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Delayed Presentation of Ischemic Stroke After COVID-19 Pneumonitis. How Late Can It Present? - An Interesting Case Report and A Review of Current Evidence

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Keywords:

COVID-19; Pneumonitis; Stroke; Coagulopathy; Anticoagulation

1. Abstract

Coronavirus disease 2019 (COVID-19) pandemic has now become a major public health concern throughout the world. Although symptoms in majority of patients are due to involvement of respiratory system, it is not unusual for other organ systems to be involved too. Micro vascular and macro vascular complications associated with this disease, contributing to the morbidity and mortality are increasingly being identified.

We report a 55-year-old gentleman who developed ischemic stroke twenty days after the initial diagnosis of COVID -19, with a background history of uncontrolled hypertension. A literature review of stroke associated with COVID-19 revealed that the presentation of stroke in COVID-19 is often atypical, pathophysiological mechanisms are multiple and different from the usual ischemic cerebrovascular events. Early preventative anti-coagulant therapy may reduce the stroke risk in some selected patients, but this needs further detailed research.

2. Introduction

The severe acute respiratory distress syndrome coronavirus-2 (SARS-COV-2) was first reported in Wuhan, China in December 2019 and subsequently became a global pandemic resulting in significant socio-economic burden throughout the world. A good proportion of patients remain asymptomatic in the early stages but can potentially develop delayed complications which can be serious and fatal in vulnerable populations [1]. Neurologic complication like stroke are increasingly recognized especially in younger patients who have no underlying vascular risk factors, with a reported incidence of up to 6% in hospitalized COVID-19 patients [2].

3. Case Presentation

A 55-year-old male patient presented to a Primary Care Health Centre for COVID-19 test after reporting contact with a COVID-19 positive patient. He was very well at initial presentation and reported no symptoms. He had nasopharyngeal and oropharyngeal swabs for COVID-19 reverse transcription polymerase chain reaction (RT-PCR) test which was reported the following day as positive with Cycle threshold (Ct) value of 16.95

As per the local primary care protocol for COVID-19 positive patients, he was called back to the Health Centre for further investigations. On examination, he was clinically well with no fever. But his vital signs examination revealed raised blood pressure of 214/129 mm of Hg, with rest of vital parameters namely respiratory rate, temperature and heart rate within normal limits. Systemic examination was unremarkable. Blood pressure was repeated several times but was persistently elevated with a systolic reading over 200.

On further exploring his past medical history it was revealed that he had a history of hypertension but stopped taking the medications for two years against medical advice. He had never smoked and had no history of any alcohol consumption. There was no family history of note and he was a chef by profession. He was commenced on Amlodipine 10 milligrams whilst he was still in emergency room in the Health Centre. In view of him being COVID-19 positive and having persistently high blood pressure, he was then referred to secondary care emergency services.

After four hours following the hospital admission, he developed left sided chest pain. An ECG showed sinus rhythm with left ventricular hypertrophy but no acute ischemic changes. A CT pulmonary angiography revealed bilateral multiple ground glass opacities with mosaic pattern indicating COVID 19 pneumonitis but no evidence of pulmonary embolism which was suspected (Figure 1). A standard blood panel work up showed normal complete blood count, renal profile, liver function tests, C-reactive protein and Troponin-T.

As per the local secondary care COVID-19 protocol, he was treated with Co-amoxiclav, Favipiravir and oral Dexamethasone. He also had low molecular Heparin as prophylaxis to prevent deep vein thrombosis. Blood pressure improved over next two days, and he was subsequently discharged to a quarantine facility after five days of hospital stay.

Twenty days after initial diagnosis of COVID-19 he presented to secondary care emergency department with symptoms of right sided weakness and numbness. Blood pressure was 209/119 mm Hg, but other vital parameters were normal. He had mild right upper motor neuron type right facial nerve palsy, drift on the right upper and lower limb with impaired sensation, right upper limb ataxia and a National Health Institutes Stroke scale (NIHSS) Score of 5. A standard blood panel work up was again normal during this hospital admission. COVID-19 test was still positive with Ct value of 22.82

CT scan of head did not demonstrate any significant abnormality (Figure 2). CT perfusion scan revealed mismatched areas of perfusion defect in the left temporo-occipital region involving the left posterior cerebral artery territory (Figure 3). CT angiogram of head and neck showed occlusion in the left posterior cerebral artery P1 segment with reduced flow distally in comparison to the contralateral side (Figure 4).

MRI of the brain showed focal areas of diffusion restriction in the left thalamus, with high signal intensity on T2, representing acute infarction (Figure 5, 6). MRA of the brain demonstrated persistent reduced flow in the left posterior cerebral in comparison to the right (Figure-7).

As the patient presented out of the treatment window period, thrombolysis was not considered. Thrombectomy could not be done either as the posterior cerebral artery occlusion was distal and hence, he was managed conservatively. Echocardiography showed normal global systolic left ventricular function with an ejection fraction of 55%, no regional wall motion abnormality and no visualized intramural thrombus was detected.

He was started on secondary prevention drugs namely statin and Aspirin. His blood pressure remained elevated and hence Lisinopril was added as well to his treatment regimen. He was discharged from hospital five days after admission. Over the next one month he made a gradual and complete recovery without any need for further intervention and is being followed up in Neurology clinic.

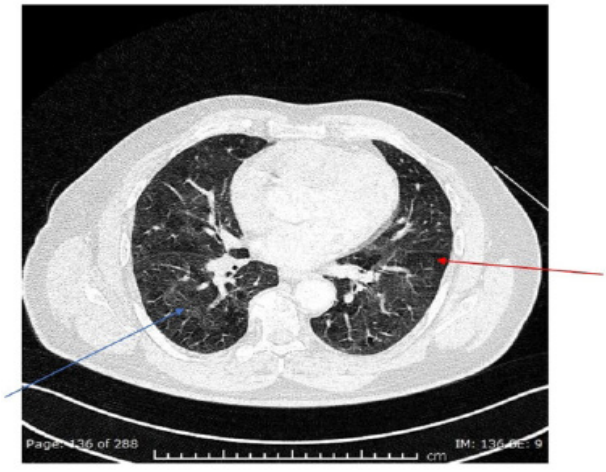

Figure 1: IV contrast enhanced axial CT pulmonary angiogram (on lung windows) demonstrates subtle ground glass opacification (blue arrow) with intervening areas of relatively normal parenchyma (red arrow) suggestive of a “mosaic” pattern. Features in keeping with pneumonitis. No pulmonary emboli detected on the scan.

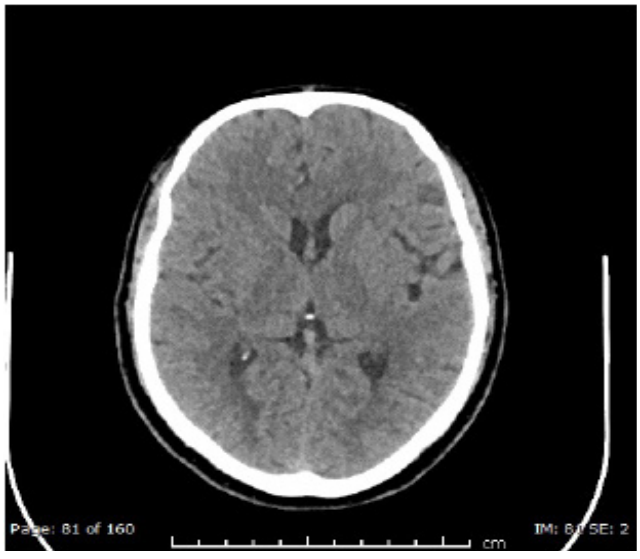

Figure 2: Non contrast axial CT head did not demonstrate any significant abnormality

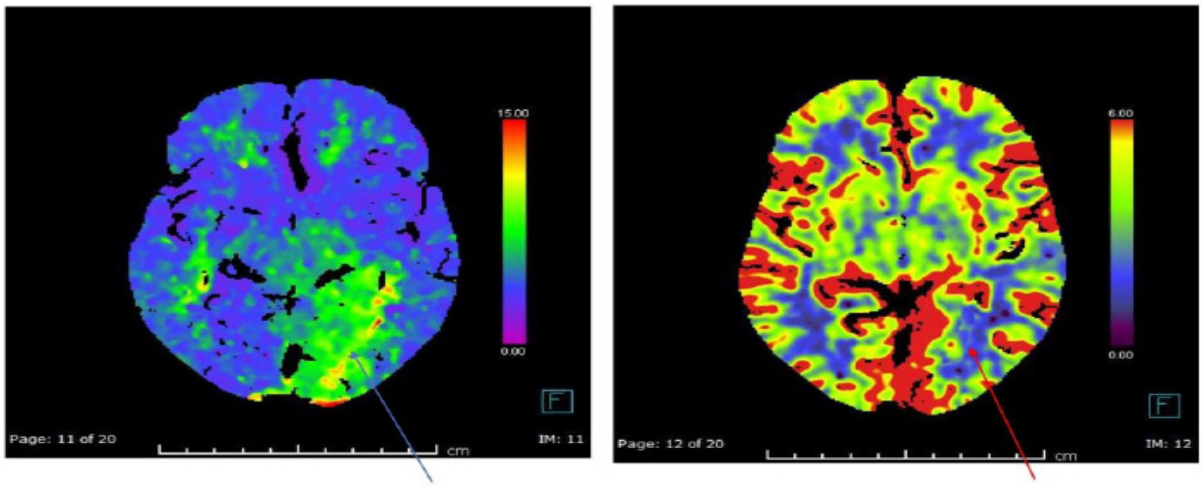

Figure 3: IV contrast enhanced axial CT with perfusion imaging demonstrates left temporo-occipital increased time to drain (blue arrow) mismatched with the cerebral blood volume (red arrow). Therefore, features suggest an ischaemic penumbra in this region.

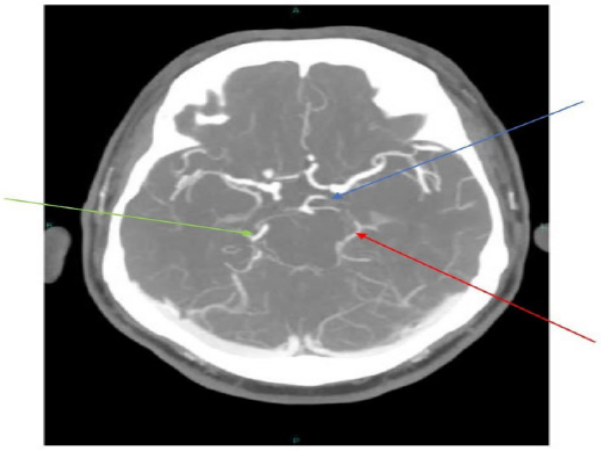

Figure 4: IV contrast enhanced CT angiogram demonstrates occlusion in the left posterior cerebral artery P1 segment (blue arrow). There is reduced flow distally (red arrow) in comparison to the contralateral side (green arrow).

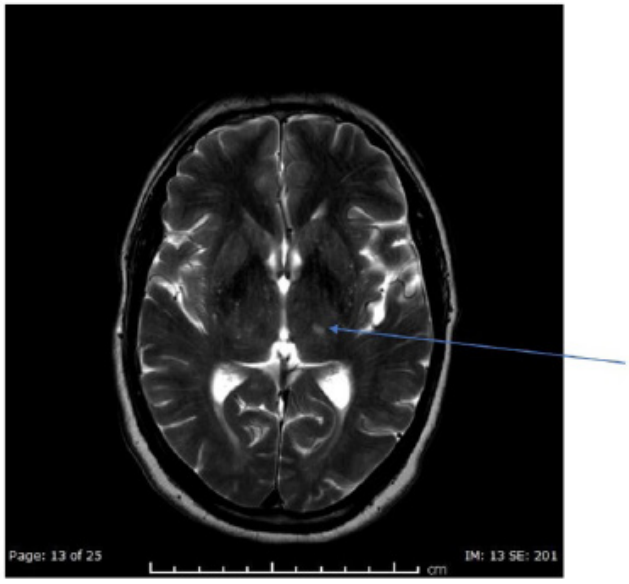

Figure 5: T2 weighted axial MRI scan demonstrates subtle hyperintensity in the left thalamus (blue arrow)

4. Discussion

Thromboembolic complications including stroke although rare, can be quite serious and sometimes fatal in COVID-19. The proposed mechanisms for COVID related stroke include a hypercoagulable state after the hyperinflammatory response [2], postinfectious immune-mediated responses [2] and direct viral-induced endothelitis [3].

Hypercoagulable state in COVID-19 resulting in cardiovascular and neurovascular complications have been reported even in early case studies [4]. Mechanism of coagulopathy related to COVID-19 is quite complex and involves complement pathway. C5a induced systemic hyper inflammatory state – otherwise named cytokine storm leads to lung injury, lymphocyte exhaustion, and an immune paresis. Further imbalance of the homeostatic interactions between complement and coagulation pathways result in a pro-coagulant state which can involve the microvasculature of vital organs causing microthrombi [5].

Earlier analysis of COVID-19 cases could not establish a causal effect on ischemic stroke, as competing vascular risk factors and mechanisms were present in most patients [6], but a later study from United States in August 2020 concluded that COVID-19 infection had a significant independent association with image confirmed acute ischemic stroke compared with control subjects even after adjusting for age, sex, and any underlying vascular risk factors [7]. Stroke presentations are frequently atypical with 67.4% having non focal neurological presentations and 45.8% involving multivascular distributions [8].

Prior to the presentation with acute stroke our patient was confirmed to have pneumonia on the CT chest done at first presentation. Interestingly a case series from Italy reported an association between stroke and pneumonia severity in COVID-19 patients, with several possible reasons like hypoxemia aggravating cerebral ischemia and stroke associated pneumonia [9].

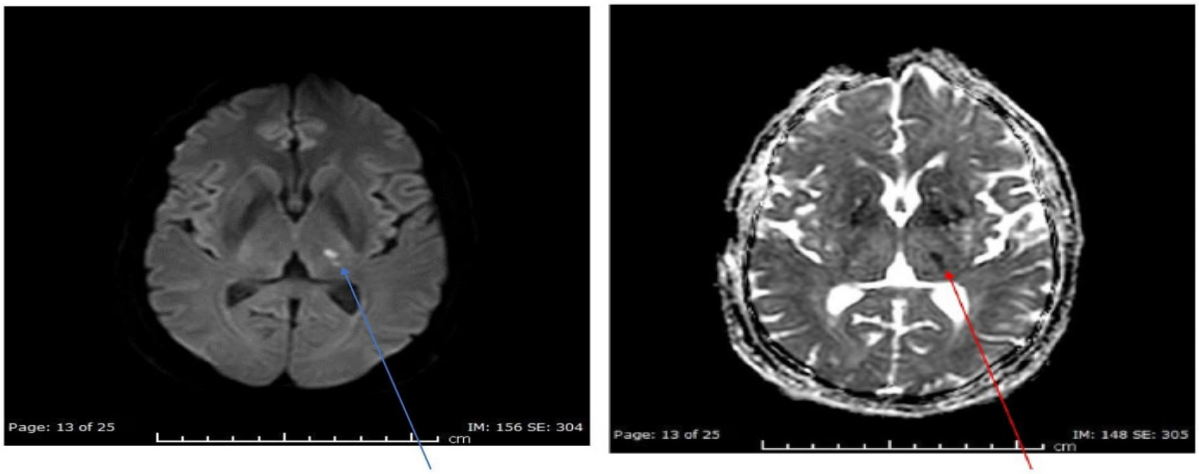

Figure 6: MRI Head DWI sequence demonstrates hyperintensity in left thalamus (blue arrow). There is corresponding signal drop out on the ADC map (red arrow) confirming restricted diffusion. Features in keeping with acute focal thalamic infarct. However, despite CT perfusion scan performed 1 day earlier, there are no ischaemic changes in the temporo-occipital lobe.

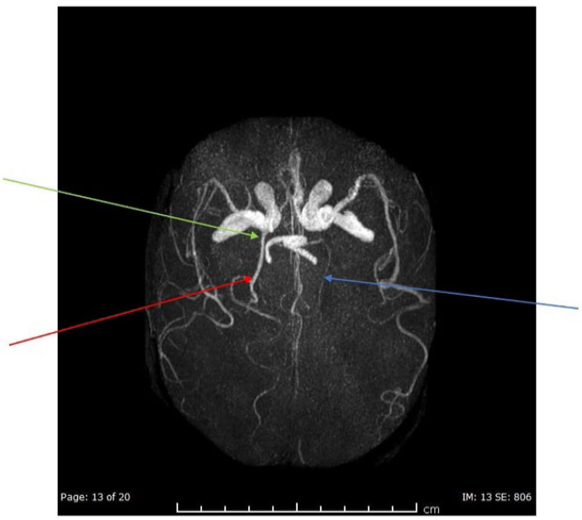

Figure 7: Time of flight 3D intracranial angiogram demonstrates persistent reduced flow in the left posterior cerebral artery (blue arrow) in comparison to the right (red arrow). Note that filling of the right side is via the posterior communicating artery (green arrow) which is a normal variant.

In our patient the presentation of stroke was delayed by 20 days from the day of COVID test positivity and happened after being discharged from hospital. A recent systematic review of COVID-19 patients affected by acute ischemic stroke indicated a delayed presentation with a mean duration of 10 +/- 8 days, from the onset of COVID-19 symptoms [10]. This is in keeping with the current knowledge of pathophysiology of severe COVID-19 infection where in an early hyperinflammatory state from cytokine storm is followed by a prothrombotic state complicated by venous and arterial thromboembolism. In the same review, reported incidence of acute ischemic stroke in COVID-19 patients ranged from 0.9% to 2.7% with a high mortality rate of 38.0%. On an average they had a moderate NIHSS score of 19±8 and a high prevalence (40.9%) of large vessel occlusion [10].

In a large UK based multicenter case control comparison study of stroke with and without COVID-19, Perry et al suggested that COVID-19 may provoke the onset of an ischemic stroke through a variety of thrombotic and inflammatory mechanisms, promoting generation of thrombus in the heart or large vessels or via small vessel occlusion. Which of these mechanisms manifests in a given patient may be determined by that individual's conventional vascular risk factors [11]. Our patient had chronic hypertension and subsequently found to have small vessel occlusion on CT angiogram which was likely the etiology of ischemic stroke.

In view of the prothrombotic risk, it has been proposed that high risk patients with COVID-19 should be anticoagulated. The duration and the type of anticoagulation and antiplatelet therapy is still unclear, but much discussion has been centered around heparin as a promising contender for anticoagulation [12].

5. Conclusion

Stroke even though rare in COVID -19 can be a serious complication especially in presence of other risk factors. Clinicians should have a low threshold for investigating such patients. In view of the potential for hypercoagulable state in severe COVID-19 infection, prophylactic anticoagulation should be considered as a part of COVID-19 treatment regimens. Further studies are also required to explore the correlation of COVID-19 pneumonia and stroke.

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