Radiology Case Reports 16 (2021) 1400–1404

Available online at www.sciencedirect.com

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journal homepage: www.elsevier.com/locate/radcr

Case Report

COVID-19 as a rare cause of facial nerve neuritis in a pediatric patient☆,☆☆,★

Sultan Zain, BA, MPH[a,*], *Kalliopi Petropoulou, MD*[b], *Kanish Mirchia, MD*[c], *Abdelmohsen Hussien, MD*[b], *Kavya Mirchia, MD*[b]

[a] *College of Medicine, SUNY Upstate Medical University, 750 E Adams St, Syracuse, NY 13210, USA*
[b] *Department of Radiology, Upstate University Hospital, 750 E Adams St, Syracuse, NY 13210, USA*
[c] *Department of Pathology, Upstate University Hospital, 750 E Adams St, Syracuse, NY 13210, USA*

ARTICLE INFO

Article history:
Received 14 March 2021
Revised 26 March 2021
Accepted 28 March 2021
Available online 1 April 2021

Keywords:
COVID-19
facial nerve palsy
pediatric
neuritis
magnetic resonance imaging
neuroradiology

ABSTRACT

COVID-19 has been noted to present with neurological symptoms in nearly 30% of patients. While children are more likely to be asymptomatic, neurological involvement has been observed. We present the case of a 23-month-old previously healthy female who was brought to the emergency room for a new-onset facial droop. The patient tested positive for COVID-19 but was otherwise asymptomatic. Magnetic resonance imaging of the brain with and without contrast revealed abnormal enhancement along the canalicular segment of the right cranial VII extending to the first genu suggestive of cranial nerve neuritis. Given that our case involves a pediatric patient with no significant comorbidities presenting with facial drop, COVID-19 should be considered on the differential when evaluating causes of new onset peripheral nerve palsies.

Introduction

COVID-19 has presented the medical community with an array of therapeutic and diagnostic challenges as a result of its far-reaching and often varied parainfectious and postinfectious sequelae affecting multiple body systems. In particular, its neurological impacts have been well-noted and fairly common with over thirty percent of patients displaying neurological symptoms [1,2] While children tend to be less symptomatic overall, some have been shown to have neurological involvement, often in the absence of respiratory symptoms [3-5]. A few cases of facial palsy associated with COVID-19 have been noted in the literature, with only one being described in a pediatric patient [14-17]. We present a rare case of a 23-month-old female with no significant comorbidities presenting with peripheral facial palsy and radiological findings consistent with neuritis in the setting of concurrent COVID-19 infection.

☆ Funding: No funding was received to assist with the preparation of this manuscript.
☆☆ Declaration of competing interest: The authors declare that they have no conflict of interest.
★ Ethics approval: This is a retrospective case report not requiring ethics approval.
* Corresponding author.
E-mail address: zains@upstate.edu (S. Zain).

https://doi.org/10.1016/j.radcr.2021.03.063

Case report

A 23-month-old previously healthy female up to date on vaccinations was brought to the emergency room by her father after he noticed drooping of her right eye and right side of her mouth three days prior. Two weeks prior, the patient had been diagnosed with a since resolved methicillin-resistant staphylococcus aureus skin infection of the scalp that was treated with trimethoprim-sulfamethaxazole. The father denied any other recent illnesses, fevers, rhinorrhea, cough, shortness of breath, or gastrointestinal symptoms. He also noted that the patient had been eating, drinking, voiding, and stooling normally. Birth history was significant for being large for gestational age. At initial presentation, the patient was afebrile with stable vitals. Physical examination revealed an obese, comfortable toddler in no acute distress with normal breath sounds.

Neurologic examination demonstrated an inability to fully close the right eye and drooping of the right side of the mouth, indicative of a peripheral cranial nerve VII lesion. Other cranial nerves were intact, sensory and motor examination revealed no abnormalities, and gait was normal.

Blood work was significant for mild leukopenia, microcytic hypochromic anemia, hyperkalemia, elevated alkaline phosphatase, and elevated AST. A COVID-19 polymerase chain reaction (PCR) test was done and came back positive. Erythrocyte sedimentation rate and C-reactive protein were within normal limits. Respiratory pathogen panel and cytomegalovirus PCR were negative.

Given the patient's symptoms, neurology was consulted and they recommended magnetic resonance imaging (MRI) of the brain with and without contrast as well as the following diagnostic studies: Lumbar puncture (with cell count, protein, glucose, and pathogen panel), cerebrospinal fluid (CSF) studies for Epstein-Barr virus (EBV), herpes simplex virus (HSV), varicella zoster virus (VZV), CSF angiotensin converting enzyme level, oligoclonal bands, IgG synthesis and/or index, CSF anti-MOG (myelin oligodendrocyte glycoprotein) antibody, CSF anti-NMO (neu- romyelitis optica) antibody, and blood titers for mycoplasma, EBV, and VZV.

The patient was admitted to the inpatient service the next day for further evaluation. She was initiated on dextrose 5 % in normal saline for hydration. Further history revealed that she had exposure to her mother, who was symptomatic and had recently tested positive for COVID-19. Repeat examination was significant for an erythematous papular rash in the groin area sparing the thighs most consistent with contact dermatitis treated with zinc oxide ointment. Concern for Bell's palsy prompted a 1 mg/kg/day dose of methylprednisolone.

The patient underwent brain MRI with and without contrast as well as lumbar puncture as per neurology's recommendations. Pathogen panel done of the CSF was unremarkable with cell culture showing no growth after 1 day. Furthermore, CSF cell count, glucose, and protein were within normal limits. Serum Lyme titers were negative. EBV IgM was negative though IgG was positive. The brain MRI demonstrated findings significant for abnormal enhancement along the canalicular segment of the right cranial VII extending to the first genu suggestive of neuritis (Fig. 1 and 2). There was also bilateral enhancement along the tympanic segment (Fig. 3) but this was interpreted as a normal finding due to coexisting venous collection. No other ab- normal parenchymal or meningeal enhancement was noted.

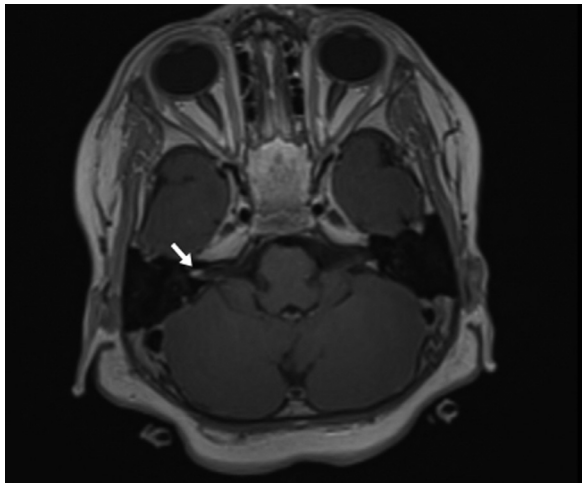

Fig. 1 – Axial T1-weighted post contrast image showing abnormal enhancement along the canalicular component of the right seventh cranial nerve extending to the first genu (white arrow)

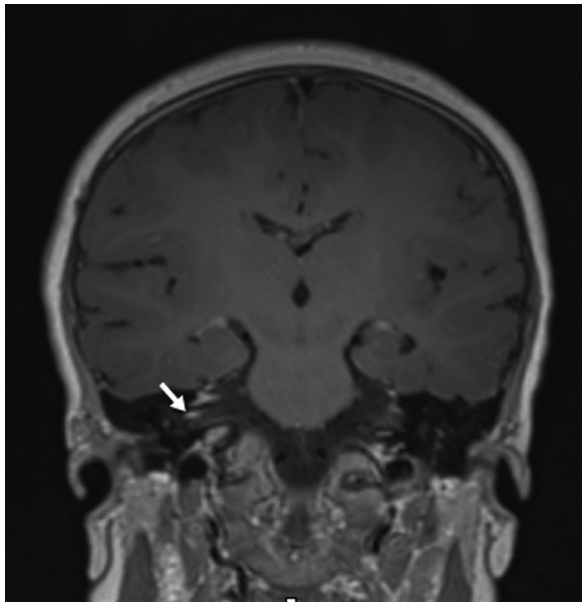

Fig. 2 – Coronal T1-weighted post contrast image showing abnormal enhancement along the canalicular component of the right seventh cranial nerve extending to the first genu (white arrow)

Given these findings, neurology agreed with pursuing a 10-day steroid course with short taper for treatment. Given the patient's neurological findings and positivity for COVID-19, a coronavirus-induced cranial nerve VII neuritis was considered most likely. Infectious disease was also consulted to help delineate between parainfectious versus postinfectious processes and advise on steroid course andtreatment. Given that patient was tolerating oral intake, the patient was discharged

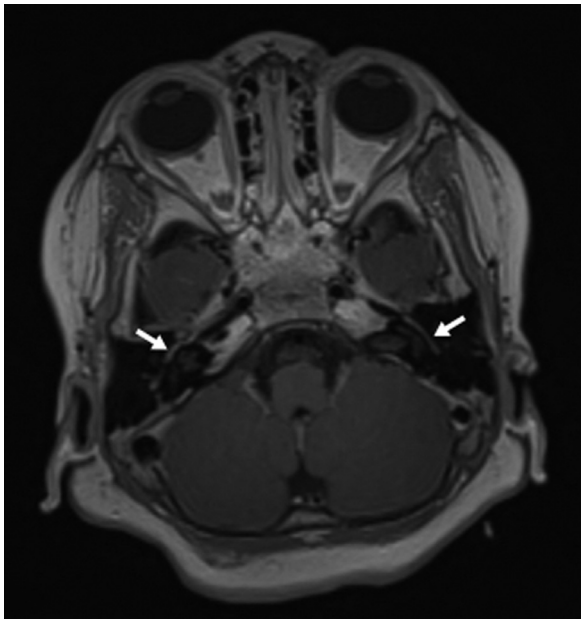

Fig. 3 – Axial T1-weighted post contrast image showing normal enhancement along the tympanic portion of facial nerve bilaterally (white arrows)

with orders to follow-up with primary care provider and take a 10-day course of prednisolone 1 mg/kg/day. She also had follow-up appointments scheduled with both infectious disease and neurology.

The patient followed up with neurology 3 weeks after discharge. Of outstanding labs at discharge, testing for anti-NMO antibodies and CSF PCR for COVID-19 was not able to be done due to insufficient quantity. CSF anti-MOG antibodies were negative. IgG synthesis and/or index and CSF angiotensin-converting enzyme were within normal limits. CSF oligoclonal bands were not noted. Rheumatological panel was also negative. Mycoplasma blood IgG was negative though VZV blood IgG was positive. Given these findings, a parainfectious COVID-19 induced cranial nerve VII myelitis seemed mostly likely.

Repeat neurological examination revealed a persistent right-sided facial weakness with flattening of the nasiolabial fold. While there was mild improvement, patient was still unable to forcefully close her right eye, leading to some dryness of the eye and tearing. Given that the patient had already completed a course of steroids, her parents were counseled that there may be a persistent, long-lasting facial weakness that could improve over time. They were also counseled to use eye drops to manage the patient's eye dryness. At a well-child visit approximately two months after her admission, the patient's facial palsy was noted to have fully resolved.

Discussion

Neurological impacts of COVID-19 have not only been varied but also fairly common, with one retrospective study finding that 36.4% of patients had neurological symptoms and/or events such as headaches, dizziness, strokes, and impaired consciousness [1]. The most commonly cited symptoms have included headaches, dizziness, taste and smell abnormalities, and impaired consciousness while cerebrovascular incidents, seizures, immune-mediated neurological syndromes, and meningoencephalitis have been found to be relatively less common [2].

Children are less symptomatic overall but in those who do manifest symptoms, neurological in volvement has been noted, often in the absence of respiratory symptoms. A study looking at 27 children who developed COVID-19 pediatric multisystem inflammatory syndrome (MIS-C) found that 14.8% of children with this syndrome had new onset neurological symptoms including encephalopathy, headaches, weakness, decreased reflexes, and cerebellar dysfunction [3].

Other manifestations have been reported such as acute disseminated encephalitis and focal cerebral arteriopathy [4,5]. An association between coronaviruses and neurological manifestations has been noted even prior to the COVID-19 pandemic, particularly with respect to febrile seizures. In a 2016 study of hospitalized patients younger than 6 years presenting with febrile seizures, human coronaviruses were detected in 9.9% of study subjects, though it should be noted that co-detection of other viruses was significant at 52.6% [6].

Radiological findings in children with COVID-associated neurological manifestations vary depending on clinical presentation. In the aforementioned study looking at children with multisystem inflammatory syndrome , all 4 children who demonstrated neurological involvement had signal changes in the splenium of the corpus callosum with 3 having T2-hyperintense lesions indicative of restricted diffusion [3]. It was suggested that these findings were secondary to an immune-mediated mechanisms as op- posed to neurotropic invasion; COVID-19 appears to cause endothelial dysfunction, which could unmask CNS antigens to the immune response [3,7]. A case report highlighting acute disseminated encephalomyelitis in a pediatric COVID-19 patient demonstrated similar findings — a T2-hyperintense and fluid-attenuated inversion recovery lesion in the splenium of the corpus callosum. The authors postulated that this pathology was likely due to a postinfectious autoimmune demyelinating process [4]. In another case report highlighting a pediatric COVID-19 patient with focal cerebral arteriopathy, MRI showed diffuse hyperintensity of the caudate, putamen, anterior limb of the internal capsule, external capsule, and the insula, likely secondary to reduced flow in the middle cerebral artery, which was demonstrated by MR angiography. This patient's nasopharynx swab and CSF both tested positive for COVID-19, indicating a parainfectious process likely secondary to a focal vasculopathy, which has been observed in association with other viruses, most notably varicella zoster [5]. It is apparent from these reports that COVID-19 can directly or indirectly lead to neurological sequelae through a parainfectious or postinfectious process.

Bell's palsy — the most common cause of lower motor neuron facial palsy — is characterized by acute onset, unilateral, forehead-sparing facial weakness that is often associated with postauricular pain, taste abnormalities, and hyperacusis [8].

Reactivation of HSV has been hypothesized to be involved in the pathogenesis of Bell's palsy but this is disputed due to a failure in isolating viral DNA from biopsy specimens [9,10]. It has also been suggested that Bell's palsy may be an autoimmune demyelinating cranial neuritis secondary to a viral infection prompting an autoimmune response to myelin [11]. This might explain why HSV DNA has not been isolated from biopsy specimens; furthermore, it explains the effectiveness of corticosteroids in treating this condition as they downregulate the immune response. There are other causes of peripheral nerve palsies, such as Lyme disease, otitis media, Ramsay Hunt syndrome, sarcoidosis, Guillain-Barré syndrome, and tumors; however, these are all associated with additional distinguishing features [12]. Interestingly, Bell's palsy has also been noted in association with certain versions of the influenza vaccine [12,13].

Acute onset peripheral facial nerve palsy has been noted in relation with COVID-19, though rarely, in a few cases [14,15]. One study noted a higher occurrence of facial palsy during the COVID-19 outbreak compared to the same period of the previous year, possibly indicating an association between COVID-19 and peripheral facial nerve palsy [16]. There has only been one case noted in the literature of facial nerve palsy associated with COVID-19 in a pediatric patient; a 6-year-old male with significant medical issues most significant for agammaglobulinemia with hyper IgM developed acute facial palsy in the setting of concurrent COVID-19 infection [17].

In this case, the radiological findings of unilateral enhancement within the canalicular segment to the first genu of cranial nerve VII were suggestive of peripheral facial nerve neuritis. While the geniculate, mastoid, and tympanic segments of the facial segments can enhance even under normal conditions due to neighboring venous plexuses, enhancement within the intracanalicular-labyrinthine segment, as seen in this patient, is more likely to be pathologic [18]. The etiology of this neuritis is less clear. With the CSF studies being grossly normal and tests for HSV and Lyme being negative, many of the other common etiologies on our differential could be ruled out. The presence of VZV IgG indicated either immunization or past infection; VZV as a cause was less likely though as there weren't any other signs and/or symptoms consistent with reactivation or primary infection. Ultimately, considering the timing of symptom onset with the presence of concurrent COVID-19 infection, it is most likely that this was a peripheral facial nerve neuritis induced by COVID-19.

Conclusion

COVID-19 has been associated with numerous neurological complications, of which new onset peripheral nerve palsy should also be noted. Our case involves a pediatric patient with no significant comorbidities, in contrast with the previous case noted in the literature. As such, while rare, COVID-19 should be considered on the differential as a cause for patients presenting with new onset peripheral nerve palsies.

Declarations

Consent to participate: All patient data has been removed and no informed consent is required to participate.

Consent for publication: All patient data has been removed and no informed consent is required to publish.

Authors' contribution: All authors contributed to writing this manuscript. All authors read and approved the final manuscript.

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