CASE REPORT

Collateral damage of COVID-19: Late presentation of cerebrovascular accident

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SUMMARY

Coronavirus disease 2019 (COVID-19) is associated with an increased risk of death due to the sequelae of inflammation and thrombosis. Patients infected with COVID-19 are at risk of ischemic stroke due to the effects of inflammatory cytokine storm, platelet activation, endothelial dysfunction and prolonged stasis. We report a case of severe COVID-19 infection who developed acute ischemic stroke during the course of his illness. The patient did not carry any traditional risk of stroke or thromboembolic event and was vaccinated. He tested positive for COVID-19 infection, but due to respiratory distress, he was intubated and ventilated. On the 11th day in intensive care unit, he was noted to be hemiplegic with poor neurological recovery. Despite being on treatment dose enoxaparin, his computed tomography confirmed a lacunar infarct in his brain and bilateral pulmonary embolisms. Twenty-five days later, he remained well and was discharged home with dabigatran and atorvastatin.

INTRODUCTION

In December 2019, Wuhan, China, reported viral pneumonia called SARS-CoV-2, a recently identified betacoronavirus of suspected zoonotic origin. It was later confirmed and named coronavirus disease 2019 (COVID-19) by the World Health Organisation (WHO). The clinical spectrum of this infection is variable, ranging from being asymptomatic to having a critical illness characterised by acute respiratory distress syndrome, resulting in hypoxic respiratory failure. COVID-19 is associated with significantly higher morbidity and mortality in the elderly and patients with co-morbidities.[1]

COVID-19 is associated with an increased risk of acute stroke, similar to other respiratory infections, which had been reported to be between 3.2 to 7.8-fold higher.[2] A systematic literature review was conducted in April 2020 which showed that the proportion of COVID-19 patients with acute ischemic stroke was estimated to be 4.9%.[3] This increased risk of ischemic stroke is due to the effects of inflammatory cytokine storm, platelet activation, endothelial dysfunction and prolonged stasis.[2,3] We report a case of COVID-19 complicated with acute ischemic stroke and pulmonary embolism in a previously well man.

CASE PRESENTATION

A 65-year-old man who completed his vaccination presented with fevers, cough, anosmia and shortness of breath. He had no underlying co-morbidities or risk factors for thrombosis. On examination, he was febrile with a temperature of 38.3ºC, a pulse rate of 110 beats per minute and normal blood pressure of 130/80 mmHg. He was in respiratory distress, tachypnoeic, with a respiratory rate of 26 breaths per minute and oxygen saturation of 81 under room air. Other pertinent findings include bilateral crackles on auscultation of the chest. Due to the increased work of breathing and poor oxygenation, he was intubated and ventilated.

Polymerase chain reaction (PCR) for SARS-CoV-2 was positive. An arterial blood gas (ABG) was performed, indicating type 2 respiratory failure. His chest radiograph (CXR) confirmed changes in COVID-19 pneumonia (Figure 1). An electrocardiogram (ECG) showed sinus tachycardia with no acute ischemic changes. Other blood test results include a normal haemoglobin 13.7 g/dL, normal white cell count of 6.8 × 10^9/L, mild thrombocytosis 567 × 10^9/L, and normal lymphocyte count of 1.15 × 10^9/L. He had mildly raised serum creatinine 127 µmol/L. His liver enzymes were normal. C-reactive protein (CRP) was slightly raised to 20.8 mg/L, D-dimer 322 ng/mL and a normal ferritin 400 mcg/L. A carotid Doppler ultrasound was performed, which was normal and had no haemodynamic repercussions. Antiphospholipid tests were negative.

Thus, our impression was of severe COVID-19 pneumonia category five. He was transferred to the intensive care unit (ICU) for ongoing care. Initial management includes intravenous methylprednisolone 140 mg and later dexamethasone 20 mg daily on a tapering dose. In addition, subcutaneous enoxaparin 60 mg twice daily was commenced for deep vein thrombosis (DVT) prophylaxis.

Whilst weaning off mechanical ventilation on day 11, his Glasgow Coma Scale (GCS)remained poor, and he was noted not to move his right side. Neurological examination confirmed right hemiplegia. Babinski's sign was negative, and his deep tendon reflexes were normal. A computed tomography (CT) scan of the head showed hypodensity over the left corona radiata (Figure 2). We also performed high-resolution CT (HRCT) and CT pulmonary angiogram (CTPA) of the lungs, which showed bilateral lower lobe pulmonary

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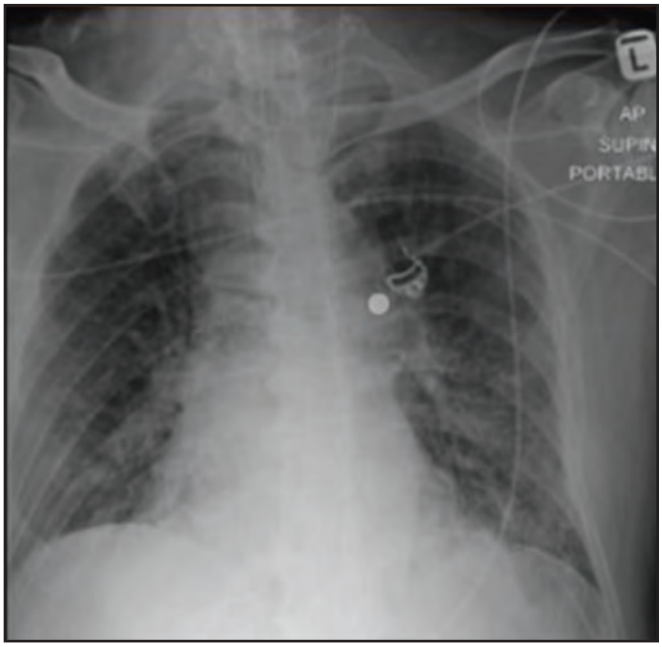

Fig. 1: Chest x-ray showed presence of ground-glass opacities in both lung

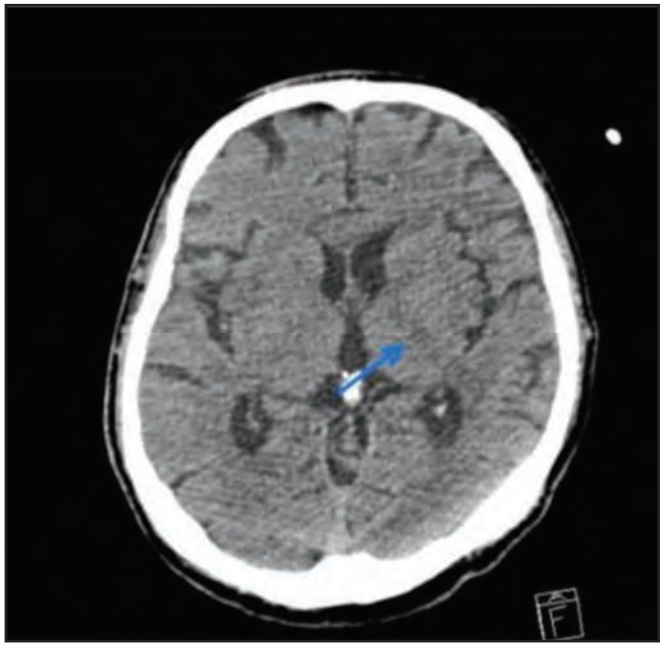

Fig. 2: A computed tomography (CT) scan of the head showed hypodensity over the left corona radiata

embolisms and organising pneumonia. During his ICU stay, his ECG on cardiac monitoring showed normal sinus rhythm.

He was later extubated and made a good recovery 3 weeks after extubation, not requiring further oxygen supplementation. His left hemiplegia improved; muscle power grade 4 over 5. His National Institutes of Health Stroke Scale (NIHSS) and modified Rankin Scale (mRS) were 5 and 3, respectively. He has been discharged home on dabigatran 15 mg and atorvastatin 40 mg. He had ongoing follow up with the physiotherapist and stroke rehabilitation team.

DISCUSSION

This case describes a confirmed case of COVID-19 infection complicated with acute ischaemic stroke and pulmonary embolism. The thrombotic events involving multiple organs in a patient with no known cardiovascular risk factors concomitant with COVID-19 infection are under-reported. The underlying pathological causes of brain ischemia and stroke in patients with COVID-19 include thrombosis, embolism and systemic hypoperfusion.

Thrombosis is caused by a cytokine storm and activation of the innate immune system, whereas embolic events are caused by pre-existing or new-onset cardiac arrhythmias. Patients with COVID-19 are found to have elevated levels of both interleukin-6 (IL-6) and CRP. IL-6 perpetuates the hypercoagulable state associated with COVID-19 through induction of tissue factor expression in mononuclear cells, triggering acute endothelial cell activation, activation of acute phase response resulting in enhanced fibrinogen production by hepatocytes and platelet hyperactivation and aggregation.[4] A thromboembolic storm can occur in patients with severe thrombosis. Clinical manifestations include right-sided cardiac overload due to pulmonary embolism or severe microvascular thrombosis like livedo racemosa or ischemia of the kidneys, muscle and liver. These will further increase the mortality risk of the patient.

Ischaemic stroke has been classified into different etiologic subgroups. These include cardioembolic, atherosclerotic, lacunar, other specific causes (dissections, vasculitis, specific genetic disorders, others) and strokes of unknown causes.[5] Revascularisation and the prevention of subsequent neuronal injury are the fundamental goals of advanced stroke treatment. Early intervention is essential in the reduction of morbidity and mortality. Predictors of premature mortality include advanced age, pre-stroke functionality, coronary artery disease and diabetes.[6] In an observational study, patients with neurological complications were found to have severe COVID-19 infection; up to 6% of patients had an acute ischemic stroke compared to 1% of patients with non-severe COVID-19 infection.[7]

Our case was challenging as the patient's presentation was noted while under sedation, and it was difficult to assess the patient's full neurological function, including GCS, power and sensation. In addition, the clinical detection of stroke could have been delayed due to deep sedation during mechanical ventilation. CT or MRI of the brain can be performed to aid the diagnosis in an intubated patient. However, rapid prehospital screening such as the Cincinnati Pre-Hospital Stroke Scale (CPSS) or Face, Arm, Speech, Time (FAST) score for prehospital screening is useful because any diagnosis is provisional until confirmed through investigation.[8]

CONCLUSION

A patient with no known cardiovascular risk factors for whom COVID-19 infection appeared to be an independent risk factor for developing acute ischaemic stroke due to the hypercoagulability state. Therefore, it is crucial to identify patients at higher risk of developing the thromboembolic disease by using the Cincinnati Pre-Hospital Stroke Scale (CPSS) or Face, Arm, Speech, Time (FAST) score as pre-admission assessment and start therapeutic dose of anticoagulation to reduce the risk of mortality and morbidity in those patients with high risk.

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CONFLICT OF INTEREST

The authors declare no competing interests with respect to the authorship and publication of this article.

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