Pseudotumor Cerebri Caused by SARS-CoV-2 Infection in a Boy

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Abstract

Keywords

► pseudotumor cerebri
► idiopathic intracranial hypertension
► SARS-CoV-2
► Mycoplasma pneumoniae

In this case report, we present the case of a 7-year-old male patient who started with diplopia and paralysis of the sixth unilateral cranial nerve after a severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection. The cranial resonance was normal and the cerebrospinal pressure was 32 cm H2O detected by lumbar puncture. The treatment with corticosteroids and acetazolamide was effective. This is the first case of idiopathic intracranial hypertension associated to SARS-CoV-2 probably due to immune-mediated process.

Introduction

The neuroinvasive propensity has been reported to be a common feature of infection by coronaviruses such as severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) (COVID-19). In fact, neurological symptoms are recognized as a frequent manifestation of COVID-19, caused by direct involvement of the nervous system or activation of an exaggerated immune-mediated response.¹ A study of 214 adult patients with COVID-19 in Wuhan found that 36.4% had neurological symptoms such as acute cerebrovascular disease, headache, dizziness, impaired consciousness, ataxia, or neuropathy.²

So far, there are no reported cases of children with neurological disorders secondary to COVID-19, which could be due to underdiagnosis of the infection because children tend to have fewer symptoms or be asymptomatic.

Case

A 7-year-old boy consulted for convergent strabismus, blurred vision, and binocular diplopia of 36 hours of evolution without headache, vomiting, or pain with eye movements. The neurological examination revealed the presence of bilateral papilloedema and sixth cranial nerve palsy of left eye. The patient was not obese and the rest of the pediatric and neurological examination was normal. Up to 3 days before, the patient had suffered a 2-week history of vomiting, cough and fever (maximum temperature 39.2°C), hypogeusia, and hyposmia. Parents had also developed fever and hyposmia.

Magnetic resonance venography imaging was normal. Cerebrospinal fluid (CSF) pressure was 32 cm H2O detected by lumbar puncture. CSF cell count, glucose, protein values, and the study of viruses and bacteria in CSF were normal. Visual-evoked potentials were also normal.

Blood test showed leukocytosis (16,470/µL) without lymphopenia, thrombocytosis (platelets: 1,057,000/µL), hypertransaminasemia (GOT 84 U/L, GPT 90 U/L), and low values of 25-OH vitamin D (16.9 ng/mL). Acute phase reactants were
high (C-reactive protein 7.71 mg/dL and ferritin 437 ng/mL). A slight increase in fibrinogen (562 mg/dL) and D-dimer levels (0.82 mg/L) was observed in coagulation analysis.

Polymerase chain reaction (PCR) SARS-CoV-2 from nasopharyngeal simple was negative. Serology blood tests for herpes simplex virus (types 1 and 2), cytomegalovirus, Epstein–Barr virus, Brucella, Borrelia burgdorferi, and Toxoplasma were negative. Both immunoglobulin G (IgG) and IgM of Mycoplasma pneumoniae were positive. Rapid immunochromatographic tests for detection of Ig of SARS-CoV-2 were positive for IgG and negative for IgM.

Chest radiography and echocardiography were normal. Due to the possible coinfection of COVID-19 and M. pneumoniae, oral azithromycin was initiated for 5 days and low molecular weight heparin due to thrombotic risk. When intracranial hypertension was detected, treatment with intravenous methylprednisolone (20 mg/kg/day) was administered for 5 days and oral acetazolamide (20 mg/kg/day) was subsequently started. The evolution was excellent. Strabismus and diplopia was resolved 2 days after starting treatment and no recurrences or other complications have been reported.

**Discussion**

Pseudotumor cerebri (PC) or idiopathic intracranial hypertension is defined as elevated intracranial pressure with normal cerebrospinal composition, neuroimaging studies, and neurological examination (with the exception of sixth cranial nerve palsy).³ The association between PC and infections including hepatitis E, hepatitis A virus, measles, and M. pneumoniae has been described, especially in children.⁴ This relationship seems to be due to inflammatory process, either primary during acute central nervous system infection or secondary to an immune-mediated process, that cause a dysfunction or the absorptive mechanism of the arachnoids’ granulations, ending in decreased central nervous system absorption.⁵ These autoimmune mechanisms cause symptoms that appear after active infection, which would justify the negative of our PCR results.

Coinfections of COVID-19 and M. pneumoniae have been described in adult patients.⁶ In this case, the presence of prolonged fever together with hyposmia and hypogeusia
and the increased C-reactive protein, ferritin, and D-dimer values suggest that this patient had suffered from a SARS-CoV-2 infection that may have been the trigger for PC. This is the first case of PC related to COVID-19.

Conflict of Interest
None declared.

References


