Covid-19 associated acute encephalopathy features on computed tomography: A case report

Ayla Turkar 1, Hediye Pınar Gunbey 2

1 University of Health Sciences, Istanbul Süreyyapasa Chest Diseases and Thoracic Surgery Training and Research Hospital, Department of Radiology, Istanbul, Turkey
2 University of Health Sciences, Kartal Lutfi Kırdar Training and Research Hospital, Department of Radiology, Istanbul, Turkey

ORCID ID of the author(s)
AT: 0000-0002-6484-0039
HPG: 0000-0001-5288-5590

Abstract
More than 83,000,000 individuals have encountered Covid-19 disease, caused by novel severe acute respiratory syndrome - Coronavirus-2 (SARS-CoV-2). Besides systemic and respiratory symptoms, neurologic involvement has begun to be discussed recently. As severe involvement, only a few meningoencephalitis and encephalopathy cases were reported in the literature. We herein present a 65-year-old male Covid-19 patient with signs of hemorrhagic encephalopathy, along with literature review.

Keywords: Covid, Brain, Imaging, CT

Introduction
According to updated data, more than 83,000,000 individuals were infected with the novel coronavirus called severe acute respiratory syndrome - Coronavirus-2 (SARS-CoV-2), and nearly 360,000 deaths were reported. Patients mostly present with fever and respiratory symptoms like cough, shortness of breath, and respiratory distress, and less often, with diarrhea. These symptoms are now well-known by clinicians and the public, due to dire consequences of this disease [1].

In addition to systemic and respiratory symptoms, neurologic involvement of SARS-CoV-2, like other coronaviruses (CoV), is recently discussed with increasing frequency in the literature [2-5]. The neuroinvasive potential of SARS-CoV-2 and neurologic symptoms such as headache, nausea, and vomiting were reported by Li et al [6]. Today there are five reported meningoencephalitis and encephalopathy cases in the literature, which show severe and exceedingly rare central nervous system involvement of SARS-CoV-2 [7-11].

We herein report acute encephalopathy in a patient who developed somnolence and blackout, incompatible with his respiratory distress and expected progress of Covid-19 disease.
Case presentation

A 65-year-old male patient presented to our hospital with throat ache, dyspnea, fever, and weakness, with a history of praying at a mosque two weeks ago and umrah visit the previous month. He was pre-diagnosed with Covid-19 and hospitalized. The general condition of the patient began to deteriorate as he was transferred to the intensive care unit on the second day, requiring mechanical ventilation. After the definitive diagnosis of Covid-19 infection with detection of SARS-CoV-2 viral nucleic acid in PCR test from nasopharyngeal swab specimen, treatment protocol was followed, and lung findings regressed. However, although the patient did not receive sedatives for three days, his neurological manifestations progressed, and he became comatose. Blood tests were not significant for the progression of disease. Cerebrospinal fluid analysis was negative for SARS-CoV-2.

On the third day of coma, a computed tomography (CT) was performed, which showed generalized brain edema with patchy hypodense areas in bilateral temporal lobes and anterior cingulate gyrus region, which led us to the diagnosis of encephalopathy. Focal hypodense changes in the thalamus were most probably compatible with necrotizing and hemorrhagic areas (Figure 1).

![Figure 1: Head CT demonstrated effacement of cerebral sulci without midline shift (A). Focal hypodense changes in bilateral thalamus suggesting hemorrhagic areas (arrows) (B, C). Focal hypodense areas in bilateral temporal lobe (open arrows) and anterior cingulate gyrus areas (arrow) suggesting parenchymal involvement of encephalopathy (D).]

We do not have a Magnetic Resonance Imaging (MRI) device in our hospital to detect hemorrhagic areas and verify the diagnosis of the disease. Due to the probability of the requirement for further neurologic treatment, the patient was referred from our chest diseases hospital to a university hospital. He had a cardiac arrest a day later, did not respond to treatment and died. The patient consent form for this case report was obtained from his family.

Discussion

A growing evidence shows neurotropism is one of common features of CoVs. The novel SARS-CoV-2, with high homology to SARS-CoV, may invade the central nervous system (CNS) to induce neuronal injury. Although the exact route of CoVs is still not reported, there are two proposed routes to CNS migration. One of them is hematogenous dissemination of the virus with infected leukocytes through compromised endothelial cells of the blood-brain barrier. The other strongly suggested mechanism is the peripheral invasion of the virus by anosmia and agueisia in Covid-19 patients. The peripheral invasion can start from nasal mucosa to olfactory bulb and thereafter spread to critical brain areas, such as the thalamus and brainstem. This trans-synaptic transfer may also occur via the Vagus nerve to brainstem. Brainstem involvement with critical neuroanatomic interconnections may clarify the dysfunction of the cardiorespiratory center and the death of infected patients.

Meningoencephalitis and acute encephalopathy of the brain are the most critical types of neuronal involvement of viral infections that can result in death or serious sequelaes. Besides clinical and laboratory examination, imaging is a powerful tool for the diagnosis and follow-up of the disease. Viral encephalitis and meningoencephalitis may present with diffuse brain edema, focal hypotenuating areas, leptomeningeal and gyral enhancement or exactly normal findings on brain CT. The three reported Covid-19 associated meningoencephalitis and encephalitis cases presented with normal brain CT findings [8, 10, 11]. MRI findings of encephalitis and meningoencephalitis are more diagnostic with focal or diffuse parenchymal / leptomeningeal involvement and contrast enhancement as in the case of Moriguchi et al. [8]. Probably published in Chinese, we could not reach the clinical and imaging information of the case reported by Xiang et al. [7].

Acute necrotizing encephalopathy (ANE) is characterized by acute encephalopathy with poor prognosis and high mortality rate. CT findings include hypodense thalamic, putaminal, cerebral, cerebellar and brainstem abnormalities, like in the case reported by Poyiadji et al. [9]. Including hemorrhagic rim enhancing lesions within the bilateral thalami, medial temporal lobes, and subinsular regions, the MRI findings of their case was also compatible with ANE. In our case, diffuse brain edema, patchy hypodense areas in temporal lobes and anterior cingulate area and bilateral thalamic hemorrhage as seen by hypodense lesions were suggestive of hemorrhagic encephalopathy in comatose state. Due to the lack of MRI in our hospital, the diagnosis could not be verified.

Conclusion

The increasing number of Covid-19 infected individuals worldwide is pushing clinicians and radiologists to become aware of rare and new findings. They must consider the possibility of neuronal invasion and its clinical sequelae in patients with COVID-19. This is one of very few reported cases of Covid-19-associated acute encephalopathy and will not be the last one as Covid-19 pandemic is foreseen to proceed in the future.

References


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