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JCIMCR OPEN ACCESS Journal of Clinical Images and Medical Case Reports

ISSN 2766-7820

Case Report

Open Access, Volume 2

Isolated neurological symptoms in a teenager with COVID-19

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Received: Apr 10, 2021

Accepted: May 06, 2021

Published: May 11, 2021

Archived: www.jcimcr.org

Abstract

Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV2) predominantly affects the respiratory system. However, COVID-19 associated Multisystem Inflammatory Syndrome in Children (MIS-C) is a state of hyper inflammatory shock affecting the cardiovascular, gastrointestinal, hematologic and central nervous system. Isolated neurological manifestations have rarely been reported, especially in children and adolescents. We describe a 17-year-old boy presenting with intermittent headaches, altered mental status and right sided weakness, was found to be positive for SARS-COV2 from nasopharyngeal sampling. His neurological symptoms lasted for three weeks and subsequently improved spontaneously.

Keywords: SARS-CoV2; Encephalitis; Headaches.

Introduction

The recent pandemic caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV2) has affected people in all age groups with varied clinical signs and symptoms. The classic symptoms of fever, cough and difficulty in breathing are uncommon in children with SARS-CoV2 [1]. Concerns for neuropsychiatric manifestations in patients infected with SARS-CoV2 have been increasingly reported in some case series [2-4]. They can range anywhere from vague symptoms of anosmia, dizziness, emotional lability, and headaches to more serious complications such as acute neurological deficits, impaired consciousness, seizures, encephalopathy, and psychosis [4-8]. However, there is a paucity of reported cases in children with neurological symptoms [9,10]. We present a case of an adolescent boy with encephalitis that could be explained by SARS-CoV2 after an initial extensive infectious and neuropsychiatric workup.

Case report

17 year old boy with a known history of Wolf-Parkinson-White syndrome status post radiofrequency ablation presented to the emergency department with intermittent episodes of headaches, difficulty in speaking, numbness and left sided weakness for one week prior to presentation. He initially developed intermittent frontal headache and neck pain associated with photophobia and phonophobia that improved with ibuprofen and acetaminophen. However, four days prior to presentation he developed an episode of left-sided weakness, difficulty in speaking, disorientation and confusion for 45 minutes to one hour and would not respond appropriately to verbal commands. Hence, he was taken to an urgent care where a urine drug screen and Computed Tomography (CT) of head were done and results were negative. On the day of presentation, he developed a similar episode of altered mental status with right-sided

upper and lower extremity weakness along with right sided facial drooling, lasting for one hour that brought him to our emergency department. He denied having fever, diarrhea, cough, rhinorrhea, abdominal pain, rash or similar episodes recently. No family member with recent illness or psychiatric disorder. No known COVID-19 exposure. In the past, he was diagnosed with Wolf Parkinson White syndrome on routine electrocardiogram screening for which he underwent radiofrequency ablation twice at 13 and 15 years of age.

In the emergency department, his vital signs were within normal rage. He was able to describe his recent episode of inability to speak, tingling numbness, right sided weakness of upper and lower extremity and right-sided facial weakness with "Heaviness" in his tongue. His complete neurological exam including cognition, strength, deep tendon reflexes, cranial nerves 2 to 12, sensation to pressure, fine touch and proprioception were intact. Cerebellar signs, Romberg's sign, Babinski's sign and pronator drift were negative. A 12 lead electrocardiogram was normal. Magnetic Resonance Imaging (MRI) of head was normal. MR angiography showed incidental hypoplastic A1 segment of the right anterior cerebral artery which was an incidental finding. MRI cervical, thoracic and lumbar spine was reported normal as well. His complete blood count, basic metabolic profile and C- reactive protein were within normal limits. Neurology and infectious disease subspecialist were consulted. Routine screening for SARS-CoV2 by Polymerase Chain Reaction (PCR) (Cepheid Inc. Sunnyvale, CA, USA) performed on a nasopharyngeal swab was positive. SARS-CoV2 Immunoglobulin (IgG) titer (Abbott Laboratories, Lake Bluff, IL, USA) was positive (9.7 S/CO). Multisystem Inflammatory Syndrome (MIS-C) associated with COVID-19 in childhood was considered, but patient did not meet criteria as he did not have fever, no evidence of systemic inflammations or other organ involvement. MIS-C workup that included ferritin, erythrocyte sedimentation rate, D-dimer, fibrinogen, prothrombin time and activated thromboplastin time and troponin were within normal range. Cerebrospinal Fluid (CSF) analysis showed lymphocytic pleocytosis (449/mm^3) with 100% lymphocytes on cytospin differential, elevated protein (218 mg/dl) and normal glucose (57 mg/dl). Due to concern for meningitis on preliminary CSF studies, he was started on intravenous ceftriaxone, vancomycin and acyclovir. CSF gram stain and culture was negative. Extensive infectious diseases work-up was done. CSF studies were sent to assess for West Nile, Epstein Barr Virus (EBV), Lyme disease, Cytomegalovirus (CMV), Herpes Simplex Virus (HSV) 1 and 2, histoplasmosis, blastomycosis, mycoplasma, Lymphocytic Choriomeningitis (LCM) virus, parvovirus, *Bartonella* and enterovirus, and all were found to be negative. Serologies for parvovirus, *Bartonella henselae*, mycoplasma and *Borrelia burgdorferi* were negative. A Purified Protein Derivative (PPD) test for tuberculosis was also negative. A 24 hour video electroencephalogram did not show any electrographic epileptiform discharges. CSF analysis for the 22 most common autoimmune encephalitis disorders including oligoclonal bands, anti N-Methyl-D-Aspartate (NMDA) receptor antibodies, and myelin oligodendrocyte glycoprotein was also negative.

During the hospital stay, he developed two episodes of altered mental status, disorientation, confusion, unable to follow verbal commands as well as emotional lability in the form of crying and agitation and right sided weakness and numbness lasting for up to 1 hour. He continued to have waxing and waning headaches. His vital signs remained stable during these episodes. A repeat COVID-19 nasopharyngeal swab was sent on day 10 of admission that remained positive. He was discharged two weeks later with no residual weakness, paresthesia, loss of sensations or focal neurological deficits. At neurology outpatient follow up 3 weeks later, he remained asymptomatic with no recurrent episodes since hospital discharge.

Discussion

Increasing evidence regarding the neuropsychiatric manifestation of COVID 19 during the pandemic is rapidly evolving. Multi-centerc studies have shown comparable neuropsychiatric symptoms in patients during Severe Respiratory Distress Syndrome (SARS) and Middle East Respiratory Syndrome (MERS) pandemics [5,6]. Various theories regarding the pathogenesis of neuropsychiatric disease include immune mediated neuronal injury, neurotropism and neuronal proliferation of the virus, neuro-inflammatory injury of endothelium causing blood stasis and direct neuronal injury [2,6]. The most common neurological symptoms seen in COVID-19 include dizziness and headache whereas the common psychiatric symptoms included anxiety depression and delirium [2,5-8]. Neurological symptoms in four children with MIS-C with a median (interquartile range) age of 12 years [8-15], included encephalopathy (4/4), headache (3/4), dysarthria or dysphagia (2/4), meningism (1/4) and cerebellar ataxia (1/4) [10]. All four patients had fever, cardiovascular shock and fever. A 14-year-old boy presenting with abdominal pain, rash and fever who met the criteria for MIS-C developed fluctuating impairment in awareness, psychosis and agitation [9]. Our patient presented with headache, altered mental status and unilateral weakness of face, upper and lower extremities without respiratory or systemic symptoms suggesting the possibility of isolated neurological symptoms. His vital signs were normal with no fever and MIS-C work up did not show elevated inflammatory markers to suggest a hyperinflammatory state.

Central nervous system disease in the form of ischemic stroke, intracerebral hemorrhage, acute disseminated encephalomyelitis, encephalitis, and Central Nervous System (CNS) vasculitis as well as peripheral nervous system involvement in the form of Gillian Barre syndrome and its variants has been reported in patients with SARS-CoV2 [4-7]. In the above mentioned series of four children, two had normal Cerebrospinal Fluid (CSF) analysis with normal protein and glucose levels [10]. However, in our patient, lymphocytic pleocytosis and elevated protein was noted in the CSF suggesting viral meningoencephalitis likely due to SARS CoV-2. PCR test for SARS-CoV2 in the CSF was not available to confirm the diagnosis, however, extensive CSF work up for viral and autoimmune encephalitis all returned negative. Signal abnormalities with diffusion restriction in the splenium of corpus callosum as well as centrum semiovale have been described on Magnetic Resonance Imaging (MRI) in children with neurological symptoms [10]. However, our patient had no abnormalities on MRI. MRA showed incidental hypoplastic A1 segment of the right anterior cerebral artery that could not explain his symptoms.

Improvement in the neurological as well as systemic symptoms in children has been shown with the use of immunomodu-

latory therapies such as steroids, intravenous immunoglobulins, anti-TNF alfa drugs and rituximab [9,10]. Our patient's symptoms improved gradually over the course of hospital stay and did not require any immunomodulatory therapy as there was no concern for MIS-C and MRI brain did not show signs of inflammation.

As the typical respiratory symptoms are less common in children with acute COVID 19, a high index of suspicion is necessary to diagnose children presenting with other organ involvement. Neuropsychiatric manifestations have been reported in a few children that have been a part of their systemic COVID-19 illness. However, isolated neurological findings in children are extremely rare and further studies are necessary to establish the pathogenesis of SARS-COV2 in the central nervous system.

Declarations

Disclosure of Conflict of Interests: The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding: The author(s) received no financial support for the research, authorship, and/or publication of this article.

Ethical approval: This was an observation. No ethical approval was needed as per the Detroit Medical Center ethics committee.

Consent to publish: The parent of the child has consented to the submission of the case report to the journal for possible publication.

Author's contribution: The first draft of the manuscript was written by Pezad Doctor and all authors commented on previous versions of the manuscript. All authors read and approved the final manuscript.

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