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Acute Encephalitis Associated with SARS-CoV-2 Confirmed in Cerebrospinal Fluid: First Case in Malaysia

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Dear Editor,

A 69-year-old male with hypertension and atrial fibrillation presented with a 4-day history of fever, cough, and breathlessness, and subsequently developed disorientation and confusion for 1 day. On arrival, his Glasgow Coma Scale was 12/15 (E4, V3, and M5). His body temperature was 37.5°C, blood pressure was 136/60 mm Hg, pulse rate was 72 beats/min , and SpO2 was 98%, with a nasal prong of 3 L/min. There were no symptoms of meningism or long-tract signs. On the following day he became increasingly restless and breathless, and required a high-flow-rate nasal cannula to maintain adequate oxygenation. A diagnosis of Coronavirus disease-2019 (COVID-19) was established based on positivity when using an antigen rapid testing kit (SD Biosensor, Inc., Suwon, Korea). However, severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) was not detected when the polymerase chain reaction (PCR) was applied to the nasopharyngeal swab sample. High-resolution CT of his lungs showed subpleural ground-glass opacities with reticulation on the background of chronic lung changes. Noncontrast brain CT only revealed an old right lenticular infarct. The opening pressure in lumbar puncture was 9.5 cmH_2O. His cerebrospinal fluid (CSF) was clear, with a protein level of 1.16 g/L, and the CSF/serum glucose ratio was 0.58. A white blood cell count of 50 cells/mm^3 with 100% lymphocytes was seen in the CSF. SARS-CoV-2 was detected in his CSF using PCR with a cycle threshold value of 36.08 and the ORF1ab gene. The results of other CSF analyses were unremarkable.

He was treated with tablet favipiravir at 1800 mg b.i.d. for 1 day followed by 900 mg b.i.d. for 5 days, antituberculosis agents (in view of a high protein level in the CSF with 100% lymphocytes), and intravenous dexamethasone at 8 mg t.d.s., which was later tapered. After 1 week, upon positivity in PCR testing of CSF for SARS-CoV-2, a diagnosis of COVID-19-associated encephalitis was made, and antituberculosis drugs were discontinued. Intravenous methylprednisolone was applied at 500 mg daily for 3 days followed by a tapering dose of oral prednisolone over 24 days. By the second day of methylprednisolone administration, he regained full consciousness after having been disorientated for 10 days. Electroencephalography (EEG) and brain MRI were not available at our center during his admission. EEG findings were normal at a clinic follow-up performed in the third week of his illness. Brain MRI was unremarkable after 1 month of illness. He subsequently remained well with no neurological sequelae.

Neurological manifestations associated with COVID-19 are reported to range from nonspecific symptoms such as dizziness, headache, and fatigue, to diseases such as Guillain–Barré syndrome, encephalitis, myelitis, and acute necrotizing hemorrhagic encephalopathy.[1] Mao et al.[2] reported that 36.4% of hospitalized COVID-19 patients had neurological manifestations. Moriguchi et al.[3] and our case have demonstrated that encephalitis is most likely due to neurotropism of SARS-CoV-2, since the virus was detected in the CSF. Wang et al.[1] found only 1 case among 41 reviewed articles in which SARS-CoV-2 was detected in the CSF.

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