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Vestibular neuritis caused by severe acute respiratory syndrome coronavirus 2 infection diagnosed by serology: Case report

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Abstract

Vestibular neuritis is a disorder selectively affecting the vestibular portion of the eighth cranial nerve generally considered to be inflammatory in nature. There have been no reports of severe acute respiratory syndrome coronavirus 2 causing vestibular neuritis. We present the case of a 42-year-old Caucasian male physician, providing care to COVID-19 patients, with no significant past medical history, who developed acute vestibular neuritis, 2 weeks following a mild respiratory illness, later diagnosed as COVID-19. Physicians should keep severe acute respiratory syndrome coronavirus 2 high on the list as a possible etiology when suspecting vestibular neuritis, given the extent and implications of the current pandemic and the high contagiousness potential.

Keywords

COVID-19, severe acute respiratory syndrome coronavirus 2, vestibular neuritis, serology, case report

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Background

As the COVID-19 pandemic progresses, reports of neurological manifestations are increasing. These manifestations can be considered as direct effects of the virus on the nervous system, para-infectious or post-infectious immune-mediated disease, and neurological complications of the systemic effects of COVID-19.[1]

Vestibular neuritis is a disorder selectively affecting the vestibular portion of the eighth cranial nerve generally considered to be inflammatory in nature.[2] The cause of vestibular neuritis is presumed to be of viral origin (e.g. the reactivation of latent herpes simplex virus (HSV) infection), but other proposed etiologies include both vascular and immunologic causes.[2] Previous studies cautioned about the neuro-invasive potential of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), because of high similarity with SARS-CoV which has a well-known neuro-invasive propensity.[3] Immediate neurologic complications in patients with COVID-19 have been previously identified and reported.[4] Vestibular neuritis as a long-term neurologic complication from SARS-CoV-2 infection has not been yet reported.

Case report

On 12 March 2020 as the first cases of COVID-19 were being reported in Michigan, but prior to general availability of SARS-CoV-2 testing, a 42-year-old male patient developed a mild acute illness characterized by congestion, sore throat, subjective fevers, dry cough and chills along with body aches and fatigue. The patient has been in his usual state of health before developing the symptoms. His only medical history includes multiple episodes of kidney stones. Symptoms improved with acetaminophen and ibuprofen and completely resolved after 5 days.

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Table 1. Clinical laboratory results.

Variable	Reference range	Results 1 (28 March 2020)	Results 2 (15 April 2020)
Blood counts			
WBC	3.5–10.1 bil/L	13.1	5.6
RBC	4.31–5.48 tril/L	4.76	4.47
Hemoglobin	13.5–17.0 g/dL	14.4	13.7
Hematocrit	40.1%–50.1%	41.9	40.6
Mean corpuscular volume	80–100 fL	88	91
Mean corpuscular hemoglobin	28–33 pg	30	31
Mean corpuscular hemoglobin concentration	32–35 g/dL	34	34
Red blood cell distribution width - coefficient of variation	12%–15%	11	12
Platelet	150–400 bil/L	326	215
Neutrophils	1.6–7.2 bil/L	11.8	3.6
Lymphocytes	1.1–4.0 bil/L	0.8	1.3
Monocytes	0.0–0.9 bil/L	0.4	0.5
Eosinophils	0.0–0.4 bil/L	0	0.2
Basophils	0.0–0.1 bil/L	0	0
Immature granulocytes	0.00–0.04 bil/L	0.05	0.03
Chemistry panels			
Sodium	135–145 mmol/L	139	
Potassium	3.5–5.2 mmol/L	3.7	
Chloride	98–111 mmol/L	109	
Carbon dioxide (CO_2)	20–29 mmol/L	18	
Anion gap	5.0–17	12	
Glucose	60–99 mg/dL	127	
BUN	7–25 mg/dL	11	
Creatinine	0.60–1.30 mg/dL	1.01	
Calcium	8.5–10.5 mg/dL	9.2	
AST	0–34 U/L	15	
Glomerular filtration rate non-African American	>59	91	
Cardiac markers			
Troponin I	0.00–0.03 ng/mL	0.01	

WBC: white blood cell; RBC: red blood cell; BUN: blood urea nitrogen; AST: aspartate aminotransferase.

Eleven days after resolution, on 28 March, the patient developed new symptoms of vertigo, nausea, and vomiting exacerbated by any movement of his head and body along with "pressure" like sensation in the occipital area of his head and mild photophobia. His symptoms acutely worsened throughout that day, resulting in the complete inability of the patient to sit up or to perform any activities of daily living because of severe vertigo and intractable vomiting. Emergency medical services (EMS) were called and the patient was taken to the Emergency Department (ED) for intractable vomiting and dry heaving. The patient denied any speech impairment, limb weakness, or hearing loss. He has never experienced similar symptoms before.

Upon presentation to the ED, his blood pressure was 130/79 mm Hg, heart rate was 93 beats per minute, respirations were 23 per minute, and oxygen saturation was 100% on room air. He looked to be in severe distress from nausea, dry heaving, non-bilious vomiting, and severe vertigo. Neurologically, his cognition was intact, and the cranial nerve exam was normal. Finger to nose movement and rapid alternating movements of the extremities were intact as well. There was no fluid behind his tympanic membranes, and no oropharyngeal exudates. A leftward beating horizontal nystagmus was noted. Gait could not be assessed as the patient was unable to even lift his head from the pillow. The rest of his neurologic exam was noted as normal. Because of the lack of auditory complaints, the patient did not have a hearing assessment.

Clinical laboratory results obtained in the ED were significant for leukocytosis with lymphopenia of 0.8 bil/L (Table 1). Cardiac markers and chemistries were unremarkable. Head computed tomography (CT) and brain CT angiography ruled out possible central vertigo etiologies. As the patient had severe vertigo upon presentation, he could not tolerate any vestibular testing.

The patient was treated with IV antiemetics (phenergan 25 mg), IV diazepam (2 mg), and IV fluids with significant improvement in his symptoms, and he was discharged home from the ED within 2 h. Nasopharyngeal SARS-CoV-2 reverse transcription polymerase chain reaction (RT-PCR) was obtained and yielded a negative result. Three days later, due to persistent vertigo, the patient was evaluated by a neurologist. His exam revealed persistent leftward nystagmus and a positive Romberg sign with a tendency toward the right. The patient was diagnosed with right vestibular neuritis. He was prescribed a course of oral prednisone (50 mg daily for 5 days) and meclizine 25 mg every 8 h as needed, with a gradual improvement of his symptoms over the next 2 weeks, resulting in minimal residual vertigo. Two weeks after his ED visit, the patient had testing that detected immunoglobulin G (IgG) specific for SARS-CoV-2. Repeat blood count at the same time demonstrated normalized values.

Discussion

Despite a negative RT-PCR result, presumably due to the temporal distance from the actual viral infection, this is the first case reported of SARS-CoV-2-induced vestibular neuritis confirmed by serology (EUROIMMUN SARS-CoV-2 IgG assay). Our patient presented with significant vestibular symptoms and did not have any elevation in the inflammatory markers or other laboratory parameters. This finding is consistent with the case series report where the investigators found no major abnormalities in the laboratory findings in

those presenting with nervous system involvement.[5] However, in contrast with our patient who presented with almost 2 weeks delay in his symptoms from the active COVID-19 infection, most neurologic manifestations reported occurred early in the illness (the median time to hospital admission was 1–2 days).[5] A couple of recent COVID-19 cases[6,7] reported on acute SARS-CoV-2 infection symptoms at the same time with vestibular neuritis symptoms.

Conclusion

ED physicians and primary care physicians should keep SARS-CoV-2 high on the list as a possible etiology when suspecting vestibular neuritis, given the extent and implications of the current pandemic and the high contagiousness potential.

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Author contributions

A.H. and M.H. drafted the manuscript and the table and reviewed multiple revisions of the manuscript. C.C., A.E.A., and M.S. were involved with manuscript preparation, multiple draft revisions, and conception of the table. All authors have reviewed and approved the manuscript for submission.

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Ethical approval

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Availability of data and materials

The data used for this case are available from the corresponding author on reasonable request.

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