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A RARE CASE OF COVID-19-ASSOCIATED TRIAD OF ACUTE ISCHEMIC STROKE, MYOCARDIAL INFARCTION AND PULMONARY EMBOLISM

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ABSTRACT
Patients with coronavirus infection can develop mild symptoms, as well as severe disease, including acute respiratory distress syndrome, vital-organ compromise, and complications due to arterial thrombosis and venous thromboembolism. We delineate the case of a 63-year-old man who presented with deterioration of COVID-19 pneumonia. COVID-19 pneumonia in our patient was complicated by right-sided acute ischemic stroke, myocardial infarction, and pulmonary embolism, eventually leading to death. Arterial thrombosis and venous thromboembolism can increase the morbidity and mortality in coronavirus disease, rendering its early diagnosis and treatment crucial for the better management and prognosis of the infection.

KEYWORDS: Coronavirus, COVID-19, pneumonia, acute respiratory distress syndrome, thromboembolism.

INTRODUCTION
Coronavirus Disease (COVID-19) outbreak emerged in Wuhan, China, in late December 2019. The disease has taken a huge toll on public health worldwide and the organism responsible is severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2). COVID-19, primarily a respiratory disease, can show a wide array of clinical presentations.[1] Acute respiratory distress syndrome (ARDS) and compromise of vital organs profoundly increase the morbidity and mortality in SARS-CoV-2 infection, but complications due to arterial and venous thromboses (VTE) such as myocardial infarction (MI), acute ischemic stroke (AIS), and pulmonary embolism (PE) can also occur. These pathologies can cause severe disability and mortality in COVID-19 patients.[2,3] The incidence of VTE is highest in the critical care setting, ranging from 25% in symptomatic patients to 69% when identified through a surveillance venous ultrasonography.[3] The underlying pathophysiology for arterial thrombosis and VTE includes direct endothelial injury from viral infection,[3] immobility, prothrombotic internal environment due to the widespread systemic inflammation, and hypoxia.[4] This is a rare case of coronavirus disease with multiple organ infarcts involving the heart, lungs, and brain within a short time. The purpose of reporting this case is to inform the health care physicians about the risk of developing these complications, so that prophylactic therapy can be considered especially in high-risk patients.

CASE PRESENTATION
A 63-year-old male with insignificant past medical history developed low-grade fever and sore throat. SARS-CoV-2 polymerase chain reaction confirmed the diagnosis of coronavirus infection, and the patient was self-isolated at home. Oral antibiotics and steroids were initiated. and fever was initially relieved. The regimen was continued for four days before the patient's condition started to deteriorate, with fever spikes becoming more frequent and the patient starting to experience dry cough and myalgia. Oxygen saturation recorded during the first 6 days remained over 95% on room air. On the seventh day, the patient developed shortness of breath and was subsequently shifted to the hospital, where he was tachypneic at the time of presentation with an oxygen saturation of 67%. Computed tomography (CT) of the chest revealed bilateral patchy infiltrates on admission, pointing towards COVID-19 pneumonia (Figure 1).

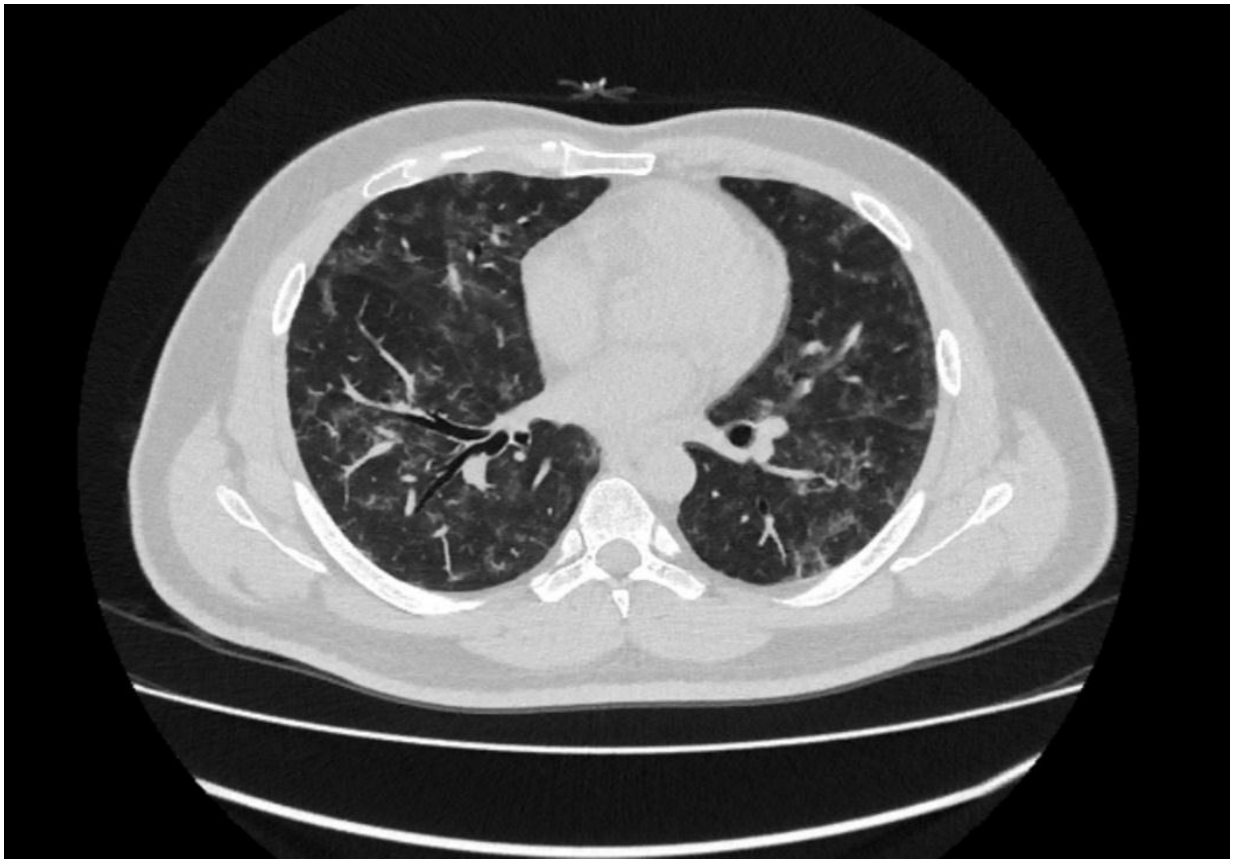
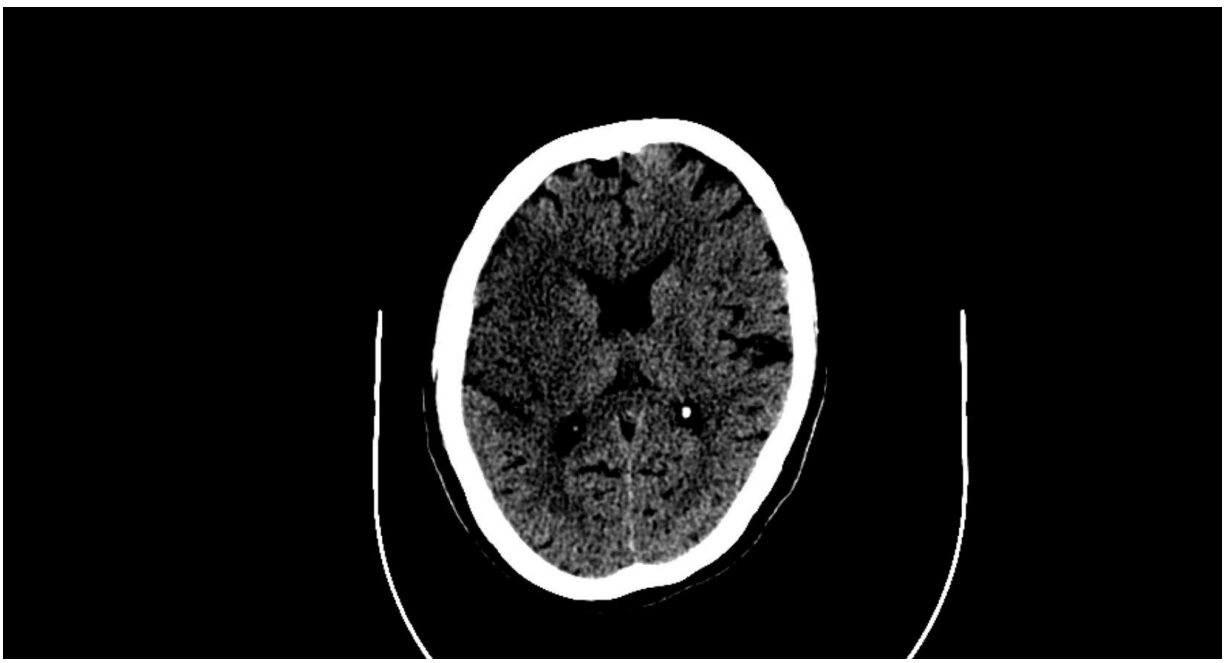


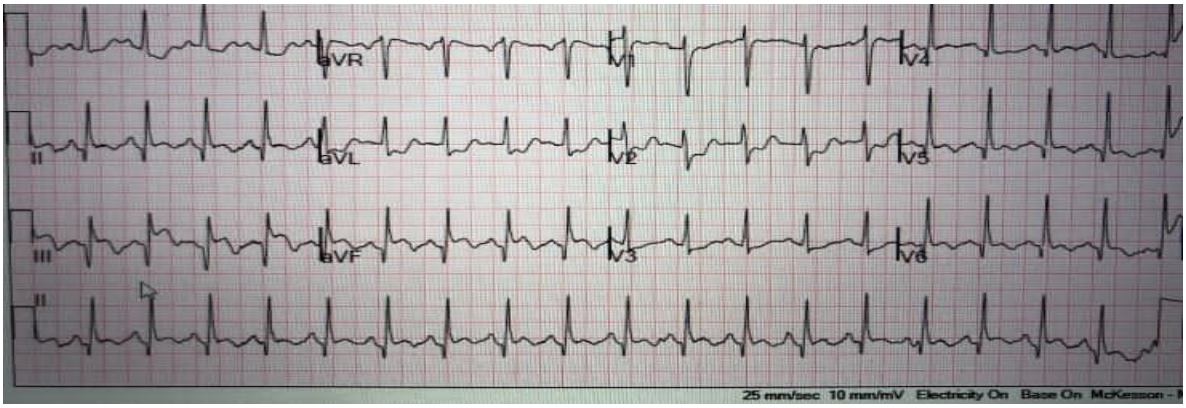
Figure 1: CT Chest revealing bilateral patchy infiltrates.

Saturation with a simple oxygen mask was 84%, but the patient was able to maintain oxygen saturation with continuous positive airway pressure. His condition remained static for the next three days. On the third post-admission day, the patient developed an altered level of consciousness with left-sided body weakness. He was clinically diagnosed with right-sided acute ischemic stroke; findings were confirmed on non-contrast brain CT (Figure 2).



Meanwhile, renal function tests deteriorated, and dialysis was done twice without any improvement in serum creatinine levels. On the fourth day of admission, saturation dropped once again, and the patient was put on

a mechanical ventilator. A 12-lead EKG at that time demonstrated inferior wall ST-elevation myocardial infarction (Figure 3) and cardiac enzymes were raised. Intravenous heparin and oral antiplatelet drugs were commenced. The patient remained on a ventilator for seven days. On the eighth day, the patient's condition deteriorated once again, with a sudden drop in saturation and elevated D-dimer levels, pointing towards a probable pulmonary embolism. Due to the sudden collapse of the patient, imaging could not be obtained.



DISCUSSION

Arterial thrombosis and VTE are serious complications of severe SARS-CoV-2 infection. COVID-19 pneumonia, if complicated by ARDS, can immensely increase the risk of large-artery thrombosis, symptomatic VTE, or adverse cardiovascular events.[3] In a survey conducted on 184 COVID-19 patients admitted in ICU, Klok et al. reported the incidence of VTE and arterial thrombosis as 27% and 3.7%, respectively. Pulmonary embolism, having an incidence of 81%, was the most common thrombotic event, followed by acute ischemic stroke.[5] In another study, the cumulative incidence of VTE was 25-49%, and that for acute ischemic stroke and acute myocardial infarction was 0.9-5% and up to 20%, respectively.[6] These studies suggest that COVID-19 is likely to create a thrombogenic environment in the body. In COVID-19 patients, an increased incidence of arterial thrombosis and VTE despite anti-thrombotic prophylaxis can be due to factors such as the onset of an inflammatory cascade, immobility, hypercoagulability due to virus-mediated systemic infection or bacterial coinfection, and indwelling catheters.[3] COVID-19 cases involving multi-organ infarctions have not been widely reported, hence making our case an unexpected event.

Ischemic Stroke

The incidence of acute ischemic stroke (AIS) is reported to be about 1.9% in COVID-19 patients with moderate-to-severe infection. Factors leading to vascular occlusion and subsequent AIS in COVID-19 patients include hypercoagulability secondary to the infection, systemic inflammation, cardiac arrhythmias leading to embolism, and vascular endothelial changes causing atherosclerotic plaque displacement.[7] Cases of AIS associated with SARS-CoV-2 infection have been widely reported,[8,12] AIS due to left middle cerebral artery occlusion along with an ST-elevation MI was reported in an 80-year-old COVID-19 patient by Moshayedi et al.[8] Valderrama et al. reported a complex case of AIS due to thromboembolic occlusion of the left intracranial internal carotid, left anterior cerebral, and proximal left middle cerebral arteries, managed by intravenous thrombolysis and mechanical thrombectomy.[9] In a study by Morassi et al., four out of six patients with severe COVID-19 pneumonia developed AIS with poor outcomes. These four patients developed acute kidney failure prior to stroke,[10] whereas our patient developed acute renal failure concomitant to the stroke and not before it. In a case series conducted by Jillella et al., the embolic type was predominant in 13 COVID-19 patients who experienced AIS during the disease.[11] COVID-19 patients have a propensity to develop large-sized brain infarcts due to thrombotic occlusion of large arteries, as seen in our patient. Along with antiplatelet and anticoagulant therapy, the role of recombinant tissue plasminogen activator therapy should also be considered.[7] Higher levels of neutrophil-lymphocyte ratio, D-dimer, C-reactive protein, serum ferritin, and fibrinogen are associated with poor prognosis of AIS in SARS-CoV-2 infection, causing severe morbidity and mortality in 75% of patients.[12]

Pulmonary Embolism

The similarity of respiratory signs and symptoms makes it challenging to diagnose PE in COVID-19 patients. Both conditions present with worsening of clinical symptoms. Chest CT angiography should be performed timely to identify superimposed PE if the clinical condition worsens suddenly or saturation drops. However, higher infectivity of the illness, immobility of the patient, and strict isolation can delay the imaging studies.[2] Multiple cases of PE in COVID-19 have been reported.[2,13,15] Vyas et al. reported a case of saddle PE managed by thrombectomy in a 32-year-old COVID-19 patient.[2] Danzi et al. reported a case of COVID-19 pneumonia and concomitant PE in a 75-year-old female without having any predisposing risk factors for thromboembolism.[13] A case of successful conservative management of PE and right heart failure in a COVID-19 patient was described by Ullah et al.[14] In our case, the

patient suddenly collapsed, and a further rise in D-dimer levels was noted before he was diagnosed with PE. The treatment of PE in COVID-19 patients is the same as that in other patients. Thrombolysis is usually done for hemodynamically unstable patients. Catheter-mediated thrombolysis and surgical embolectomy can be used in moderate and large-sized PE cases where systemic thrombolysis is not feasible.[2] Patients with PE are more likely to require critical care management and mechanical ventilation than patients without PE.[15]

Myocardial Infarction

It is well-documented that acute respiratory infection can increase the risk of acute coronary syndrome.[16] Our patient suddenly deteriorated when he had an MI and needed ventilatory support afterward. It is very important to consider MI as a possible differential diagnosis if the clinical condition of a COVID-19 patient starts to deteriorate suddenly.[17] Acute MI has been reported to occur in coronavirus disease.[8,18,19] Elevated levels of proinflammatory cytokines can cause diffuse coronary thrombosis in COVID-19 patients without any history or risk factors for coronary artery disease, as reported by Tedeschi et al., who observed acute infero-posterolateral STEMI in a 60-year-old male with coronavirus disease. Angiography showed a large thrombus in the dominant right coronary artery without clear evidence of atherosclerosis.[18] To perform PCI in a safe environment without putting the healthcare workers at risk for virus exposure is itself a challenge. Hence fibrinolytic therapy might be preferred for STEMI management in COVID-19 patients.[19] Isolated ECG changes can also occur in COVID-19 patients without any other evidence of myocardial injury such as elevated cardiac enzymes, abnormal transthoracic echocardiogram, or arrhythmias. Hence, the diagnosis of MI in a COVID-19 patient becomes tricky, especially if the prior history of ischemic heart disease is absent.[20]

ABBREVIATIONS

AIS: Acute ischemic stroke.
ARDS: Acute respiratory distress syndrome.
COVID-19: Coronavirus disease.
CT: Computerised tomography.
ECG: Electrocardiogram.
PE: Pulmonary embolism.
SARS-CoV-2: Severe acute respiratory syndrome coronavirus-2.
VTE: Venous thromboembolism.

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