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Acute Stroke in COVID-19 Patients

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Abstract The novel Coronavirus-19 (COVID-19) outbreak has brought unprecedented implications globally for the medical community and continues to present challenges when delivering neurological care and intervention. Here we describe six patients with acute cerebrovascular symptoms and concurrent COVID-19 infection who presented to our hospital. This is a retrospective case series study of six patients presenting with neurological, either with or without respiratory symptoms. Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection was suspected in the patients. Testing was conducted by BioReference Lab with the reverse-transcriptase polymerase chain reaction (RT-PCR) assay for most patients. Brain imaging revealed evidence of acute cerebral infarction. Ethical approval and informed consent waivers when appropriate were granted by the Mount Sinai Institutional Review Board. The study was conducted at NYC Health + Hospitals-Queens in Jamaica, New York. All patients were men, ages 39 to 69 years, four of whom were found to have associated risk factors for stroke. Inflammatory markers were elevated in most patients and brain imaging revealed ischemic infarcts. With the exception of one patient who expired, five patients made a significant recovery. Ischemic stroke was one of many newfound complications related to the COVID-19 infection. In our case reports, many patients did not present with respiratory symptoms, however, all tested positive for COVID-19. Neurological sequelae related to COVID-19 apart from cerebrovascular disease continue to be discovered. With growing evidence that the central nervous system is vulnerable to the devastating effects of COVID-19, testing should be expedited in all patients presenting with neurological symptoms during the COVID-19 pandemic.

Keywords: *CVA/Acute stroke, COVID-19, coronavirus, neurology, hypercoagulability*

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1. Background

The outbreak of Coronavirus 2019 (COVID-19) disease has caused widespread implications for medical practice including acute stroke care [1]. As of September 2020, 26 millions people have been infected globally [2]. Clinical presentation can range from mild symptoms of fever and respiratory infection to severe pneumonia, acute kidney injury, myopericarditis, and multi-system manifestations that necessitate intensive care [3,4]. There is accumulating evidence of cerebrovascular events in COVID-19 patients attributed to a state of hypercoagulability [5]. The reported rate of detection of stroke in COVID-19 patients ranges from 2.4% to 6% [6,7]. In addition, studies have shown higher incidence of stroke in adults with COVID-19 infection as compared to influenza [8].

Various mechanisms have been proposed to explain the occurrence of ischemic strokes following COVID-19 infection, including the activation of a distinct hypercoagulable state, leading to both localized and disseminated thrombosis. A proposed theory involves the pro-inflammatory cascade regulated by specific immunologic cytokines, such as interleukin-6, implicated in cytokine storm and systemic inflammation [9]. In addition, emerging reports suggest that the presence of antiphospholipid antibodies in the setting of COVID-19 may also contribute to coagulopathy in this population [10,11].

Diagnosis and management of acute stroke during the COVID-19 pandemic has heralded novel challenges for both patients and physicians with the need to maintain quality care amidst limited resources and avoid viral transmission from patients to physicians. In order to characterize acute stroke care during the COVID-19 pandemic, it is important to evaluate research and findings and produce evidence-based guidelines.

We reviewed clinical courses, imaging findings, and outcomes of six COVID-19 patients admitted with acute ischemic strokes at New York City Health + Hospital, Queens Hospital Center, between March and April 2020. Two patients presented with cerebrovascular accident (CVA) symptoms without the main symptoms of COVID-19 infection and tested positive later, two presented with altered mental status and were found to have neurological and pulmonary compromise, and two presented with respiratory compromise and developed neurological symptoms thereafter.

A total of 5 patients had laboratory-confirmed COVID-19 infection via nasopharyngeal swab Bioreference polymerase chain reaction (PCR) test. One of the six patients with a very high clinical suspicion for COVID-19 (based on clinical, radiologic, biochemistry data), tested negative and a repeat test was not obtainable given that the patient passed away prior to sample collection.

Diagnosis of acute stroke was confirmed using brain imaging (CT head and/or MRI).

2. Case Presentation

2.1. CASE #1

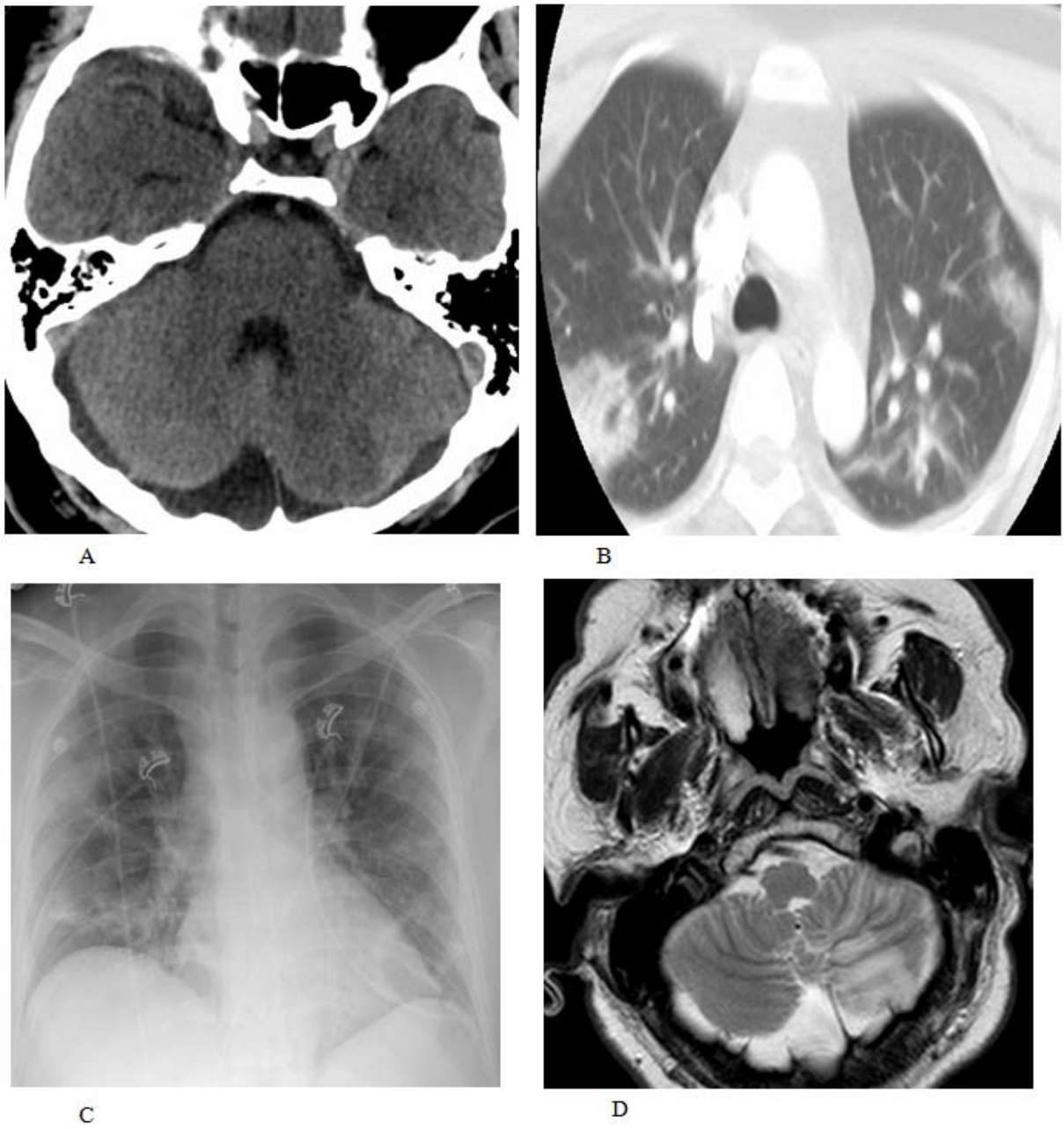

Figure 1.

A 39-year-old male with no past medical history presented to the emergency department (ED) with difficulty ambulating for one day. His initial symptoms of diaphoresis and weakness started shortly after showering. He had an unsteady gait and left-sided arm weakness. He denied falls. Review of systems was negative for visual changes, hearing or speech difficulty, sensory changes, and urinary or fecal incontinence. The patient denied fever, chills, activity or appetite changes, nasal congestion, sore throat, cough, chest pain, shortness of breath (SOB), nausea, vomiting or diarrhea, abdominal pain, myalgias, joint pain, dysuria or hematuria. He had no known allergies or history of smoking, alcohol abuse, or illegal drug use. Family history was significant for diabetes on his mother's side. Vital signs on presentation were temperature (T) of 99.8 F, normal pulse with blood pressure (BP) of 124/80, respiratory rate (RR) of 16 and oxygen saturation of 96% on room air. Neurological exam revealed mildly decreased strength (4/5) of the left upper extremity (LUE), left finger to nose dysmetria, mild left-sided ataxia, mildly abnormal left heel to shin, LUE hyperreflexia, intact sensation to light touch throughout,

positive Romberg sign to the left, and unsteady wide-based gait. Blood work was significant for neutrophilia with absolute lymphopenia but normal white blood cell (WBC) count, mild hyponatremia of 133 mEq/L, chloride of 94 mEq/L, glucose of 123 mg/dL, A1C of 6%, alkaline phosphatase of 133 IU/L, alanine aminotransferase (ALT) of 99 IU/L, aspartate aminotransferase (AST) of 78 IU/L, and direct bilirubin (DB) of 0.4 mg/dL. Lipid panel showed total cholesterol (TC) of 113 mg/dl, high- density lipoprotein (HDL) of 19 mg/dl, low-density lipoprotein of 73 mg/dl, triglycerides of 108 mg/dl and cholesterol to HDL ratio of 6. The coagulation profile and thyroid stimulating hormone (TSH) were within normal limits. Non-contrast CT of the head (NCCT) (Figure 1) demonstrated acute to subacute left cerebellar ischemic change versus an artifact and cerebellar volume loss. CT angiography (CTA) of the head and neck revealed no internal carotid artery (ICA) stenosis, but there was truncation of the proximal left posterior inferior cerebellar artery, suggesting occlusion. MRI brain without contrast confirmed CT head findings of acute to subacute cerebellar infarct of the left cerebellum. Additionally, CTA of the neck revealed multifocal rounded and linear opacities of the upper zones of the lungs bilaterally. These findings were consistent with COVID-19 infection pattern. The respiratory viral panel and influenza swab were negative; COVID-19 PCR testing was positive. The patient received aspirin 325 mg, followed by aspirin 81 mg daily, and atorvastatin 80 mg. He was also started on azithromycin and hydroxychloroquine for management of suspected COVID-19 infection. Of note, the patient was discharged before COVID-19 PCR test had resulted. A follow-up call was made to inform him of the positive COVID-19 test results, and at that time, the patient reported a new onset of respiratory symptoms.

2.2. CASE #2

A 69-year-old male with a past medical history of type 2 diabetes mellitus (DM) and hypertension presented to the ED with altered mental status and fever. At the time of admission, the patient was non-verbal secondary to depressed level of consciousness; therefore, limited history was obtained from emergency medical services (EMS), as family members were unreachable. The initial encounter with EMS revealed that the patient was hypoxemic with oxygen saturation (SpO2) in the 70s on room air, which improved to 93% on oxygen supplementation via non-rebreather mask at a flow rate of 15L/min. The patient's entire family was reported to be sick with upper respiratory symptoms, raising the possibility of COVID-19 infection. Vital signs were significant for temperature of 99F, tachycardia with heart rate (HR) of 129, BP of 154/99, and RR of 36 with SpO2 of 94% on maximal non-rebreather mask. On physical examination, the patient was found to be awake, and not oriented to person, place, or time. Lung exam was significant for respiratory distress with bilateral rales up to the middle zones of the lungs. Neurological examination was very limited as the patient was not able to participate. Venous blood gas revealed pH of 7.522, PCO2 29.4, PO2 72.1, and lactate of 2.6. Troponin T was negative and electrocardiogram (ECG) showed sinus tachycardia. Laboratory work up suggestive of COVID-19 infection included normal white blood cells (WBC), hemoglobin (Hgb), and hematocrit (Hct), neutrophilia of 83.9% (RR: 44.0 - 70.0%) with absolute lymphopenia 0.8 (1.0 - 4.9 x 10(3)/mcl).

Chest X-Ray demonstrated bilateral extensive pulmonary consolidation (Figure 2). CTA of the chest revealed patchy and ground glass opacities in both lungs, more prominent in the lower lobes. NCCT head was significant for extensive acute to subacute left middle cerebral artery (MCA) territory ischemic infarct. The COVID-19 intranasal swab was collected and reported to be negative; given the high suspicion of COVID-19 infection, a repeat test was ordered, but could not be obtained as the patient passed away prior to sample collection.

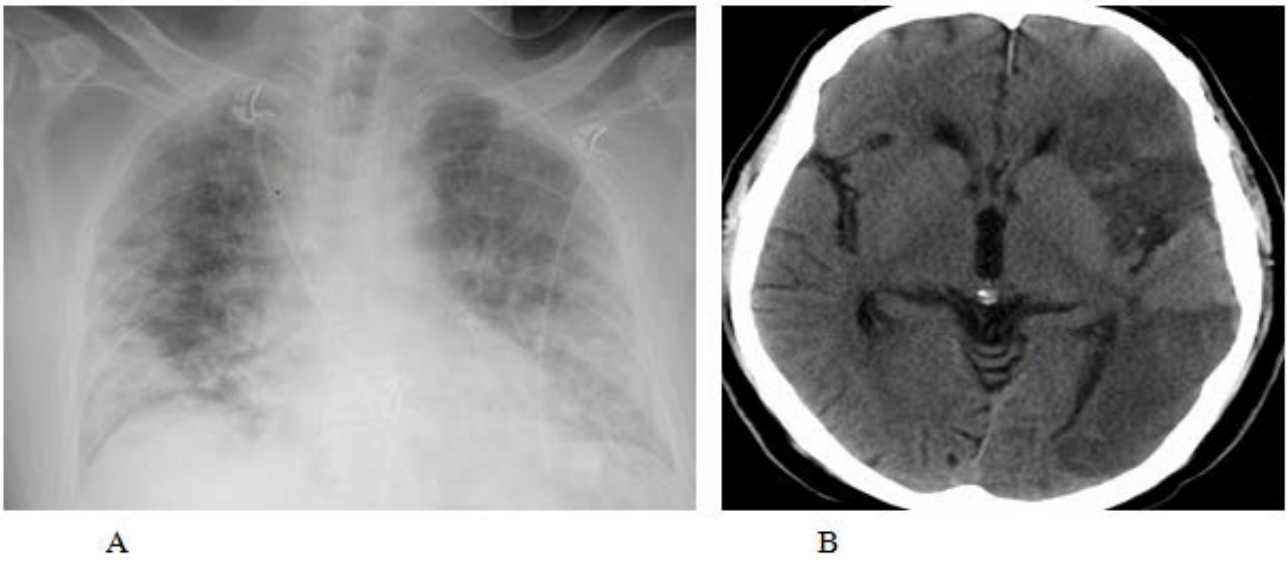

Figure 2.

2.3. CASE #3

A 49-year-old male with a past medical history of hypersensitivity lung disease, obesity, and asthma, presented with sudden onset LUE weakness and decreased sensation. Patient also reported a "seizure-like" activity the night before, and since then he had been feeling acute left arm weakness associated with decreased grip strength. Review of systems was positive for cough, fever, and shortness of breath for 2 weeks which had been progressively worsening, and negative for urinary incontinence, tongue biting, and loss of consciousness (LOC). The patient denied smoking or tobacco use. He endorsed alcohol use of approximately 6 cans of beer weekly. Family history was significant for heart disease and stroke in his mother. Vital signs on presentation were temperature of 97.8F (oral), BP of 118/80, HR of 91, RR of 18, and SpO2 97%. Neurological examination was significant for decreased LUE strength with diminished sensation to pinprick and light touch, and otherwise normal strength and sensation in all other extremities. Left pronator drift was present with increased deep tendon reflexes (DTR).

Studies were significant for presumptively positive COVID-19 testing(clinically and radiologically), mildly elevated ferritin, fibrinogen, IL-6, CRP, ESR, LDH, D-dimer, ALP, AST/ALT (negative Hepatitis B and C) and Troponin T of 0.013 (RR <0.010). Pro-BNP was found to be 4,044. ECG showed sinus rhythm with LBBB (left bundle branch block). NCCT head revealed acute to subacute right frontoparietal ischemic infarct (Figure 1). CTA of the neck/head failed to reveal any stenosis or occlusion, but showed bilateral ground glass opacities of the upper lung portions. Chest X-Ray was consistent with viral pneumonia (Figure 3). A transthoracic echocardiogram (TTE) revealed left atrium dilatation, severely decreased left ventricular (LV) ejection fraction (EF) of 10% with grade III LV diastolic dysfunction, and abnormal segmental wall motion. Akinetic apex with a small apical thrombus were reported on repeated TTE. The patient was managed with hydroxychloroquine, ceftriaxone, azithromycin, vitamin C, thiamine, zinc, aspirin 325 mg, atorvastatin 80 mg (started and held), and therapeutic low molecular weight heparin (Enoxaparin). He was discharged home on warfarin with instructions to follow up with the appropriate specialties.

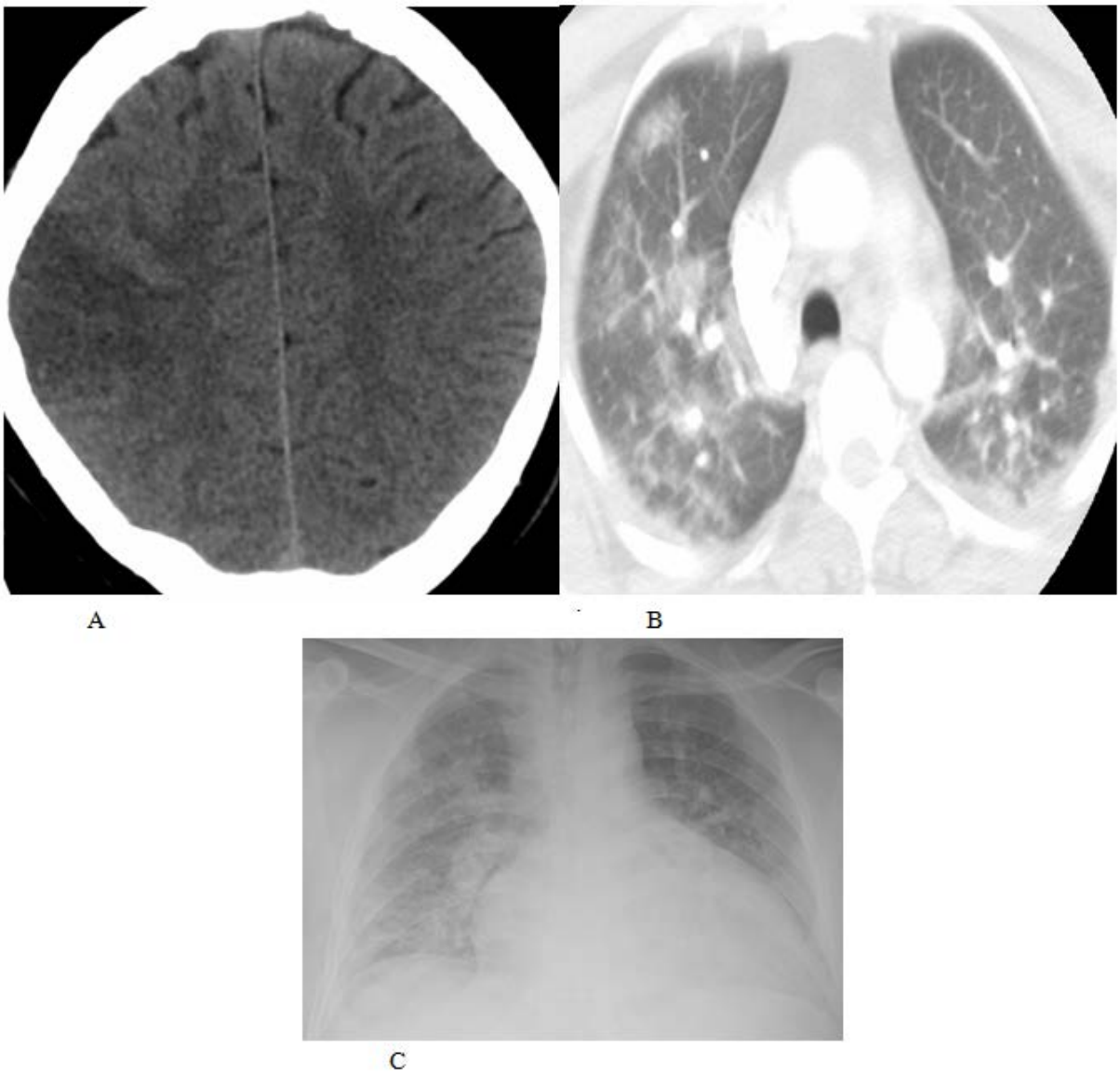

Figure 3.

2.4. CASE #4

A 56-year-old male with no known past medical history presented with altered mental status. The patient was found unconscious in the subway. Further history was not obtainable, as the patient was minimally responsive and non-verbal at time of admission. Vital signs in ED were temperature of 97F, BP of 110/67, HR of 90, RR of 24 and hypoxemia with SpO2 84%. The neurological exam was significant for mildly decreased strength of the left lower extremity as compared to the right. However, the rest of the exam was unreliable given lack of cooperation due to the patient's altered mental status. Cardiopulmonary examination was normal. Bloodwork was significant for leukocytosis (WBC 19.34) with neutrophilia, mild microcytic anemia (hemoglobin of 12.4, hematocrit of 35.7, and MCV of 78.8), sodium (Na) of 130, chloride (Cl) of 90, lactic acid 3.0, Troponin T 0.289, Pro-BNP 1584, HbA1c 6.2 and positive COVID-19 PCR testing. CRP, ESR, LDH, ferritin, IL-6 and D-dimer were found to be elevated. ECG revealed sinus rhythm with no acute ischemic changes. Chest X-Ray showed bibasilar pulmonary opacities (Figure A). NCCT head was consistent with an acute left temporal infarct (Figure 4). CTA chest with pulmonary embolism (PE) protocol was negative for a PE, but positive for bilateral lower lobe consolidation. TTE revealed LV concentric hypertrophy and reduced EF of 40% with abnormal segmental wall motion. The patient was managed with piperacillin-tazobactam, azithromycin, hydroxychloroquine, aspirin 325 mg, therapeutic Enoxaparin, and supplemental oxygen. He was discharged to a skilled nursing facility with aspirin, clopidogrel, atorvastatin, and lisinopril.

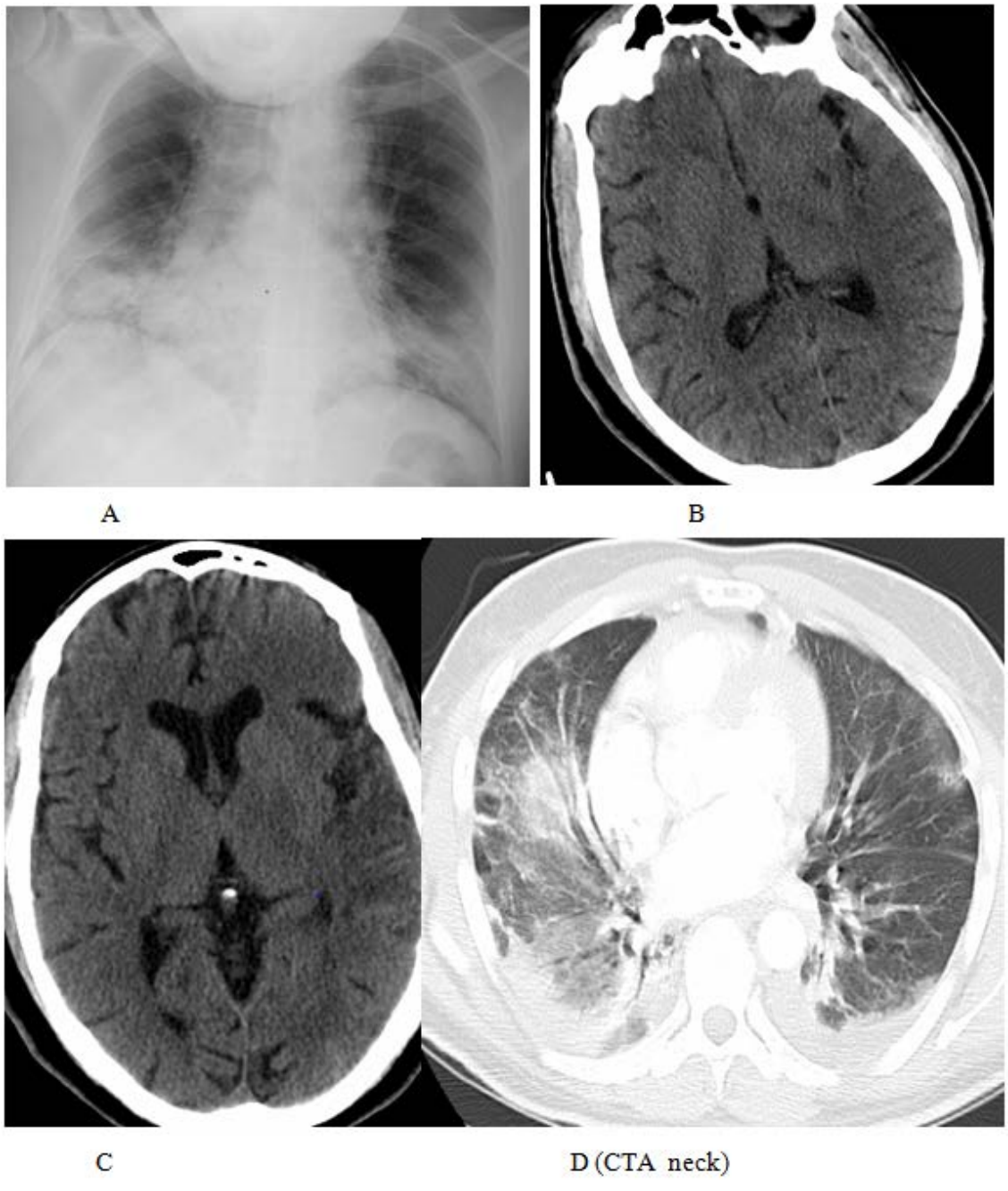

Figure 4.

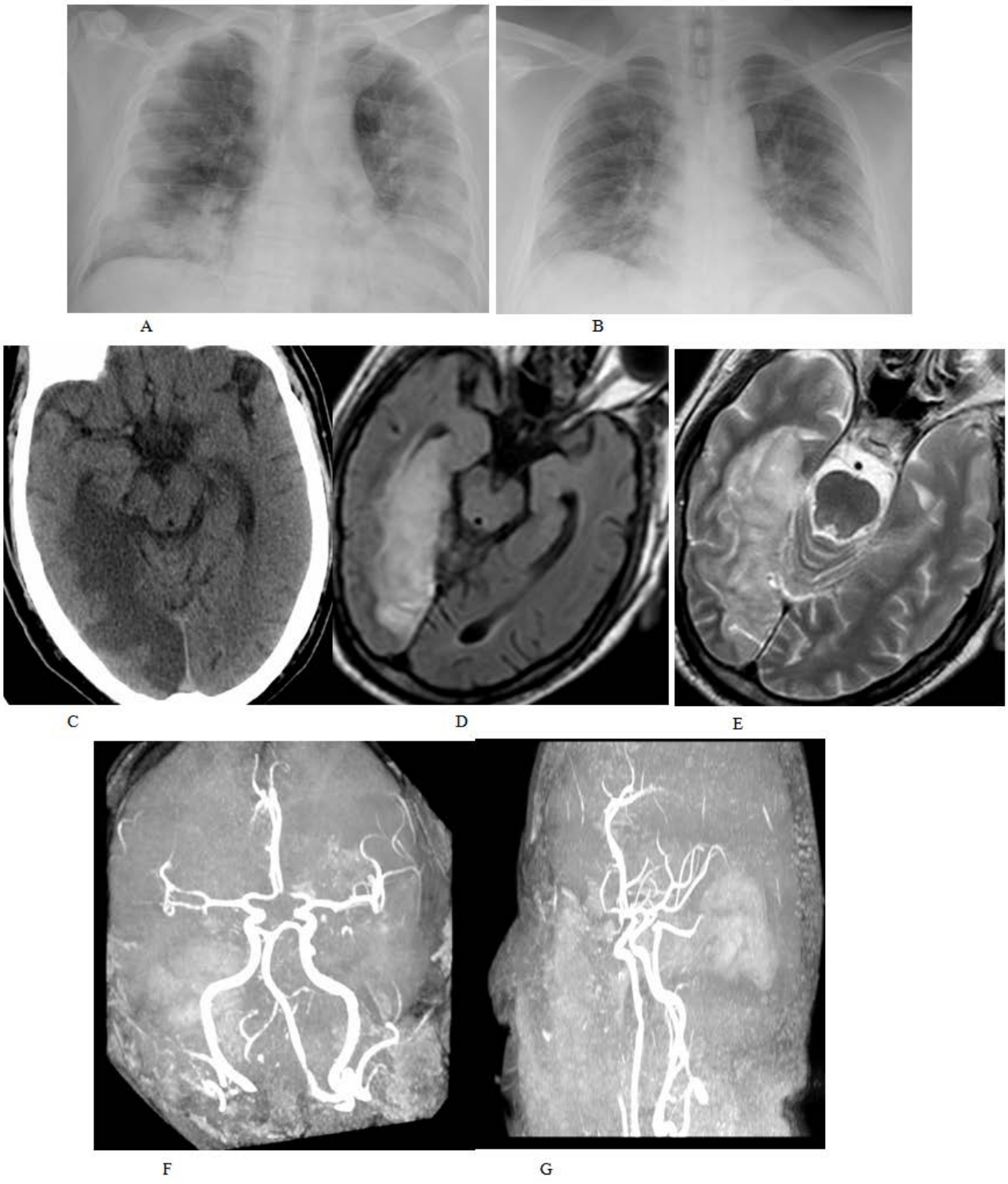

Figure 5.

2.5. CASE #5

A 51-year-old male with a past medical history of type 2 DM presented with shortness of breath. He initially presented a week prior to the ED prior and was discharged home with Tylenol. He returned due the ED with worsening shortness of breath for two days. His symptoms started approximately 15 days prior with fever, chills, dry cough, body aches, headache, back pain, joint pain, myalgias, decreased appetite and oral intake, and decreased activity. The patient denied nasal congestion, abdominal pain, nausea, vomiting, diarrhea, constipation, dysuria, and hematuria. He works as an Uber driver, and the last time he drove was 20 days ago. He denied sick contacts and recent travel. The patient smokes 5 cigarettes daily and drinks 2 cans of beer weekly. Family history was significant for DM and depression. The patient was found to be positive for COVID-19, for which he received hydroxychloroquine, ceftriaxone, azithromycin, dexamethasone, and tocilizumab. The patient was managed with supplemental oxygen via a non-rebreather mask, eventually requiring maximal titration. Enoxaparin was started for therapeutic anticoagulation. Four days later, the patient's mental status had acutely worsened. NCCT head revealed acute right posterior

cerebral artery territory infarcts, including the right occipital lobe and right posterior thalamic regions (Figure 5). MRI brain without contrast was also consistent with a right posterior cerebral territorial infarct. MRA demonstrated abrupt cut off with no flow related signal intensity seen in the distal right posterior cerebral artery extending from the distal P1/P2 segment. Marked decreased caliber of the left dominant vertebral artery at the vertebrobasilar junction was noted, which seemed to be congenital. TTE was unremarkable. Patient's condition was complicated by metabolic, respiratory, and electrolyte derangements; AKI and disseminated intravascular coagulation (DIC), all of which normalized after appropriate management. The patient was discharged to subacute rehabilitation 27 days after admission on warfarin, metoprolol, atorvastatin, and sitagliptin/metformin.

2.6. CASE #6

A 49-year-old male with a past medical history of hypertension presented with complaints of right upper extremity (RUE) weakness and slurred speech for over a week. The patient initially came to ED with shortness of breath two weeks prior. He was found to be positive for COVID-19, and he was managed with hydroxychloroquine, ceftriaxone, azithromycin, acetaminophen, and supplemental oxygen. He was placed in the ED Observation Unit and discharged due to improvement in symptoms. Shortly after discharge, the patient started to have right hand weakness and decreased grip strength. He also complained of a frontal, pounding headache and neck pain. He had slurred speech and difficulty finding words. Review of systems was negative for associated numbness, lower extremity weakness, phonophobia, photophobia, dysphagia, shortness of breath, bowel or bladder symptoms. The patient denied smoking and tobacco use, alcohol consumption, and illicit drug use. Vital signs on presentation were normal, however body mass index was 31.55 kg/m^2. On physical examination, he was alert and oriented to time, place and person. He had normal strength and sensation in upper and lower extremities bilaterally. He exhibited word-finding difficulty, but otherwise all other aspects of the neurological exam remained intact. Hoffman and Tinel's signs were positive on the right. Patient was also found to have cervical tenderness on lateral neck movement. Deep tendon reflexes were 2+ and symmetric bilaterally. Brudzinski and Kernig signs were negative for meningeal irritation. Labs were significant for mild elevation of CRP 5.6 and LDH 338. Ferritin, D-dimer, INR/PT and aPTT were within normal limits. CBC revealed a mild normocytic anemia with hemoglobin of 12.7 and hematocrit of 39.8 though during his initial presentation he was noted to have neutrophilia with lymphopenia. NCCT head revealed areas of abnormal low-attenuation with associated loss of gray/white matter differentiation and mild sulcal effacement in the left posterior frontal and parietal areas, predominantly the subcortical/deep white matter and to a lesser degree the adjacent overlying cortex (Figure 6). MRI brain without contrast showed acute to subacute infarcts in the left frontal and left parietal-occipital regions. MRA head without contrast and CTA neck with contrast failed to reveal any significant intracranial stenosis or occlusion. TTE was unremarkable. The patient was managed with aspirin 325 mg and clopidogrel 300 mg. The patient was discharged with aspirin 81 mg and clopidogrel 75 mg.

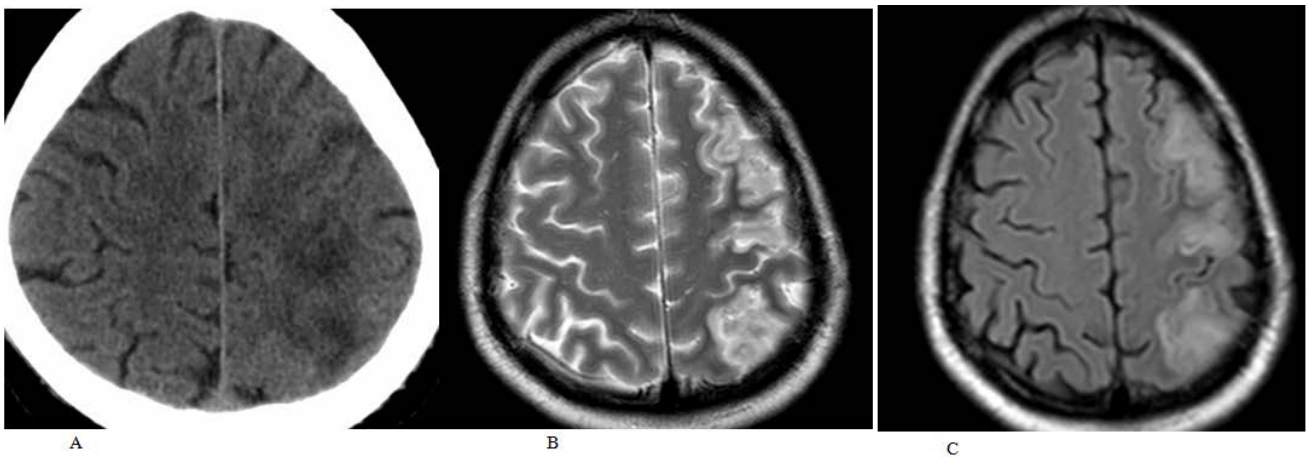

Figure 6.

3. Discussion

We report the clinical and imaging findings of six male patients who were hospitalized for acute cerebrovascular symptoms associated with COVID-19 infection or due to pulmonary compromise associated with neurological symptoms. The diagnosis was confirmed with COVID-19 PCR testing, along with brain imaging.

In this study, four of the six patients did not have known risk factors for stroke (prior history of strokes or myocardial infarctions, atherosclerotic disease on CTA/MRA head and neck, embolic risk i.e atrial fibrillation, old age, or hypercoagulable conditions apart from COVID-19-associated hypercoagulability). One patient had a few risk factors including age, type 2 diabetes and hypertension. Another patient was noted to have apical akinesia and an apical thrombus on TTE. The time between onset of stroke and COVID-19 was unclear; the first patient had difficulty walking, the second and fourth patients were brought in with altered mental status, the third patient presented with left hemiparesis and hemisensory deficits, the fifth patient developed a stroke four days after respiratory symptoms, and the sixth patient presented with right hemiparesis and dysarthria. While

most patients did not have severe respiratory symptoms, the COVID-19 PCR was found to be positive. One of the six patients had tested negative and a repeat was not obtainable given that the patient passed away prior to sample collection. All patients had varying lab parameters, although some labs were not collected in all patients. Chest imaging, when performed, showed extensive pulmonary consolidation. Notably, brain imaging revealed infarcts for all patients.

Stroke care has witnessed unprecedented challenges [13,18,19,20] and institutions have adjusted stroke algorithms to ensure physician's safety and optimization of patient care. Although neurologists have reported a fall in the number of stroke cases, one study reported 24% decreased stroke presentation to hospitals during COVID-19 [18,22,23]. This could be due to patients not presenting to a hospital and from fear of viral exposure [24]. COVID-19 may increase the risk of stroke through various mechanisms such as hypercoagulability [6], overwhelming systemic infection or 'cytokine storm' [25,26], cardio-embolism [24], or direct viral invasion of endothelial cells [25], and nervous system [6] via human ACE2 (angiotensin-converting enzyme) receptor. The inflammatory prothrombotic state is reflected in raised CRP, ferritin, D-dimer and fibrinogen as seen in our patients and reports from other researchers [29]. In our report, the predominant stroke type was ischemic with variable proposed mechanisms, including small vessel disease, large artery atherosclerosis, cardio-embolic, and embolic source of unknown significance.

Table 1. Investigations for acute stroke in COVID -19 infection

Labs ✶ (RR)	Case number					
	1	2	3	4	5	6
Age, Sex	39, M	69, M	49, M	56, M	51, M	49, M
COVID-19 (Not detected)	+	-	+	+	+	+
Repeat COVID-19	Nn	NC	Nn	Nn	Nn	Nn
Flu/RVP (Not detected)	-/-	NC	NC	-/NC	NC	NC
Hemoglobin (14.0-18.0 g/dL)	15.2	14.4	15.9	12.4	14.3	12.7
Hematocrit (42.0-52.2 %)	44.7	44.4	46.5	35.7↓	41.8	39.8
White blood cell (4.8 - 10.80 x 10(3)/mcl)	N	N	4.74 ↓	19.34 ↑↑	12.09 ↑	N
Neutrophils (44.0 - 70.0%)	86.5% ↑	83.9% ↑	N	82.9 ↑	84.3% ↑	N / 80.7↑
Absolute lymphocyte count (1.0 - 4.9 x 10(3)/mcl)	0.5 ↓	0.8 ↓	N	N	N	N / 0.96↓
Platelets (150 - 450 x 10(3)/mcl)	191	281	358	198	302	287
Sodium (136 - 145 mmol/L)	133	N	N	130	N	133
Chloride (98 - 108 mmol/L)	94	N	N	90	N	N
Potassium (3.5 - 5.1 mmol/L)	4.3	N	N	4.2	N	N / N
Glucose (74 - 110 mg/dL)	123	180	107	172	127	103/117
BUN/Creatinine (6-23/0.7-1.2 mg/dL)	N	N	N	N	N	N
Alkaline phosphatase (40 - 129 U/L)	173 ↑	NC	161 ↑	205 ↑	N	NC
Alanine aminotransferase (0 - 41 U/L)	99 ↑	NC	76 ↑	48 ↑	N	NC
Aspartate aminotransferase (5 - 40 U/L)	78 ↑	NC	72 ↑	80 ↑	58 ↑	NC
Total cholesterol (10 - 199 mg/dL)	113	NC	169	135	217 ↑	NC
High density lipoprotein (=>40 mg/dL)	19 ↓	NC	34 ↓	26 ↓	22 ↓	NC
Low density lipoprotein (<=100 mg/dL)	73	NC	105 ↑	85	UTC	NC
Triglycerides (10 - 149 mg/dL)	108	NC	154 ↑	119	483 ↑	NC
Procalcitonin (0.02 - 0.10 ng/mL)	NC	NC	NC	NC	0.58 ↑	NC
C- reactive protein (<=5 mg/L)	NC	NC	70 ↑	114.4 ↑↑	>300 ↑↑↑	5.6 ↑
ESR (0 - 10 mm/hr)	NC	NC	85 ↑	129 ↑↑	NC	NC
Ferritin (30 - 400 ng/mL)	NC	NC	963 ↑	949 ↑	980 ↑	368
Lactate dehydrogenase (135 - 225 U/L)	NC	NC	666 ↑	620 ↑	>900 ↑↑	338 ↑
IL-6 (0.0 - 15.5 pg/mL)	NC	NC	17.4 ↑	123.1 ↑↑	>180.6 ↑↑	NC
Beta 2 glycoprotein (-)	NC	NC	NC	NC	+	NC
Cardiolipin Antibody (-)	NC	NC	NC	NC	+	NC
Fibrinogen (200 - 393 mg/dL)	NC	NC	719 ↑	N	968 ↑↑	NC
D-Dimer (0 - 243 ng/mL) ✶ ✶	NC	NC	720 ↑	14981 ↑↑↑	2061 ↑↑	N
Protime/INR 10.0 - 13.0 seconds)	N	N	N	N	N	N
Activated partial thromboplastin time (25.1 - 36.5 seconds)	N	NC	N	N	N	N
HbA1c (25.1 - 36.5 seconds)	6	NC	N	6.2	8.6	5.6
Glomerular filtration rate (>=60 ml/min/1.73m2)	>60	>60	>60	>60	>60	>60

(+) positive, (-) negative, (Nn) None, (NC) not collected, (N) normal, (RR) reference ranges (aka: normal ranges), (✶) all lab values according to Northwell Laboratories, (✶ ✶) cut off value for NYC Health + Hospitals-Queens is age of the patient times 5.

It has been suggested that COVID-19 infection can be a cause of stroke as demonstrated in a study [6] by Ling et al. from China. Of patients with confirmed COVID-19 infection, 36.4% had neurological manifestations with severe infections associated with older age, comorbidities, and few typical symptoms of COVID-19. Stroke symptoms occurred at a median of 10 days after symptom onset and the most common neurological symptoms were dizziness (16.8%) and headache (13.1%); acute cerebrovascular disease (5.7%) and impaired consciousness (14.8%) were particularly seen with severe infections. Another study [30] found COVID-19 to be an independent risk factor for imaging confirmed acute ischemic stroke. Most commonly associated comorbidities reported were hypertension (17.1%), cardiovascular disease (CVD) (16.4%), and diabetes (9.7%) [31,32].

The study from the US on hospitalized patients with COVID-19 [12] revealed that overall patients presented with severe symptoms and manifestations which required ICU care; this finding correlated with those from the Chinese study. Moreover, the hospital adopted a protocol of therapeutic anticoagulation in patients with high D-dimer levels, which could have led to lower thrombo-embolic complications.

Some prognostic markers of poor survival reported in literature include higher fibrin degradation products, prolonged prothrombin time, activated partial thromboplastin time and platelets, and inflammatory cytokine which increases risk of cerebrovascular disease [3,33,34,35,36]. This trend was also seen in findings from other researchers.

In conclusion, acute cerebrovascular symptoms can complicate the course of COVID-19 infection with or without vascular risk factors. In order to achieve optimal patient safety and outcomes, it is crucial to increase awareness about this novel crisis in regards to stroke care. Screening should be fast-tracked with rapid viral testing [37], and stroke management should be commenced simultaneously as delay in diagnosis may contribute to further deterioration in condition. In light of risks of viral transmission, understaffing, and other resource limitations, patients should be evaluated promptly for acute neurologic changes to reduce morbidity and mortality. Longitudinal studies would be valuable for determining if previous infection with COVID-19 contributes to future risk of cerebrovascular events, and to what extent severity of the preceding viral infection plays a role. Detection of risk factors for stroke in young patients diagnosed with COVID-19 will ultimately lead to early prevention and treatment strategies, resulting in better outcomes. A hypercoagulable workup in this unique population with no risk factors is warranted and, importantly, can reveal the mechanism for stroke and possibly other thrombotic events [38]. Lastly, prospective studies are needed to explore the neurological manifestations, long-term sequelae, and appropriate management of acute cerebrovascular symptoms with COVID-19 infection.

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