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**COVID-19 presenting as stroke.**

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**ABSTRACT**

**Objective:** Acute stroke remains a medical emergency even during the COVID-19 pandemic. Most patients with COVID-19 present with constitutional and respiratory symptoms, some patients present with atypical symptoms including gastrointestinal, cardiovascular, or neurological symptoms. Here we present a series of four COVID-19 patients with acute stroke as a presenting symptom.

**Methods:** We searched the hospital databases for patients presenting with acute strokes and suspected COVID-19 features. All patients that had imaging confirmed strokes and PCR confirmed COVID-19 were included in the study. Patients admitted to the hospital with PCR confirmed COVID-19 disease whose hospital course was complicated with acute stroke while inpatient were excluded from the study. Retrospective patient data were obtained from electronic medical records. Informed consent was obtained.

**Results:** We identified four patients presenting with imaging confirmed acute strokes and PCR confirmed SARS-CoV-2 infection. We elucidate the clinical characteristics, imaging findings, and the clinical course.

**Conclusions:** Timely assessment and hyperacute treatment is the key to minimize mortality and morbidity of patients with acute stroke. Stroke teams should be wary of the fact that COVID-19 patients can present with cerebrovascular accidents and dawn appropriate personal protective

equipment in every suspected patient. Further studies are urgently needed for a comprehensive understanding of the neurological pathology of COVID-19 and its effects on the nervous system.

## INTRODUCTION

In December 2019, the first reports of the corona Virus Disease 2019 (COVID-19) – an illness caused by the novel severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) – emerged from Wuhan, Hubei Province, China.[1] Since then, this disease has become a worldwide pandemic, with over one-hundred thousand deaths to date.[2, 3] Symptoms of SARS-Cov-2 infection range from asymptomatic disease to life-threatening acute respiratory distress syndrome (ARDS), severe pneumonia, acute kidney injury (AKI), myocarditis, eventual multi-organ failure, and death.[4, 5] Recent literature reported multiple neurological manifestations including cerebrovascular accidents in patients with severe infection.[6] So far, there are no reported cases of COVID-19 presenting with strokes in the literature. Here, we report a case series of four patients that presented with ischemic stroke in the setting of PCR-confirmed SARS-CoV-2 infection.

## METHODS

We searched the hospital database for patients presenting with acute strokes and suspected COVID-19 features. All patients that had imaging confirmed strokes and PCR confirmed COVID-19 were included in the study. Patients admitted to the hospital with PCR confirmed COVID-19 disease whose hospital course was complicated with acute stroke while inpatient were excluded from the study. Retrospective patient data was obtained from electronic medical records. Informed consent was obtained.

**RESULTS:**

We identified four patients that presented with imaging confirmed acute stroke and PCR confirmed COVID-19 disease. We elucidate the clinical characteristics, imaging findings, and the clinical course. Laboratory data are presented in table 1.

**Patient 1**

73-year-old male with a past medical history of hypertension, dyslipidemia, and carotid stenosis presented to the emergency department(ED) with fever, respiratory distress, and altered mental status. Review of systems was positive for dyspepsia, nausea, vomiting, reduced oral intake for two days, and negative for fevers or chills at home. He had a sick contact at home. Vital signs on presentation were fever of 101, tachycardia with heart rate(HR) of 102, hypoxemia with saturating 85% on 100% non-rebreather. The patient was intubated in the ED for hypoxic respiratory failure. A computed tomography (CT) of the head was performed for altered mental status which demonstrated loss of gray-white differentiation at the left occipital and parietal lobes, consistent with acute infarct (figure 1a). CT of the chest demonstrated bilateral peripheral patchy airspace opacities and diffuse ground-glass opacities, characteristic for atypical pneumonia/viral infection from COVID-19 (Figure 1b). Electrocardiogram (EKG) was within normal limits. Blood work was significant for leukocytosis with lymphopenia. Urine analysis was within normal limits. C-reactive protein was elevated to 26mg/dl (0-0.4mg/dl). D-Dimer was not checked during the hospital course. COVID-19 Polymerase chain reaction (PCR) detected the virus. Blood and urine cultures did not yield any growth. A repeat CT of the head demonstrated progressive large acute infarct of the left MCA territory with hyperdense appearance of left MCA vessels consistent with acute thrombus. The patient was deemed not a candidate for thrombolysis or neuro-intervention due to poor functional status. The patient was treated with aspirin and other supportive measures. No abnormal heart rhythm was noted on

telemetry monitoring. The family eventually chose for comfort measures and terminally extubated the patient.

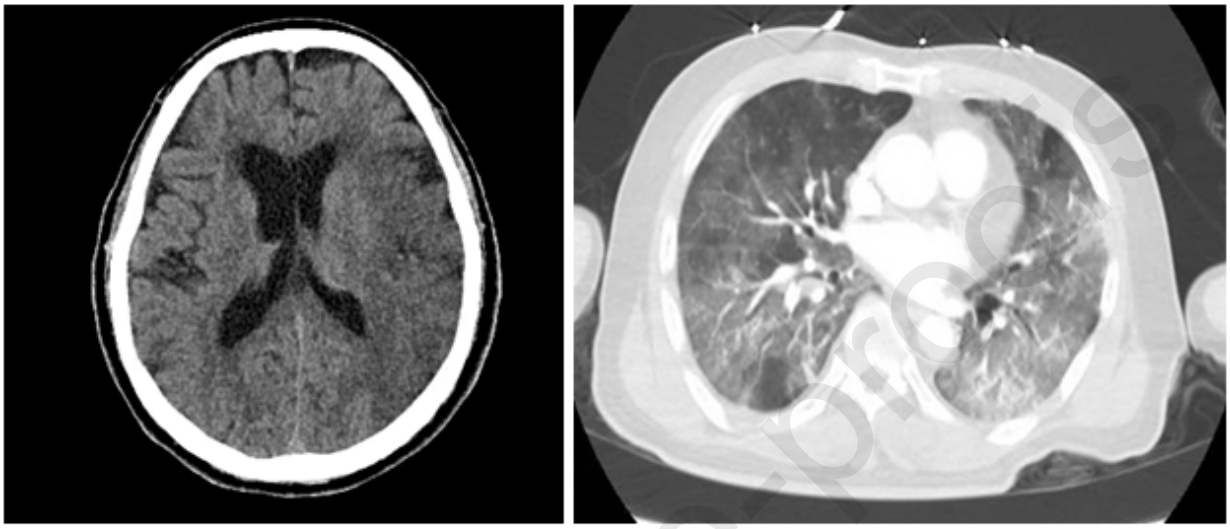
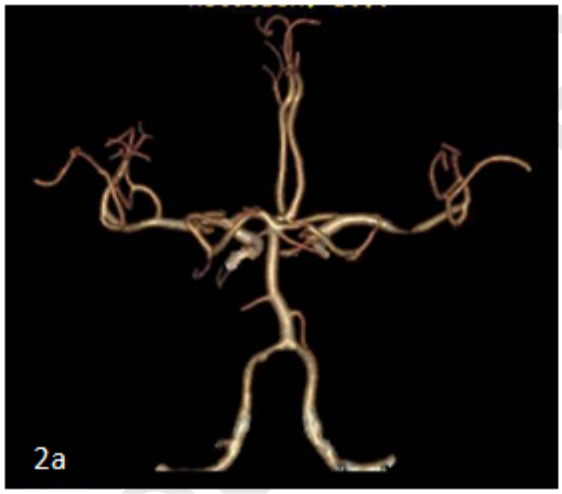


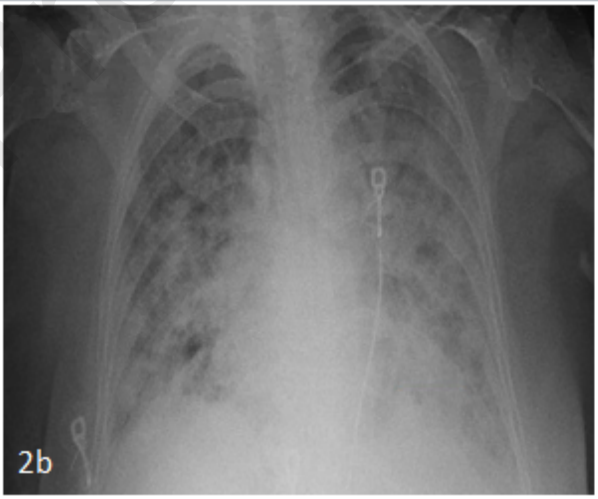
Figure 1a: A CT of the head demonstrated loss of gray-white differentiation at the left occipital and parietal lobes, consistent with acute infarct
Figure 1b: CT chest demonstrating bilateral peripheral dominant patchy airspace opacities and diffuse ground glass opacities, characteristic for atypical pneumonia/viral infection from COVID-19

**Patient 2**

83-year-old female with a past medical history of frequent urinary tract infections, hypertension, hyperlipidemia, diabetes mellitus type 2, neuropathy presented to ED with fever, facial droop, slurred speech, and reduced oral intake. Initial vital signs were significant for temperature of 101.4, HR of 94, blood pressure (BP) of 172/64, saturating 94% on room air. The examination was significant for left facial droop and slurred speech with no other significant neurological deficits. National Institute of health stroke scale(NIHSS) was calculated to be 2. Blood work was significant for leucopenia with lymphopenia. Urine analysis was within normal limits, chest X-Ray showed bilateral peripheral opacities. CT head was negative for any acute changes. CTA of head and neck demonstrated no large vessel occlusion, focal moderate stenosis of right MCA

(Figure 2a), and incidental pulmonary ground-glass opacities in bilateral apices. COVID-19 was suspected given the fevers and CT findings. COVID-19 PCR detected the virus. D-DImer and other inflammatory markers were not checked. CXR progressively worsened with bilateral infiltrates (Figure 2b). Urine and blood cultures did not yield any growth. No arrhythmias were noted during the hospitalization. On day-3 of hospitalization, the patient developed left-sided hemineglect, worsening left-sided facial droop with left hemiparesis. The speech was normal. NIHSS was calculated to be 16. Repeat CT of the head demonstrated a new moderate hypodensity in the right frontal lobe representing acute infarct (Figure 2c). The patient was deemed too high risk for thrombolysis or neuro-intervention. A CT angiogram was planned prior to starting Integrellin but her respiratory status worsened requiring intubation and mechanical ventilation. Soon after, the family decided to withdraw care.



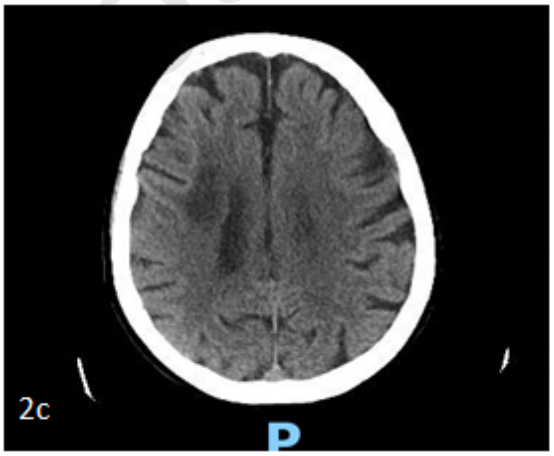



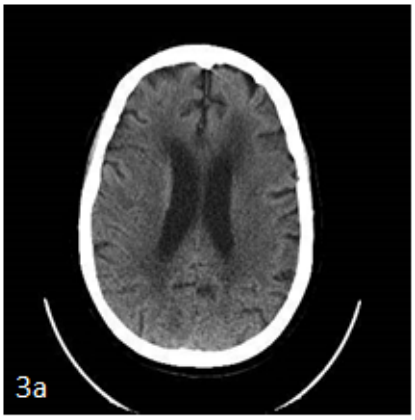

Figure 2a: CTA of head and neck demonstrated no large vessel occlusion, focal moderate stenosis of right MCA

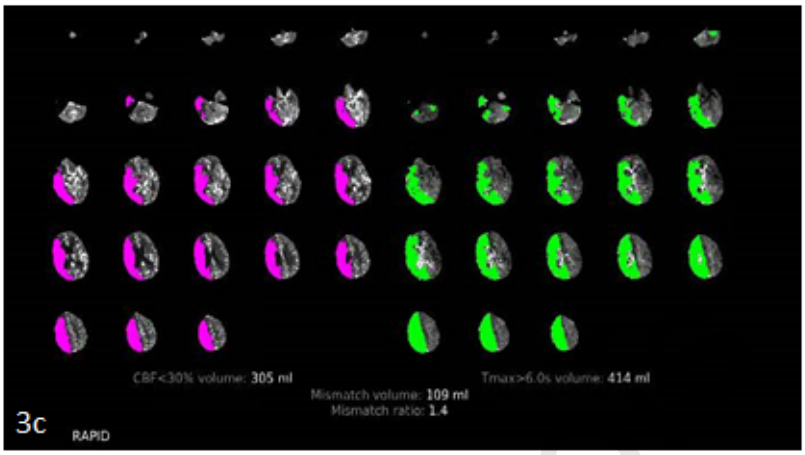
Figure 2b: CXR demonstrating worsening bilateral opacities

Figure 2c: CT of head demonstrated new moderate hypodensity in the right frontal lobe representing acute infarct

**Patient 3**

80 Year-old-female with history of Hypertension brought in to the ED for a chief complaint of altered mental status and Left-sided weakness. The family denied history of fever or cough but reported that the patient has been falling frequently in the past week. The patient was intubated for airway protection and code stroke was activated. Vital signs in ED were significant for Temp of 100.2, HR 101, BP 130/77, Examination was significant for left hemiplegic and aphasia. NIHSS was calculated to be 36. CT Head revealed acute right MCA stroke (figure 3a). CTA of head and neck demonstrated occlusion of the right internal carotid artery at origin and incidental bilateral patchy apical lung opacities (Figure 3b). CT perfusion demonstrated 305 cc core infarct in the right MCA distribution and 109 cc surrounding ischemic penumbra (Figure 3c). The patient was deemed not a suitable candidate for any acute neuro-intervention due to large core infarct. Considering characteristic CT findings the patient was tested for COVID-19 PCR which detected the virus. Laboratory data on admission demonstrated leukocytosis with lymphopenia, elevated d-dimer (13966 ng/ml DDU) along with elevated Lactate Dehydrogenase (712 U/L) and elevated C - reactive protein(16.24 mg/dl). Patient's hospital course was complicated by acute kidney injury and progressively increasing oxygen requirements. On the third day of admission, her family chose for terminal extubation with comfort measures.



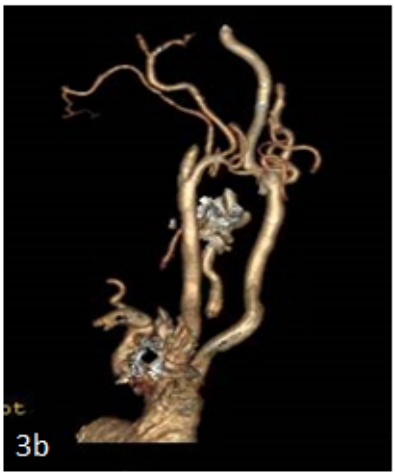
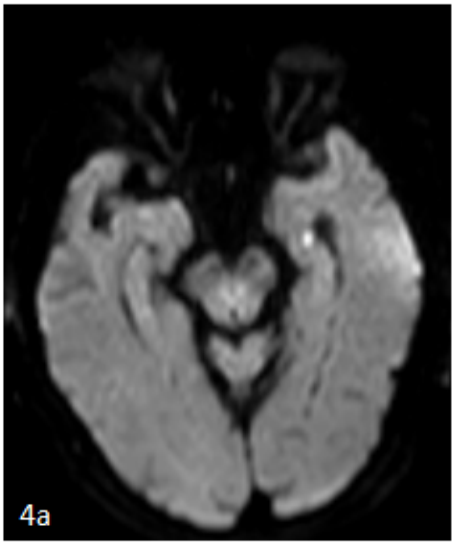




Figure 3a: CT Head demonstrating acute right MCA stroke
Figure 3b: CTA of head and neck demonstrating occlusion of right internal carotid artery at origin
Figure 3c: CT perfusion demonstrating large core infarct

**Patient 4**

An 88-year-old female with a past medical history of hypertension, chronic kidney disease, and hyperlipidemia presented to ED with a transient 15-minute episode of right arm weakness and numbness along with word-finding difficulty. Vital signs on presentation were within normal limits. The initial neurological examination was normal. Code stroke was activated. CT head did not show any acute findings. EKG showed normal sinus rhythm. Thrombolysis was deferred, and she was admitted with a diagnosis of transient ischemic attack. As the patient complained of mild shortness of breath with dry cough, Covid-19 PCR was sent which detected the virus. D-Dimer (<880 ng/ml) was elevated to 3,442ng/ml, other inflammatory markers were elevated. Magnetic Resonance Imaging (MRI) showed an acute infarct in the left medial temporal lobe (Figure 4a). Magnetic resonance angiogram (MRA) of head and neck revealed mild stenosis of the right M1

segment (Figure 4B). No arrhythmias were noted on telemetry. Patient was treated with aspirin, statins, and was discharged to a rehab facility with an event monitor.



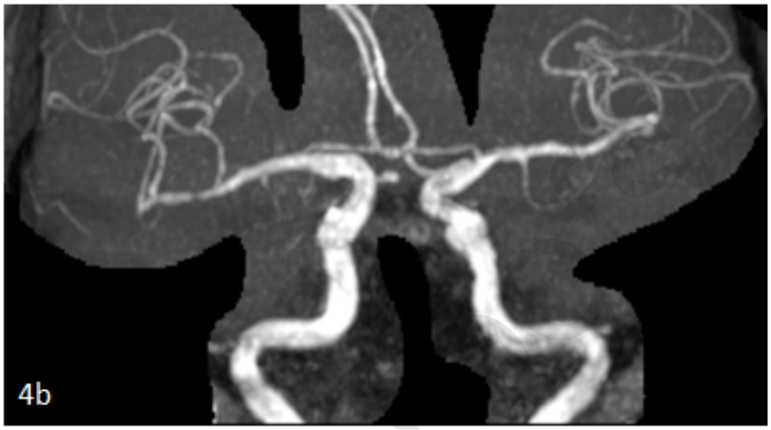




Figure 4a: MRI of brain demonstrating acute infarct in the left medial temporal lobe
Figure 4 b: MRA of head and neck demonstrating mild stenosis of right M1 segment

**Discussion**

To our knowledge, this is the first series of PCR confirmed COVID-19 cases presenting as cerebrovascular accident. In the study of Mao et.al, 5.7% of patients developed cerebrovascular disease later in the course of illness in patients with severe infection.[6] In a study by Li Y et.al, the incidence of stroke in COVID-19 patients was about 5% with a median age of 71.6 years.[7] These patients were associated with severe disease and had a higher incidence of risk factors like hypertension, diabetes, coronary artery disease, and previous cerebrovascular disease.[7] Average time of onset of stroke after COVID-19 diagnosis was 12 days.[7] Elevated CRP and D-dimer indicating high inflammatory state and abnormalities with coagulation cascade respectively might play in role in strokes with COVID-19 infection.[7] All the four cases in our report presented with cerebrovascular accident rather in early stages of illness.

The SARS-CoV-2 virus formerly called the novel coronavirus is the seventh coronavirus that infects humans.[8] SARS-CoV-2 is genetically similar to SARS-CoV-1.[9] SARS-CoV-1 in 2003 affected about 8000 patients with few reports of neurological manifestations, mostly peripheral neuropathy and encephalitis.[10] Similar to SARS-CoV-1, current SARS-CoV-2 affects the neurological system in about 36.7% as per Mao et.al.[6] Most coronavirus are neurotropic, similarly few studies speculate that SARS-CoV-2 is neurotropic.[11] Furthermore, there are reports of SARS-CoV-2 being identified in cerebrospinal fluid by PCR.[12] Angiotensin converting enzyme -2 (ACE) receptors are the major entry points for SARS-Cov-2 and other coronaviruses.[13] Although ACE-2 receptors are present on the nervous system, various pathophysiology have been proposed to explain the entry of SARS-CoV-2 into the nervous system – direct injury (blood and blood brain barrier), hypoxic injury, ACE2 receptors, immune injury.[11, 14]

The pathophysiology behind the cerebrovascular accidents is still to be determined. Recent bacterial or viral infections have been known to cause strokes by increasing cardioembolism as well as arterio-arterial embolism.[15] A recent study from the Netherlands by Klock et al. demonstrated that 31% of critically ill ICU patients develop thrombotic complications.[16] Another study looking at activated partial thromboplastin time-based clot waveform analysis(CWA) in COVID-19 patients concluded that CWA parameters demonstrate hypercoagulability that precedes or coincides with severe illness.[17] Multiple reports of pulmonary embolism are currently available in the literature.[18]Autopsy and pathology findings are scarcely available, but recent autopsy findings suggest thrombotic microangiopathy in multiple organs especially in the lungs.[19-21] No autopsy reports of the brain are available at the time of this writing. With this evidence, the most plausible mechanism of early cerebrovascular accidents could be hypercoagulability leading to macro and micro thrombi formation in the vessels. Other

pathophysiology could be directly related to the infection or hypoxia. Further studies are urgently needed for comprehensive understanding neurological pathology of COVID-19 and its effects on the nervous system.

## TREATMENT IMPLICATIONS

COVID-19 pandemic necessitates extra measures to be taken to provide the care for stroke patients along with measures aimed at minimizing the spread of the infection. Some of the challenges we encounter with acute stroke patients are their inability to effectively communicate due to speech problems, altered mental status, inadequate history, because of the limitations of visitors by hospital policies, etc.

Khosravani. et al. proposed a Protected Code Stroke (PCS) concept during this pandemic which provides a framework for key elements like screening guidelines, PPE, and crisis resource management[22]. Based on previous studies, recommendations for PCS include: Paramedics should develop an infectious screening policy on all patients with stroke-like presentations, before bringing them to the hospital.[22] Outside transfers should be minimized, and even the ones that need transfer should have an infectious screening before the transfer.[22] A dedicated neurology hot-spot along with a mobile CT unit for COVID-19 patients with stroke-like symptoms is beneficial.[23] Clinically stable patients after thrombolysis can be monitored on non-intensive care units.[23]

## CONCLUSION

Stroke teams should be wary of the fact that COVID-19 patients can present with cerebrovascular accidents and dawn appropriate personal protective equipment in every suspected patient. Plans should be developed not to neglect the management of acute

cerebrovascular accidents, even though the control of COVID-19 infection is our biggest priority. More research is needed to identify the neurological implications of COVID-19 disease.

**Table 1. Pertinent laboratory findings**

| **Laboratory Findings** | **Patient 1** | **Patient 2** | **Patient 3** | **Patient 4** |
|---|---|---|---|---|
| | | | | |
| White blood cell count (4.8-10.8 K/μL) | 12.32 | 4.95 | 18.89 | 7.5 |
| Neutrophils (1.4-6.5 K/μL) | 10.95 | 3.41 | 16.36 | 5.3 |
| Lymphocytes (1.2-3..4 K/μL) | .67 | 1.07 | 1.15 | 1.5 |
| Platelet count (130 - 400per $mm^3$) | 182 | 138 | 380 | 176 |
| Hemoglobin(12-16 g/dL) | 15.5 | 15.1 | 12.9 | 11.6 |
| Albumin(3.5-5.2g/dL) | 2.7 | 4.1 | 3.2 | 2.9 |
| Alanine aminotransferase(0-41 U/L) | 55 | 25 | 18 | 14 |
| Aspartate aminotransferase(0-41 U/L) | 47 | 31 | 34 | 18 |
| Lactate dehydrogenase (50-242 U/L) | NA | NA | 712 | 200 |
| Creatinine(.7-1.5 mg/dL) | 1.1 | 1 | 1.6 | 1.8 |
| Cardiac Troponin T (<.01 pg/ml) | <0.01 | <0.01 | .11 | .14 |
| Prothrombin Time(9.95-12.87 sec) | 19.70 | 13.5 | 15.2 | 13.5 |
| Activated partial thromboplastin time (27-39.2 sec) | 27.6 | 27.2 | 38.2 | 27.7 |
| D-Dimer (0-230 ng/mL) | NA | NA | 13966 | 3442 |
| Ferritin (15-150 ng/L) | NA | NA | 891 | 135.9 |
| Procalcitonin (0.02-0.10 ng/mL) | 14 | NA | 0.49 | .08 |
| C-reactive protein (0.00-0.40mg/dL) | 26.22 | NA | 16.24 | 12.7 |

| | | | | |
|---|---|---|---|---|
| IL-6 (0-6.3 pg/mL) | NA | NA | NA | 8.5 |

Notes: The first available laboratory data are presented here. NA: Not available.

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- Acute stroke is a medical emergency even during the COVID-19 pandemic. Most COVID-19 patients present with constitutional and respiratory symptoms.
- Recent literature reported about 5% patients developed cerebrovascular disease later in the course of illness. Here, we report a case series of four patients that presented with acute ischemic stroke.
- Stroke teams should be wary of the fact that COVID-19 patients can present with cerebrovascular accidents and dawn appropriate personal protective equipment in every suspected patient.