Case Report

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TANAFFOS

F-18 FDG PET/CT as a One-Stop Shop Imaging Modality for Assessment of Neurologic and Pulmonary Manifestations of COVID-19

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Neurologic manifestations are now being increasingly encountered in patients who are admitted for respiratory symptoms of COVID-19. A 67-year-old male with a recent history of Wernicke's aphasia was referred to the nuclear medicine department for risk stratification of malignancy in pulmonary nodule by 18F-FDG PET-CT scan. PET-CT revealed decreased metabolic activity in the left temporoparietal lobe of the brain consistent with recent CVA and excluded malignancy in the pulmonary nodule with low-grade metabolic activity. Incidentally noted, new bilateral pulmonary hypermetabolic ground glass opacities rising suspicious for covid-19 infection which was confirmed by PCR of nasopharyngeal mucosa sample. These findings highlight the value of 18F-FDG PET-CT in the assessment of COVID-19 infection especially in non-pulmonary presentations like early neurologic manifestation.

Keywords: COVID-19; Wernicke's aphasia; PET-CT scan; PCR

INTRODUCTION

Neurologic manifestations are now being increasingly encountered in patients who are admitted for respiratory symptoms of coronavirus infectious disease-2019 (COVID-19) (1). However, debates are still present for any causal relationship of this issue. Neurological deficits more commonly occur late in the course of the disease in old patients with severe respiratory symptoms and competing risk factors for ischemic events. All of this evidence implies the contributory role of proinflammatory condition and the resultant coagulopathy as well as the additive role of underlying risk factors rather than the direct effect of coronavirus on the cerebral vessels or parenchyma (2).

Here we describe a COVID-19-positive patient without severe respiratory disease and any underlying vasculopathy-related risk factors in whom neurologic manifestation preceded the respiratory symptoms.

CASE SUMMARIES

A 67-year-old non-smoker male patient presented to Masih Daneshvari Hospital, Tehran, Iran with a 3-day history of Wernicke's aphasia followed by fever, cough, myalgia, and. Brain magnetic resonance imaging (MRI) was performed and revealed a low signal area in T1 and a high signal in T2 in the left parietal lobe (Figure 1).

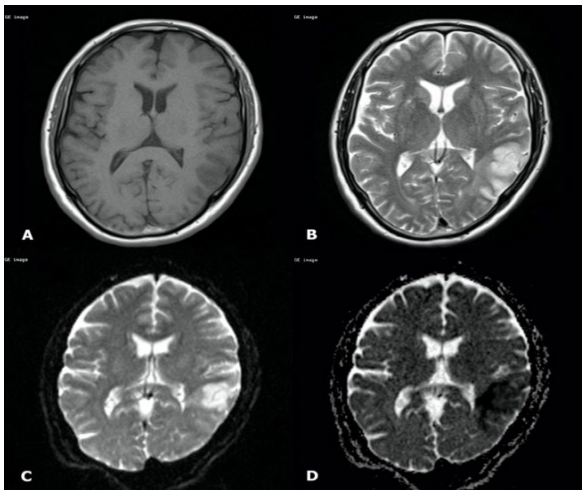

Figure 1. MR Imaging Findings of our 67 years old patient with cerebrovascular accident (CVA). (A) Unenhanced T1-weighted MR image shows an area of low signal intensity in the left parietal lobe. (B) T2-weighted MR image shows an area of high signal intensity in the left parietal lobe. (C) Diffusion-weighted MR image shows an area of high signal intensity in the left parietal lobe. (D) ADC map shows an area of low signal intensity in the left parietal lobe.

Regarding the persistent cough and constitutional symptoms, the patient underwent a chest computed tomography (CT) scan on 6 May 2020 which revealed a 15 mm parenchymal nodule in the left upper lobe (LUL) in addition to a cavitary nodule and scar in the right upper lobe representative of old tuberculosis (Figure 2). Therefore, on 17 May 2020, the patient was referred to the nuclear medicine department for a positron emission tomography-CT (PET-CT) scan with 18F-fluorodeoxyglucose (18F-FDG), as a non-invasive highly sensitive test for further risk stratification of potential malignancy of the parenchymal nodule. The 18F-FDG PET/CT study demonstrated a hypodense lesion with decreased metabolic activity in the left temporoparietal lobe (Figure 3) consistent with the patient's recent history of cerebrovascular accident (CVA) presenting with Wernicke's aphasia. In addition, the scan demonstrated a low-grade metabolic activity in the mentioned pulmonary nodule which excluded malignancy with a negative predictive value of 97%.

Incidentally noted, there were multifocal hypermetabolic ground glass opacities throughout both lung fields (Figure 2) rising suspicious for SARS-COVID 19 infection which was confirmed by real-time polymerase chain reaction of the nasopharyngeal mucosa sample.

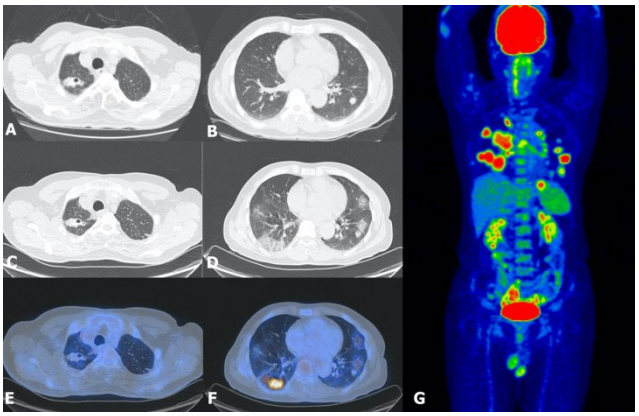

Figure 2. (A and B) A cavitary nodule and scar in the right upper lobe representative of old tuberculosis in addition to a 15mm parenchymal nodule in LUL in the initial chest CT scan, 6 May 2020.
(C, D, E, F, and G) FDG PET/CT shows low-grade metabolic activity in the mentioned pulmonary nodule in addition to multifocal hypermetabolic ground glass opacities throughout both lung fields, 17 May 2020

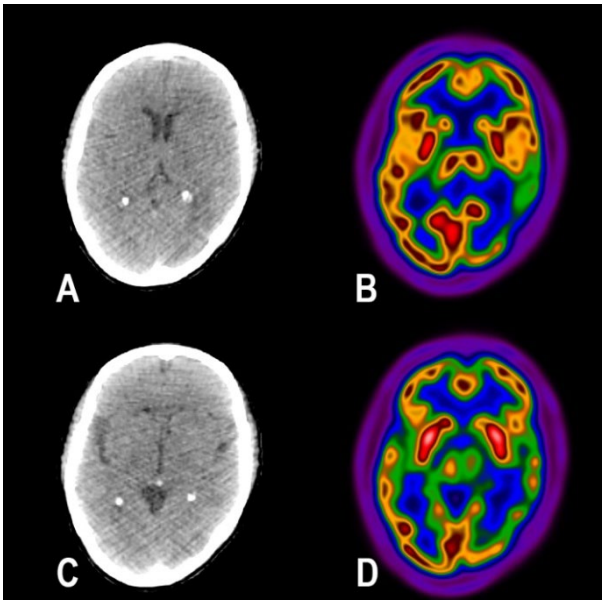

Figure 3. FDG PET/CT revealed decreased metabolic activity in the left temporoparietal lobe of the brain.

Regarding the patient's progressive shortness of breath, he was then hospitalized and underwent supportive care along with antiviral therapy [Kaletra®: lopinavir/ritonavir]. There was no need for intubation or respiratory support during his 4-day course of hospitalization.

DISCUSSION

While fever, cough, and dyspnea are the main symptoms of COVID-19, non-respiratory presentations have been increasingly recognized, including neurological manifestations (3–5).

Limited comprehensive reports are available on PCR-positive COVID-19 patients who developed neurological symptoms during the course of the disease and autopsy data are scares. The most important issues that should be addressed in this context is the casual relation versus a simple coincidence as well as the direct versus indirect role of coronavirus in developing neurological symptom. It seems that various mechanisms are contributed to this setting. (1) The frequency of neurologic deficit has been reported to be 36% in COVID-19 patients and is much more common in severe cases accompanied by respiratory failure and poor prognosis. (6) Therefore, it is assumed that individuals with severe COVID-19 may experience a coagulopathy status as a result of a surge of proinflammatory cytokines, leading to cerebrovascular and cardiovascular ischemic events. This is supported by the elevated levels of C-reactive protein (CRP) and D-Dimer observed in such cases. However, this current case did not develop severe respiratory symptoms and a drop in blood oxygen saturation nor he met the criteria for intensive care admission or mechanical respiratory support having normal CRP at the time of the neurologic symptom onset. Thus, a direct role of coronavirus may be presumed to activate endothelial cells and arterial occlusion.

According to current literature, people with vasculopathy-related risk factors are more likely to experience ischemic stroke at an older age. Beyrouti et al. (7) reported 6 cases of COVID-positive patients with coincident respiratory and neurologic symptoms. All of the patients had underlying risk factors such as hypertension, hyperlipidemia, previous history of stroke, and atrial fibrillation. However, this evidence does not fully support the causal relationship between coronavirus and ischemic stroke.

Zirpe et al. (8) reported multiple mechanisms that may play a role in the pathophysiology of neurologic symptoms in COVID-19 patients. Some of these hypothesized mechanisms are the transfer across synapses from the infected neurons, entry via the olfactory nerve, transfer via vascular endothelium, or white cells traversing the blood-brain barrier. However, in this current case, no competing risk factors for large-vessel stroke were identified. This may advocate an independent role for coronavirus infection to develop ischemic stroke but indicates the need to be validated by large-scale studies with multivariate regression analysis.

In this current case, neurologic manifestation developed early in the course of the disease even before the onset of respiratory symptoms. Such a pattern of temporality may imply the direct effect of coronavirus on endothelial cell dysfunction in the great vessel as the underlying pathophysiologic mechanism of ischemic stroke. The presence of myalgia as a common constitutional symptom of coronavirus infection before the onset of neurologic manifestation decreases the possibility of nosocomial infection of COVID-19 in this current case.

Our findings about our patient support the direct effect of coronavirus on CNS and an independent role in developing ischemic stroke in addition to other contributing factors such as the excess release of proinflammatory cytokines and coagulopathy state occurring in a severe systemic inflammatory condition. In this regard, routine prophylactic anticoagulation in COVID-19 patients should be considered with caution, and selecting the best approach needs further investigation. Berlit and colleagues (9) found that the nervous system is often indirectly, but sometimes directly, involved. This is important for the diagnostic and therapeutic approaches, and also the prognosis of COVID-19 patients especially in pulmonary-dominated intensive care situations (9).

Our findings suggest that F-18 FDG PET/CT may have a role in the assessment of both neurologic and pulmonary manifestations of COVID-19 as a one-stop shop whole body imaging modality.

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