中文題目：SARS-CoV-2 感染後兩個月出現的免疫性腦炎

英文題目：Autoimmune encephalitis after 2 months of SARS-CoV-2 infection

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Introduction:

SARS-CoV-2 infection, also called COVID-19, affects the respiratory system predominantly, but previous investigations found that the virus can also invade central nervous system (CNS) system and resulted in the neurological complications. The neurological complications are found in these patients during or after COVID-19 infection, which can even seem as the one of the most common symptom [1]. The study[2] disclosed and explored onset of encephalitis from COVID-19 presentation and type of encephalitis, in which several cases showed post-COVID-19 encephalitis and presented as autoimmune encephalitis (AE). After episode of COVID-19 infection, they assumed that there is unclarified mechanism to trigger the systemic autoimmune reaction, furthermore deteriorate to AE[3-5]. In this case description, we will disclose the details of the index patient and hope to provide the possible clinical clues and experimental management.

Case Presentation:

A 30-year-old man had acute consciousness disturbance on 2022/07/12. He had COVID-19 infection on 2022/06/05 and the associated symptoms subsided within one week of isolation. Associated signs and symptoms included severe headache, nausea, vomiting. Due to sudden onset of consciousness disturbance, he was brough to our emergency department. The vital signs revealed no fever and mild hypotension. Physical examination revealed disorientation, irritability and lower abdominal pain. Laboratory tests disclosed leukocytosis (White blood cell 12.76 x 10^3/μL), neutrophilia (86%), mild hyponatremia (133mmol/L), elevated lactate (2.44mmol/L) and high serum glucose (129mg/dL); Urine drug screen demonstrated no evidence of drug abuse but Benzodiazepine was detected owing to sedative agent used in the emergency department. Under the impression of CNS infection, a series of surveys were arranged. Lumbar puncture disclosed elevated total protein (131.8 mg/dL) and lactate (3.9 mg/dL) and normal level of glucose(61mg/dL). Brain computed tomography (CT) revealed no remarkable finding; abdominal CT showed distention of urinary bladder. Then, brain magnetic resonance imaging (MRI) demonstrated slightly prominent leptomeningeal enhancement

in the occipital lobes, accompanied with very subtle ivy sign in T2 FLAIR, with differential diagnosis of encephalitis. Common viral and bacterial pathogen in cerebrospinal fluid (Multiplex PCR for meningitis & encephalitis panel) and tuberculosis were checked and showed negative finding. Electroencephalography (EEG) disclosed generalized intermittent slowing and background slowing over bilateral hemispheres, in favor of cortical dysfunction over bilateral hemispheres.

Based on the negative findings of workup for infectious diseases, autoimmune encephalitis was listed into the differential diagnosis. Blood test for limbic encephalitis panel was performed, including anti-NMDAR, anti-AMPAR1 and 2, and so on, and revealed negative finding; other autoimmune disease maker including antinuclear antibodies, rheumatoid factor, anti-thyroid peroxidase antibodies were also negative.

Empirical antibiotic with Ceftriaxone, Acyclovir and Isoniazid, Ethambutol, Rifampin, Pyrazinamide (HERZ) were administrated, but the improvement was limited. The patient still had worsening, confused consciousness and general weakness with muscle power around grade 3 symmetrically. For the suspected autoimmune encephalitis, pulse therapy with Methylprednisolone 1000mg for 5 days was administered [6], and the treatment resulted in rapid and obvious improvement of consciousness and cognitive function. The patient was able to answer simple questions correctly. When the pulse therapy completed, the patient was able to manage complex questions and to walk with walker. However, the urine retention did not improve and underactive bladder was noted via Video Urodynamics; syndrome of inappropriate antidiuretic hormone secretion was also noted. Both of those were considered as complication of encephalitis.

Discussion:

Typically, AE is owing to antibody-mediated brain inflammatory process, involving the limbic system predominantly. Many cases of AE have no obvious imaging findings, especially early in the course of the disease.

In our patient, although no definite serologic marker was detected, the clinical presentation and response to treatment strongly indicated antibody negative autoimmune encephalitis[7].

Preceding study[8] showed that most of the patients were diagnosed with encephalitis after 14.5(10.8-18.2) days COVID-19 infection in average and the incidence of encephalitis in COVID-19 group was 0.215%. Besides, it also mentioned that there are asymptomatic carriers of COVID-19 and only encephalitis symptoms were noted. In the era of COVID-19 pandemic, we should remain vigilant while approaching the patients who manifest neurological symptoms.

Previously, some case reports[9, 10] have checked myelin oligodendrocyte glycoprotein (MOG) antibody, which is a protein that is located on the surface of myelin sheaths in the CNS and as a predictor for neurological inflammation. Due to limited knowledge about secondary AE associated with SARS-CoV-2 infection, several markers are proposed and can be utilized appropriately while AE was suspected.

Conclusion:

We describe a case of a young man with encephalitis. The common etiology of encephalitis including infection and structural abnormality were ruled out at first. Additionally, his cognitive function and muscle power improved after steroid pulse therapy. Autoimmune encephalitis was diagnosed on basis of response. The neurological injury might be secondary to post–COVID-19 autoimmune syndromes chronologically. We should be aware of immune-mediated neurological sequalae and even encephalitis after COVID-19 infection.

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