invite further discourse on PVT in the setting of COVID-19.

Case Report

A 35-year-old man presented to the ED complaining of worsening nonproductive cough, fever, chills, myalgias, anosmia, and pleuritic chest pain. Symptom onset was 11 days before, after returning from a trip to New York, which was a COVID-endemic region at the time. Several close-contacts also presented with similar symptoms. Two days before, he visited an outpatient clinic where he was started on antibiotic treatment for community-acquired pneumonia and received COVID-19 testing, the result of which was pending. Medical history included obesity and hypertension treated with hydrochlorothiazide and losartan.

In the ED, his physical examination was generally benign. Vital signs were significant for tachycardia to 113 beats/min and tachypnea; however, he maintained adequate oxygen saturation on room air. ECG showed sinus tachycardia with a rate of 115 with nonspecific ST segment changes. Chest radiography showed bibasilar peripheral patchy opacities and obscuration of both costophrenic angles. Laboratory values were significant for lymphopenia (lymphocyte percent of 10), despite normal WBC count, elevated creatinine level, and transaminitis. He was given IV ceftriaxone and azithromycin.

He was admitted to the hospital floor, with COVID-polymerase chain reaction testing, the result of which was positive the following day. Due to an elevated D-dimer value and his presenting symptom of chest pain, anticoagulation therapy with heparin was initiated.

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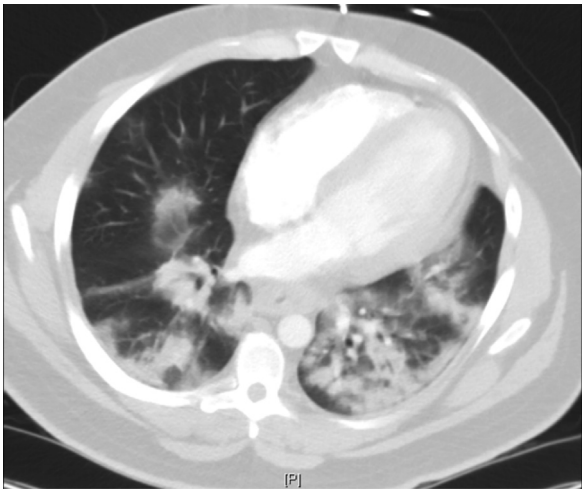

Figure 1 – *Axial CT scan of the chest shows features that are consistent with COVID-19: scattered peribronchovascular and peripheral mid to lower lung predominant ground-glass opacities and consolidations.*

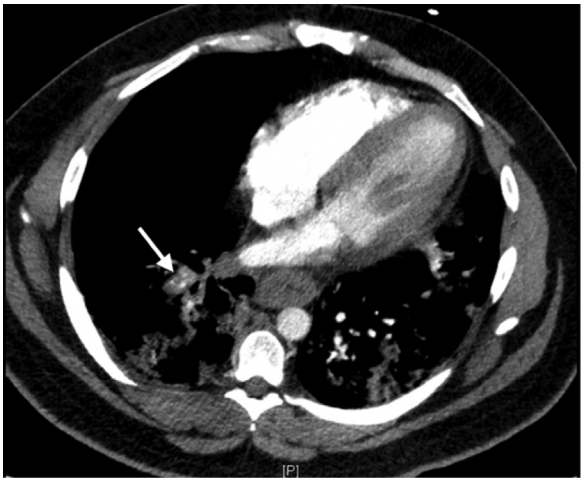

Figure 2 – *Axial contrast enhanced CT images of the chest: nonocclusive filling defect within the lateral basilar segmental pulmonary artery (white arrow), which is consistent with pulmonary arterial thrombo-embolic disease.*

Lower extremity ultrasound studies were negative for deep VTE.

His respiratory status remained stable on room air; however, he was persistently tachycardic, which prompted a chest CT scan with pulmonary embolism protocol on hospital day 4. Imaging revealed bilateral mid to lower lung peripheral predominant ground-glass and consolidative opacities and acute pulmonary thromboembolic arterial disease in segmental and subsegmental branches of the right lower lobe pulmonary arteries (Figs 1 and 2). Additionally, an expansile filling defect in a branch of the right inferior pulmonary vein was identified, which is consistent with acute PVT (Fig 3). The patient's condition remained stable, and he was discharged home 2 days later on oral anticoagulation.

Discussion

PVT is a rare and potentially lethal phenomenon. Its incidence is unknown because most literature consists of case reports, yet it remains underdiagnosed.[4] PVT most often arises after lung transplantations and lobectomies or in the setting of malignancy.[5,6] Most patients are asymptomatic; however, nonspecific symptoms (such as dyspnea, cough, and hemoptysis that are the result of pulmonary edema or infarction) can manifest. Complications include pulmonary infarction, pulmonary edema, right ventricular failure, and, less commonly, arterial embolism in the form of stroke and limb ischemia.

The diagnosis of PVT is made primarily through imaging because clinical signs are not specific. The preferred modalities include transesophageal echocardiography, CT scanning, MRI, and pulmonary angiography.[5,7] The decision to use a particular modality over another depends on the clinical context including suspected cause and clinical condition of the patient.[5,8]

CT offers several advantages for diagnosing PVT. The technique of pulmonary venous phase contrast CT scanning, which is the the standard for diagnosing pulmonary embolism, has been shown to improve the diagnosis of PVT over other CT techniques. A longer delayed phase can help to reduce artifacts from heart motion or dense contrast medium.[5,9] A case report has demonstrated the use of 64-multidetector CT scan to detect thrombi in the proximal left lower pulmonary vein, which is an area that commonly is obscured by artifact and pulmonary air during evaluation with transthoracic or transesophageal echocardiography alone.[10]

PVT management is based on the cause and severity of presentation and can include antibiotics, anticoagulation, thrombectomy, and/or pulmonary resection.[5] Unfortunately, there is no expert consensus on optimal management due to a lack of high-quality data. Systemic anticoagulation is recommended in all cases of PVT to decrease the risk of embolization, unless contraindicated.[4,9,11] However, there is no consensus on the preferred duration or type of anticoagulation.[5] Thrombectomy or lobe resection can be performed for larger and more obstructive thrombi.[12]

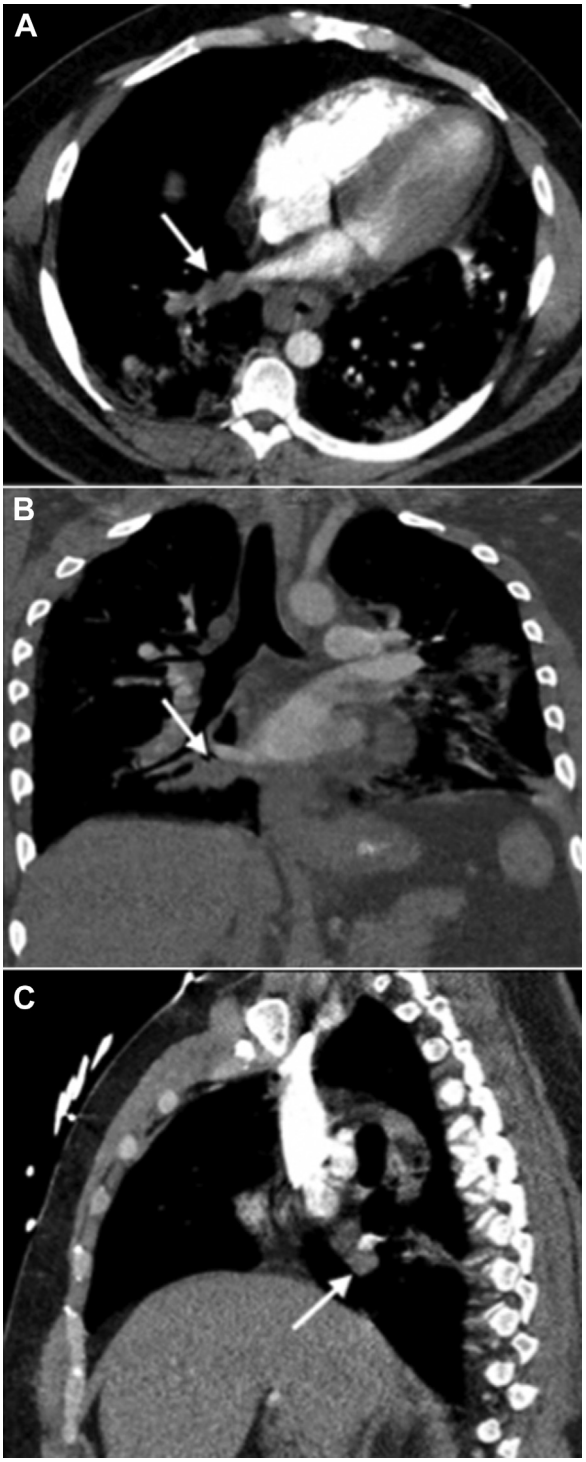

Figure 3 – *A, Axial, B, coronal, and C, sagittal contrast enhanced CT images of the chest: filling defect with focal distention of a branch of the right inferior pulmonary vein (white arrows), consistent with occlusive pulmonary venous thrombosis.*

Substantial evidence demonstrates positive association between COVID-19 and a hypercoagulable state. VTE, including DVT and pulmonary embolism, is seen at higher rates in patients with COVID-19 compared with patients who do not have COVID-19, often despite prophylactic-anticoagulation.[13-15] Arterial thromboembolism, including stroke, myocardial infarction, and acute limb ischemia, has also been positively associated with COVID-19.[16,17]

To our knowledge, this is the first documented case of PVT in the setting of COVID-19, corroborating the reported hypercoagulability in COVID-19. Increased clinical suspicion for PVT in the setting of COVID-19 is warranted, possibly lowering the threshold for diagnostic and therapeutic measures. The incidence of PVT may be increased among patients with COVID-19 compared with patients who do not have it. This case may also suggest a need for more aggressive measures for prophylaxis and treatment of PVT and other thromboembolic events in patients with COVID-19. These questions necessitate further study. Nevertheless, clinicians should be aware of the potential for PVT in COVID-19 and use their best clinical judgment to guide management decisions.

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