CASE REPORT

COVID-19 MAY INCREASE THE RISK OF ISCHEMIC STROKE: A CASE REPORT

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ABSTRACT

A 72-year-old patient with well-controlled HT and a risk factor such as a coronary stent 20 years ago. In the 3rd week of the Corona virus infection, he had a stroke in the unusual localised micro-embolic watershed nature. However, no cardioembolic or cerebrovascular risk factor was detected in the etiological investigation of stroke, and there were no cerebrovascular complaints until this event. In the literature, there is a relationship between many viruses, especially varicella, and stroke in embolic nature. This case suggests that COVID-19 infection may also cause stroke or increase risk of stroke.

Keywords: Covid-19, coronavirus, ischemic stroke, microembolism.

INTRODUCTION

As known viruses can cause temporary ischemic attacks or stroke (1). SARS-CoV-2 (2019-nCoV) highly contagious and mortal pathogenic coronavirus and the growing evidence shows that coronaviruses can also invade the central nervous system and causes neurological diseases. In the presented case, he had an unusual localized embolic stroke in the 3rd week of corona virus infection, although he had no cardioembolic risk factor and no cerebrovascular complaints (TIA, amarosis fugax, stroke etc.) until this event.

CASE REPORT

A 72-year-old male patient has a history of coronary stent in 2000 and HT for 20 years. He uses metoprolol 25 mg 1x1 po, furosemide 40mg 1x1 po and clopidogrel 75 mg 1x1 po. The patient who had no previous respiratory complaints (asthma, COPD, etc.) applied to the hospital a few days after coming from France on February 21, due to cough and blood in sputum. In the Torax CT taken here, a mass lesion was detected in the right lower lobe of the lung (Figure 1a and 1b), levofoxacine 500 mg 1x1 and clarithromysyn 500
A mass lesion extending to different levels of the lower right lobe of the lung (Ia and Ib) in Thoracic CT taken on February 22. mg 2x1 were started, and biopsy was recommended due to possible cancer risk.

When fever and respiratory distress increased, thorax CT was requested again on March 19. In that thorax CT new glass view style lesions was added in the left (Figure 2a) and right (Figure 2b) upper lung lobe and CRP: 23.6 (0-0.5 mg/dl), sedimentation: 89 (0-15mm), amylase: 182 (20-160U/l), WBC: 13.9 (When 3.5-10 K/ul), lymphosite 7.7% (15-50%) in laboratuary results. Nature of new lesions and disease progression rate was resemble of Coronavirus disease-2019 (COVID-19). Then RNA PCR was performed for coronavirus and it result as positive.

Hydroxichloroquine 200 mg 2x1 po+moxifloxasine 400 mg 1x1 IV theraphy+meropenem 1 gr 2x1 IV therapy started for coronavirus pneumonia. Later, on March 20, there was a short loss of consciousness while performing a tru-cat lung biopsy for the mass lesion that in right lower lung lobe. Glucose: 104 mg/dl, ECG: 87 beats/minute, sinus rhythm and BP: 130/85 mmHg were observed at the time of loss of consciousness. After awakening, weakness was detected in his left arm and leg of the patinet, and the right capsula interna, splenium, anterior cerebral artey (ACA) and middle cerebral artey (MCA) and lenticlostriate arteries (LSA) and MCA (Figure 3a) and MCA and posterior cerebral artey (PCA) (Figure 3b) watershead infarct (WS) area was detected in the diffusion weighted MRI (WS) area was detected in the diffusion weighted MRI. In the first neurological evaluation the motor examination revealed 3/5 strength in the upper left extremity and 2/5 strength in the lower left extremity. In Torax CT taken on March 25, the lesion in the right lower lobe (Figure 4a) and right and left upper lobe (Figure 4b) lobe were regressed. Heart rate was recorded 100 beats/minute with sinus rhythm by 12 lead ECG and 24-hours 12 lead-ECG holter was in normal range. Echocardiographi was normal, carotis-vertebral doppler USG flow was sufficient, and there was not stenosis. Then carotis and serebral MR angiogram performed and it was in normal range too. Glucose: 92mg/dl, LDL: 115.9 mg/dl, VLDL: 31.49 mg/dl, TG: 157 mg/dl, HDL: 24 mg/dl. COVID PCR turn to negative on March 28. And he had had very mild paresis in right side (-5/5 and 4/5) in last neuroglical examination. Of note, informed consent was signed by the patient for this report.

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In diffusion weighted MRI, right capsula interna and splenium( long white arrow), anterior cerebral artey (ACA) and middle cerebral artey(MCA) (short white arrow), lenticlostriate arteries (LSA) and MCA (blac arrow) (IIIa), MCA and posterior cerebral artey (PCA) (IIIb) watershead infarct area was detected. Reduction areas seen in apparent diffusion coefficient (ADC) (IIIc).
DISCUSSION AND CONCLUSION

Although there were some atherosclerotic risk factors (HT, coronary stent) in this case, the baseline cardiac rhythm was sinus, echocardiographic evaluation and cerebrovascular evaluation (Doppler USG and MR angiogram) was normal and there was not any cerebral ischemic events history before. In addition, the type and distribution of the infarct had unusual pattern as multiple cortical watershed (CWS) infarcts and deep watershed area such as the capsular interna. While both embolic and hemodynamic mechanisms can cause CWS (2), hemodynamic impairment (HDI) mostly cause deep watershed (DWS) infarction, and this is most common in the rosary-like pattern in the centrum-semioval (2,3). These case made us think that the underlying coronavirus infection may be a primary factor or secondary facilitator in stroke. As it is known, stroke is one of the most common causes of mortality and morbidity in the World (4).

While diabetes, arteriosclerotic disease and hypertension are the most common causes of stroke, viruses can also cause temporary ischemic attacks and stroke (1). We are an important cause of stroke, especially in severe carotid disease (5), and both microembolism (ME) and HDI are considered to have a role in that infarcts (6).

SARS-CoV-2 (2019-nCoV) highly contagious and mortal pathogenic coronavirus, which appeared in Wuhan in December 2019. The growing evidence for this shows that coronaviruses are not always limited to the respiratory tract and can also invade the central nervous system that causes neurological diseases. In some patients with this COVID-19, neurological symptoms such as headache, nausea, anosmia and vomiting have been observed besides respiratory diseases (7). SARS-CoV-2 (2019-nCoV) has many similarities with viruses such as severe acute respiratory syndrome coronavirus (SARS-CoV) and Middle East respiratory syndrome coronavirus (MERS-CoV). SARS-CoV infection has also been reported to be severely infected in the brain stem from both patients and experimental animals in the brain (7).

When respiratory viruses can cause neurological damage by creating dysfunction of the cardiorespiratory center in the brain stem (8). Some coronaviruses have also been shown to spread in the lungs and lower respiratory tract to the medullary cardiovascular center via a mechanoreceptors and chemoreceptors synapse-associated route (7).

The old coronavirus type SARS - CoV enters human host cells via angiotensin converting enzyme 2(ACE2), which is expressed in the human airway epithelium, lung parenchyma, vascularendothel, kidney cells and small intestine cells. It is believed that a similar receptor is used in SARS - CoV2 (9). There are some finding as an increase of 3.5% for cardiac arrest and 2.4% for hemorrhagic stroke in MERS virus infection (10).

Among the most well-known neuropathological viruses, human respiratory syncytial virus (hRSV), influenza virus (IV), coronavirus (CoV) and human metapneumovirus (hMPV) can cause febrile or non-febrile seizures, status epilepticus, encephalopathy and encephalitis. VZV, CMV and HIV has also been associated with stroke. VZV is the only virus with definitive evidence of ischemia or infarction in the cerebral arteries (11). While HIV and CMV have been found to infect smooth muscle cells in the coronary or renal arteries, it has been suggested that they may also be related to ischemia of the cerebral arteries (11). Mao et al. found that among severe COVID-19 patients, about 88% (78/88) had neurological symptoms such as acute cerebrovascular diseases and impaired consciousness in a 214-patient cohort (12).

As a result, a patient with advanced age, well-controlled HT and a risk factor such as coronary stent 20 years ago. But we could not find any cardiembolic or cerebrovascular risk factor in work up and he had not any cerebrovascular complaints until this event. Herein, this case had unusual localized watershed stroke in the 3rd week of corona virus infection. And the lesions were mostly microembolic CWS type more than...
rosary-like DWS type, that more significant for HDI. This suggests that COVID-19 infection can cause or predispose to stroke especially in CWS type.

REFERENCES


Ethics
Informed Consent: Informed consent form was signed by the patient.

Copyright Transfer Form: Copyright Transfer Form was signed by the author.

Peer-review: Internally peer-reviewed.


Conflict of Interest: No conflict of interest was declared by the author.

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