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Case Report

A Rare Cause of Childhood Encephalitis: Human Coronavirus HKU1

Çocukluk Çağında Ensefalitin Nadir Bir Nedeni: Koronavirüs HKU1

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ABSTRACT

Coronaviruses are primarily known for causing respiratory infections; however, emerging evidence suggests they may also cause neurological complications. We present a lethal case of encephalitis in a previously healthy five-year-old boy, in whom Human Coronavirus HKU1 (HCoV-HKU1) was identified through respiratory microarray and postmortem cerebrospinal fluid (CSF) PCR analysis. This report underscores the necessity of including coronaviruses in the differential diagnosis of pediatric encephalitis of unknown etiology.

Keywords: *coronavirüs hku1, encephalitis*

ÖZET

Koronavirüsler daha sık olarak solunum yolu hastalıklarına yol açar. Ancak bu virüsler nadiren de olsa sinir sistemini tutarak nörolojik hastalıklara da yol açabilir. Ani başlayan ateş, kusma, baş ağrısı ve nöbet ile gelen 5 yaşında erkek olguda solunum yolu mikroarray sürüntüsünde koronavirüs HKU 1 saptandı. Mortalite ile sonuçlanan koronavirüs kaynaklı ensefalit olgusunu sunmayı planladık.

Keywords: *koronavirüs hku1, ensefalit*

INTRODUCTION

Viral respiratory infections remain a significant public health concern due to their high morbidity and mortality, especially among children, the elderly, and immunocompromised individuals. The most prevalent respiratory viruses include human respiratory syncytial virus (hRSV), influenza virus (IV), human coronavirus (HCoV), and human metapneumovirus (hMPV). These viruses are frequently responsible for bronchiolitis and pneumonia, resulting in heightened hospitalization rates during the winter months. In addition to respiratory track involvement, they have been increasingly linked to neurological manifestations such as seizures, status epilepticus, encephalopathy, and encephalitis [1].

CASE REPORT

A previously healthy 5-year-old boy presented with a sudden onset of malaise, poor appetite, headache, and vomiting. Shortly after admission, he experienced a

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generalized tonic-clonic seizure lasting approximately two minutes, accompanied by ocular fixation, loss of consciousness, and urinary incontinence. Recurrent brief seizures followed.

Initial clinical assessment revealed: fever of 38.2°C, tachycardia (180 bpm), irregular respiration (38 breaths/min), and normotension (BP: 100/60 mmHg). The patient was unresponsive, with absent orientation and cooperation. Pupils were bilaterally dilated with absence of light reflexes, optic disc borders were blurred, and retinal hemorrhages were observed. He exhibited withdrawal to painful stimuli in the lower extremities and had bilateral extensor plantar responses.

Phenytoin (20 mg/kg IV) was administered intravenously after two benzodiazepine doses en route to the hospital. Empirical treatment with ceftriaxone, vancomycin, and acyclovir was initiated for suspected encephalitis, along with maintenance antiepileptic therapy.

Laboratory results revealed hyperglycemia (glucose: 226 mg/dL), metabolic acidosis (pH: 7.33, HCO_3: 13.7, pCO_2: 19 mmHg), elevated AST (78 U/L), leukocytosis (11,270/mm^3), anemia (Hb: 10.4 g/dL), and mild elevation of CRP (1.06 mg/dL). Other parameters, including liver enzymes, electrolytes, and calcium levels, were within reference ranges. Bicarbonate supplementation and insulin infusion were initiated.

Cranial MRI revealed bilateral periventricular cytotoxic edema with diffusion restriction, decreased cerebral perfusion, brainstem CSF space obliteration, and a 22 mm uncal herniation. An arachnoid cyst with increased diffusion was also observed in the retrocerebellar area. Figure 1 demonstrates diffusion-weighted magnetic resonance images (A, B) and a sagittal T2-weighted image (C) of the patient. Due to herniation risk, lumbar puncture was contraindicated. Antiedema therapy with hypertonic saline was initiated. Despite intensive care management, the patient succumbed on day 14.

HCoV-HKU1 was detected in the respiratory tract swab and confirmed in the postmortem CSF sample via PCR analysis.

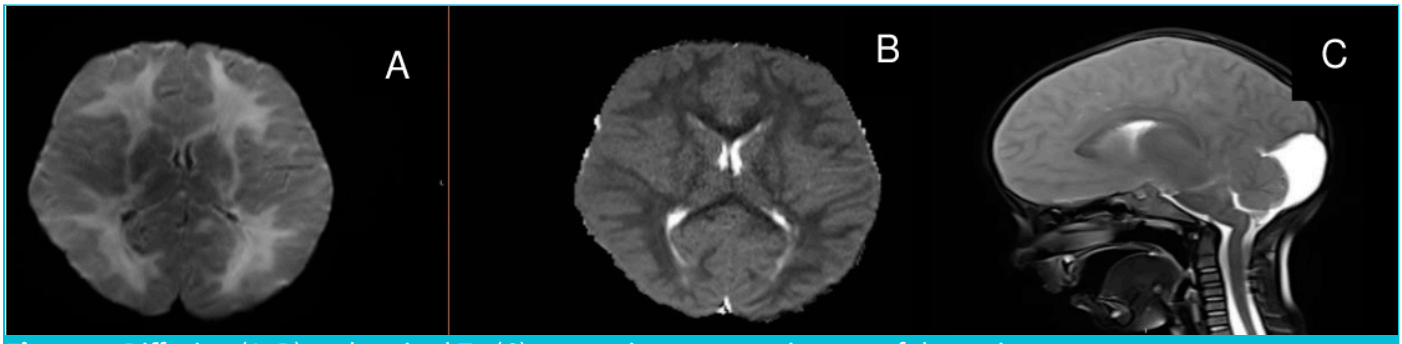

Figure 1. Diffusion (A, B) and sagittal T2 (C) magnetic resonance images of the patient

DISCUSSION

Both blood-brain and blood-CNS barriers play an important role in protecting the brain from unwanted molecules, pathogens and cells. The blood-brain barrier consists of cerebral microvascular endothelium, astrocytes, pericytes and the extracellular matrix. Viral pathogens invade the CNS via various routes, including the olfactory nerve, peripheral nerves or the haematogenous route.[2] Coronaviruses frequently cause enteric and respiratory disease. Coronavirus OC43 (HCoV-OC43), Coronavirus 229E, SARS-CoV and MERS-CoV frequently cause disease in humans. Notably, they have been described as neurotropic and neuroinvasive, causing multiple sclerosis and encephalomyelitis in humans and other hosts[3].

Human coronaviruses are enveloped RNA viruses, and seven distinct types have been identified: Coronavirus 229E (HCoV-229E), Coronavirus OC43 (HCoV-OC43), SARS-CoV, Coronavirus NL63 (HCoV-NL63, New Haven), Coronavirus HKU1, MERS-CoV, 2019-nCoV (SARS-CoV-2, Wuhan coronavirus). Coronaviruses account for 1.6% of respiratory tract infections based on nasopharyngeal aspirate samples. HCoV-OC43 was identified as the most prevalent coronavirus detected, followed by HCoV-NL63, CoV-HKU1 and HCoV-229E[4]. Although CoV-HKU1 infections are most commonly associated with the upper respiratory tract, more severe disease (pneumonia, acute bronchiolitis and

asthma exacerbation) may occur, especially in those with underlying disease. In young children, CoV- HKU1 infection is associated with a high rate of febrile seizures (50%)[1]. In a study conducted for respiratory pathogens related to viral etiological causes of febrile seizures and involving 174 paediatric patients, CoV-OC43 was the most common agent in patients younger than 12 months. In addition, CoV-HKU1 infection is also associated with febrile seizures in young children[6].

Many studies have evaluated the neurological symptoms of coronaviruses in children. In a retrospective cohort study conducted in the United States, 1683 respiratory tract samples from children were evaluated, 84 (5%) were positive for coronaviruses (229E, HKU1, NL63 and OC43). Of these, five children (8%) had meningoencephalitis and/or febrile seizures [2,7]. A prospective multicentre study conducted in China confirmed the association of coronaviruses, especially HKU1, with febrile seizures: Among 4181 participants, 87 (2.1%) tested positive for coronavirus (229E, HKU1, NL63 and/or OC43). Of the 13 children positive for HKU1, five (38%) had febrile seizures[8].

Finally, there are five case reports of coronavirus-associated neurological complications in children: acute flaccid paralysis, acute disseminated encephalomyelitis (ADEM), Guillain-Barre syndrome (GBS) and two fatal cases of encephalitis (both with immunodeficiency).

A retrospective cohort study of neurological complications associated with HCoV infections in adults in Finland was conducted. OC43 was detected in 28 of 14000 individuals. Meningitis, convulsions, headache and vertigo were observed in four of these patients[9].

In conclusion, coronaviruses, which mainly cause respiratory tract infections, may rarely cause encephalitis with a fatal course. It is recommended that patients diagnosed with encephalitis with an undetected causative agent should also be evaluated for non-COVID-19 coronaviruses.

Patient Consent Form / Hasta Onam Formu

The parents' of this patient consent was obtained for this study.

Conflict of Interest / Çıkar Çatışması

The authors declared no conflicts of interest with respect to authorship and/or publication of the article.

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