Stroke in a young COVID -19 patient

Kulothungan Gunasekaran MD[1], Kwesi Amoah MD[1], Venkat Rajasurya MD FCCP [2], Michael G Buscher DO[1]

[1] Division of Pulmonary and Critical Care, Yale-New Haven Health Bridgeport Hospital, Bridgeport, CT 06610.

[2] Department of Pulmonary and Critical Care, Novant Health System, Winston-Salem, NC 27103.

Correspondence to:

Kulothungan Gunasekaran, MD

Division of Pulmonary Critical Care Medicine

Yale-New Haven Health Bridgeport Hospital

267 Grant Street

Bridgeport, CT 06605, USA

Phone: 203-384-5009

Fax: 203-330-7498

Email: stankuloth@gmail.com.

The authors express no conflict of interest.

Stroke in a young COVID -19 patient

Learning point for Clinicians: Recent autopsy and pathology reports suggest that COVID patients are at high risk for thromboembolic complications. The exact pathophysiology behind the cerebrovascular accidents is still not clear. The physician should be wary of the fact that COVID-19 patients can develop cerebrovascular accidents, and we recommend close monitoring of neurologic status.

A 40-year-old female without past medical history presented to our emergency department with complaints of cough, fever, and shortness of breath. Blood pressure was 104/74 mmHg; temperature was 98.6°F, pulse 110 beats/min, respiratory rate 40 breaths/min, and oxygen saturation 88% on a non-rebreather mask. Her COVID PCR (Cepheid; Sunnyvale, California, USA) was positive and the chest X-ray showed diffuse, patchy, ground-glass attenuation of the lungs. Because of ongoing hypoxia along with respiratory distress, she was intubated and admitted to the intensive care unit. White cell count was 14,000 cells/mm^3 (91% neutrophils) with a platelet count of 303x1000/µL. Her D-Dimer was 28.07 ng/ml, fibrinogen was 860 mg/dL, ferritin was 3079 ng/ml, and serum creatinine was 1.43 mg/dL. She was also found to be in diabetic ketoacidosis (DKA) with a blood glucose level of 876 mg/dL and an anion gap of 26. She was treated with a course of hydroxychloroquine, tocilizumab, and methylprednisolone. Her DKA was successfully treated with an appropriate regimen, including insulin drip. She was initially started on midazolam for sedation, and later transitioned to propofol and dexmedetomidine. On day 7 of intubation she became unresponsive, and physical examination showed sluggish pupils and absent corneal responses – even after holding sedation. She also developed polyuria, with almost 3 liters of urine output and the development of hypernatremia (sodium 155 mmol/L). Computed

tomography of the head showed a large right middle cerebral artery (MCA) territory infarct with extensive mass effect, including midline shift and downward herniation. Her diabetes insipidus was thought to be central in nature because of the massive stroke. Her pro-coagulant work-up, including lupus anticoagulant, was negative. The bedside echocardiogram did not reveal any septal defects. There was no neurosurgical intervention given her poor prognosis. After a family discussion, the patient was provided with comfort measures.

Although COVID-19 has been observed to affect the respiratory system primarily, neurological involvement has already been reported. Recently Dr.Oxley and colleagues described a case series of large-vessel strokes as an initial presentation in COVID-19 patients younger than 50 years of age.[1] Our case is similar, except for the fact that our patient developed a stroke during hospitalization. The other neurologic manifestations reported in the literature include headache, dizziness, ataxia, alteration of sensorium, and encephalitis. Cerebrovascular events had been described in 5.7% (5/76) and 5.88% (13/221) of patients in two case series from Wuhan, China.[2] Human endothelial cells express ACE2, CD147, sialic acid receptor, and transmembrane protease serine 2 , which increases the affinity of the co-factors bound by the COVID-19 virus to access host cells. All systemic manifestations, including thrombotic complications, are thought to be caused by direct endothelial dysfunction in COVID-19 infected patients.[3, 4] Furthermore, the risk of a large vessel and cardiogenic stroke in individuals with respiratory tract infections without classic stroke risk factors have already been described in the literature.[5] As we do not yet know the exact mechanism of stroke in this patient population, we recommend close neurologic monitoring of this patient population and further investigations to elucidate the association of thrombotic complications in COVID-19 infected patients.

References:

1. Oxley T, Mocco J, Majidi S, Kellner C, Shoirah H, Singh I et al. Large-Vessel Stroke as a Presenting Feature of Covid-19 in the Young. New England Journal of Medicine. 2020;382(20):e60.

2. Lahiri D, Ardila A. COVID-19 Pandemic: A Neurological Perspective. Cureus. 2020; 12(4): e7889.

3. Chen Z, Mi L, Xu J, Yu J, Wang X, Jiang J et al. Function of HAb18G/CD147 in Invasion of Host Cells by Severe Acute Respiratory Syndrome Coronavirus. The Journal of Infectious Diseases. 2005;191(5):755-760.

4. Varga Z, Flammer A, Steiger P, Haberecker M, Andermatt R, Zinkernagel A et al. Endothelial cell infection and endotheliitis in COVID-19. The Lancet.2020;395(10234):1417-1418.

5. Paganini-Hill A, Lozano E, Fischberg G, Perez Barreto M, Rajamani K, Ameriso S et al. Infection and Risk of Ischemic Stroke. Stroke. 2003;34(2):452-457.

Figure Legend:

Figure 1: Computed Tomography of the brain showing large MCA territory infarct with extensive mass effect including midline shift (A) axial view and (B) coronal view.

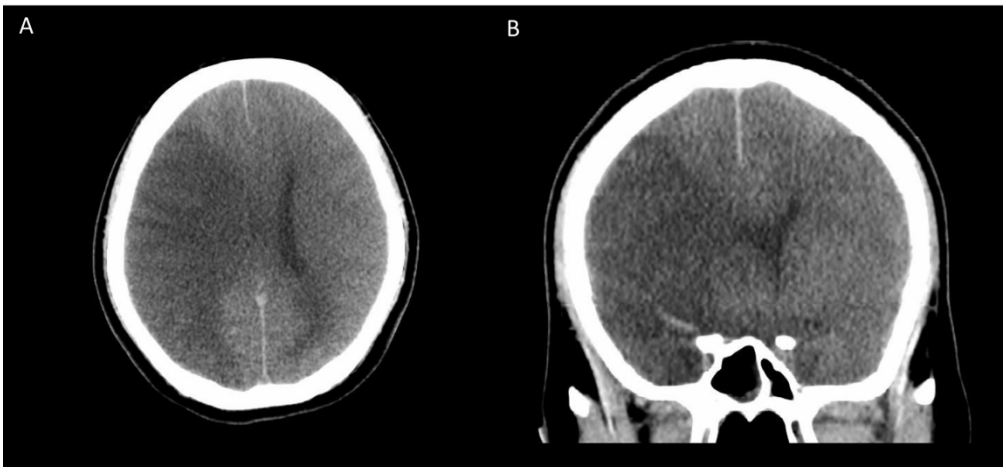

Figure 1: Computed Tomography of the brain showing large MCA territory infarct with extensive mass effect including midline shift (A) axial view and (B) coronal view.

249x115mm (300 x 300 DPI)