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OLGU SUNUMU/CASE REPORT

A Case of SARS-CoV-2 Associated Pediatric Encephalitis

SARS-CoV-2 İlişkili Pediatrik Ensefalit Olgusu

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Abstract

SARS-CoV-2, which causes COVID-19, binds to the angiotensin-converting enzyme 2 receptors to infect human cells. Glial cells, and neurons in the brain have been reported to contain angiotensin-converting enzyme receptors, making them potential targets of SARS-CoV-2. This provides neurotrophic properties to the SARS-CoV-2. Encephalitis is defined as inflammation of the brain parenchyma caused by a viral infection or autoimmune causes. In this article, we present a case of encephalitis, which was brought with cognitive impairment and decreased responsiveness, which may be associated with SARS-CoV-2 as a result of the evaluations.

Öz

COVID-19' a neden olan SARS-CoV-2, insan hücrelerini enfekte etmek için anjiyotensin dönüştürücü enzim 2 reseptörüne bağlanır. Beyindeki glial hücreler ve nöronlar; anjiyotensin dönüştürücü enzim 2 reseptörü içerdiğinden dolayı SARS-CoV-2'nin potansiyel hedeflerindendir. Bu durum SARS-CoV-2'ye nörotrofik bir özellik sağlar. Ensefalit ise viral enfeksiyonun veya otoimmün nedenlerin yol açtığı beyin parankiminin inflamasyonu olarak tanımlanır. Bu yazıda değerlendirmeler sonucunda SARS-CoV-2 ile ilişkili olabileceği düşünülen, bilinç kaybı ve tepkisizlik ile getirilen ensefalit olgusu sunuldu.

Introduction

The COVID-19 pandemic, caused by the SARS-CoV-2, remains a global threat. A new one is added to the clinical manifestation of COVID-19 every day. In addition, it has been stated that the transmission and spread of the virus increase as new variants emerge (1). To eliminate this threat, research aimed at unraveling the mechanism of action of the virus continues.

SARS-CoV-2 binds to the angiotensin-converting enzyme 2 (ACE-2) receptor in order to infect human cells, as in SARS-CoV. Therefore, cells expressing ACE-2 are targets for the virus. In addition to endothelial and arterial smooth muscle cells in the brain, glial cells and neurons have been reported to contain ACE-2 receptors, making them potential targets of SARS-CoV-2 (2). The fact that cells in the nervous system have this receptor indicates that one of the targets of the virus is the nervous system. This provides neurotrophic properties to the SARS-CoV-2 virus (3).

Encephalitis is defined as inflammation of the brain parenchyma caused by a viral infection or autoimmune causes. Cerebrospinal fluid (CSF) pleocytosis, imaging changes, or focal abnormalities on electroencephalogram (EEG) are considered clinical evidence of brain inflammation (4).

In this article, we present a case of encephalitis, brought by loss of consciousness and decreased response, which may be associated with SARS-CoV-2 as a result of the evaluations.

Case Report

A 15-year-old male patient was brought to the emergency department with cognitive impairment and decreased responsiveness. He was brought by ambulance to the emergency department, where he was found to be stuporous with stable vital signs.

It was learned that his current complaints started two days ago. His family stated that the patient had a mild fever for two days and that he started to lose consciousness after vomiting.

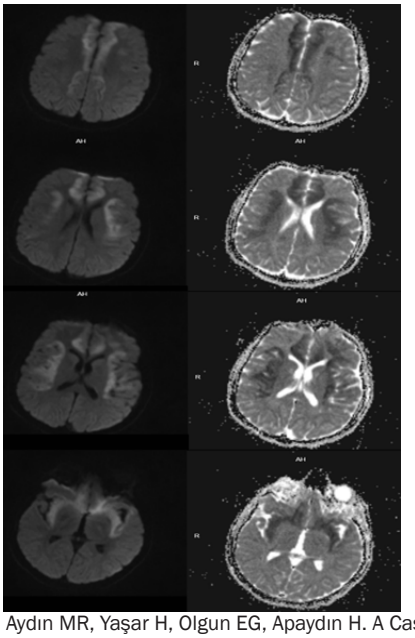
He was not taking any drugs, family and social histories were unremarkable. In vital signs; body temperature 37.8 C, heart rate 101 beats/minute, respiratory rate 24, blood pressure 130/85 mm/Hg. In his neurological examination, Glasgow Coma Scale score was seven points (E:2, V:2, M:3), it was found that he opened his eyes to pain, made meaningless sounds, and had abnormal flexion with pain, and decreased his level of consciousness. Kernig's sign, Brudzinski's sign, and nuchal rigidity were negative. Pupils were isochoric and light reaction was present. Respiratory system examination revealed rales in the lung bases, other system examinations were unremarkable.

The test results revealed as sodium (Na): 136 (135-145) milliequivalents per liter (mEq / L), potassium (K): 4.7 (3.5-5.5) millimoles per liter (mmol/L), alanine aminotransferase (ALT) : 14 (0-50) international units per milliliter (IU / ml), aspartate aminotransferase (AST) : 41 (0-50) IU / ml, leukocyte : 16.100 (4600-10200) / mm3, C reactive protein (CRP) : 53 (0-5) milligrams per liter (mg / L), ferritin: 1102 (20- 500) nanograms per milliliter (ng/mL) and D-Dimer: 1200 (69–243) ng/mL.

Due to the high fever among the patient's current symptoms, a nasopharyngeal swab sample was taken and a PCR evaluation was sent.

In the brain diffusion magnetic resonance imaging; in the bilateral insular cortex, acute infarct areas with diffusion restriction extending to the amygdala, left superior temporal gyrus, inferior frontal gyri, frontal and parietal operculum, straight gyri, superior frontal gyri, and cingulate gyri were noted. (Figure-1)

Figure I. Hyperintense areas on diffusion-weighted magnetic resonance imaging (left side) and hypointense areas on ADC map (right side)

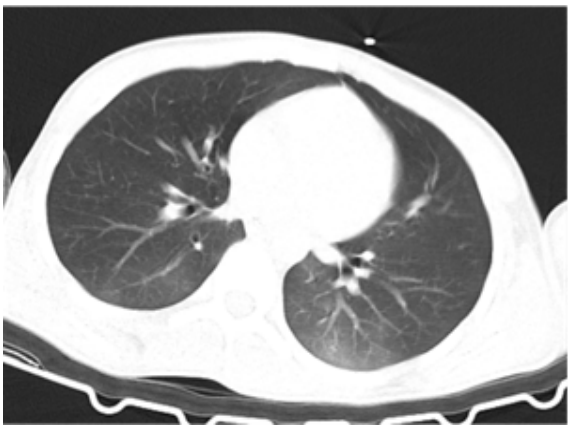


In thorax computed tomography imaging; slight ground glass appearances were detected in the lower lobes of the lung. (Figure-2)

The patient was intubated and referred to the intensive care unit with a pre-diagnosis of SARS-CoV-2-associated encephalitis. After a short time; SARS-CoV-2 polymerase chain reaction (PCR) examination of the patient's nasopharyngeal swab sample was also positive.

Lumbar puncture (LP) could not be performed due to the development of cerebral edema in the intensive care follow-ups. The patient who had cardiac arrest in the intensive care unit was accepted as exitus as there was no response to cardiopulmonary resuscitation.

Figure II. Ground glass appearances in thorax computed tomography imaging



Discussion

Neurological manifestations caused by SARS-CoV-2 include loss of taste and smell, encephalitis, dementia, ischemic stroke, Guillain Barre Syndrome, and psychosis (5).

Jordan et al. stated that the main clinical symptoms of encephalitis/meningitis associated with SARS-CoV-2 were impaired consciousness (59.38%), seizure (21.88%), delirium (18.75%), and headache (18.75%) (6).

Our case was brought to the emergency room with cognitive impairment and decreased responsiveness.

Researchers have probably identified two ways for SARS-CoV-2 to cause central nervous system (CNS) involvement. The first is hematogenous spread and the second is retrograde dissemination of neurons via indirect routes. In addition, the neurotropic mechanism of SARS-CoV-2 has not yet been elucidated (7).

Rothan et al. stated that SARS-CoV-2 also has the ability to directly invade the nervous system. Considering the known neurotropism of SARS-CoV strains, it has been stated that SARS-CoV-2 can spread directly to the central nervous system (CNS) causing meningitis and encephalitis (8). Netland et al. stated in their study that SARS-CoV-2 infection may cause an increase in the secretion of inflammatory factors such as TNF-alpha, IL-1, and IL-6, which may be the cause of neuropsychiatric symptoms (9).

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It has been stated that SARS-CoV-2 can be directly shown to be neuroinvasive in cerebrospinal fluid examination (10). Huo et al. reported that CSF evaluation was reported as normal in most of the patients with COVID-19-related encephalopathy in their systematic review study. In the same review, it was emphasized that detailed nervous system physical examination and positive SARS-CoV-2 detection rate in CSF are very important to provide direct neurotropic evidence of SARS-CoV-2 (11). CSF evaluation could not be performed in our case because there was cerebral edema.

In SARS-CoV-2-associated encephalitis, infection or inflammation can involve any part of the brain; it has been shown that it can involve the temporal lobe (15.63%), white matter (12.5%), anterior lobe (9.38%), and corpus callosum (9.38%) (11). Neuroimaging abnormalities in SARS-CoV-2-associated encephalitis were reported to occur with high T2/FLAIR signal hyperintensity, usually in the subcortical white matter or other parts of the brain injury, and there were many COVID-19 patients (38.71%) without significant neuroimaging changes in encephalitis (12). In our case, brain diffusion magnetic resonance imaging revealed acute infarct areas showing diffusion restriction extending to the bilateral insular cortex, amygdala, left superior temporal gyrus, inferior frontal gyri, frontal and parietal operculum, straight gyrus, superior frontal gyrus, and cingulate gyri.

Almost all patients with SARS-CoV-2 associated encephalitis have been shown to have lung abnormalities (13). In our case; ground glass areas that may be associated with COVID-19 were detected in the patient's thorax computed tomography imaging.

It has been reported that the presence of neurological disease in COVID-19 patients is associated with higher mortality, impaired consciousness, resistant epilepsy, and severe physical disability (11). Our case deteriorated rapidly and resulted in death.

As a result, neurological manifestations can be seen in COVID-19 symptoms. Patients with neurological symptoms should also be evaluated for COVID-19. In the suspicion or presence of neurological pathologies, the diagnosis of COVID-19 should be kept in mind by clinicians. Neuroinvasive studies to be conducted will contribute to the elucidation of the neurological involvement mechanism of SARS-CoV-2.

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