

Vestibular neuronitis associated with an adult male COVID-19 infection: A case report

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Abstract

In the present era of the coronavirus 2019 (COVID-19) pandemic, it has been observed that the severe acute respiratory syndrome, coronavirus 2 (SARS-CoV-2) infection does not only affect the respiratory tract, but also triggers various neurological symptoms in one-third of patients. The most prominent of such symptoms is anosmia, which is independent of rhinologic symptoms such as nasal obstruction, discharge, and pain that cannot be otherwise explained. Vestibular neuronitis ranks third among the causes of peripheral vestibular vertigo, characterized by nausea, vomiting, and dizziness that develops within minutes or hours. Although the etiopathogenesis remains poorly known, neuronitis is generally considered to be attributable to the viral or post-viral inflammation of the vestibular branch of the eighth cranial nerve. This report presents a case of vestibular neuronitis, which is likely to be a manifestation of acute vestibular neuronitis associated with COVID-19.

Keywords: Anosmia; COVID-19; COVID-19 Pandemics; Vertigo; Vestibular Neuronitis.

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Introduction

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection presents as an interstitial pulmonary disease characterized by diffuse acute inflammation. SARS-CoV-2 virus, a member of the Coronaviridae family, contains four structural proteins including the S (spike), M (membrane), and two N (nucleocapsid; containing the RNA genome) proteins. The S protein binds to the angiotensin-converting enzyme-2 (ACE2) receptor of the host cell membrane.¹ Neuroinvasive effects have been demonstrated in patients infected with Middle East Respiratory Syndrome-Coronavirus (MERS-CoV) and SARS-CoV-2, and thus these two infections may share certain properties. Moreover, SARS-CoV-2 invades cells via the ACE2 receptors, which are important components of the neural renin-angiotensin system.² During the coronavirus 2019 (COVID-19) pandemic, patients present

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to emergency services with different complaints. In this report, we present a case of intractable nausea and vertigo in a patient positive for COVID-19 infection, which is likely to be a manifestation of acute vestibular neuronitis associated with COVID-19.

Case Report

In December 2020, a 60-year-old male patient applied to Van Yüzüncü Yil University Dursun Odabas Medical Center Emergency Clinic with the complaint of sudden-onset severe vertigo, dizziness, nausea, vomiting, diarrhoea, and abdominal pain. The patient also stated that he had to keep his eyes closed because he could not tolerate the surrounding environment. On arrival, his haemodynamics were stable, he had no fever, blood oxygen level was 93%, and the blood pressure was 130/60 mmHg. Bilateral otoscopic and audiological examinations were normal. An examination revealed spontaneous nystagmus with a fast component to the right. The head-thrust test was positive on the right side. The Dix-Hallpike test was negative. No improvement was noted in the symptoms on repeated maneuvers. Other cranial nerve examinations were normal. The patient had a history of hypertension and coronary artery disease. No central pathology was detected on neurological and radiological examinations including contrast-enhanced cranial Computed Tomography (CT) and diffusion-weighted

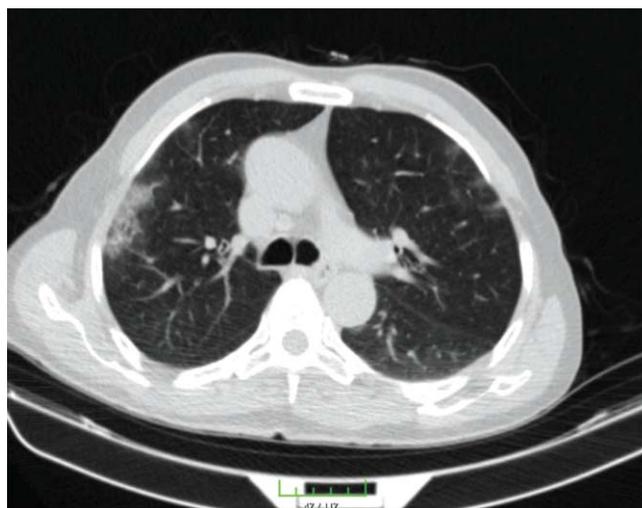


Figure: CT image showing ground-glass infiltrates.

Magnetic Resonance Imaging (MRI). Thoracic CT (Figure) was performed due to increased C-reactive protein (CRP) (25 mg/L) and D-dimer (2.99 $\mu\text{g/mL}$) levels. The radiographic appearance of the chest was compatible with COVID-19 infection symptoms and the Polymerase Chain Reaction (PCR) test was positive. Therefore, the patient was hospitalized and isolated and was initiated on a 7-day therapy including hydroxychloroquine, steroid, favipiravir, betahistine, and dimenhydrinate. However, the patient lacked most of the typical COVID-19 symptoms (fever, dry cough, malaise, muscle-joint pain, and shortness of breath), while he only had diarrhoea and persistent vertigo and nausea. Coagulation parameters including complete blood count, fibrinogen level, and platelet count were within normal limits. After a seven-day hospital stay, the symptoms improved, and the patient was able to tolerate an oral diet and was eventually discharged home. After discharge, he was advised to quarantine for seven more days. An informed consent form was obtained from the patient for the publication of this case report.

Discussion

Clinicians worldwide have emphasized that the infective symptoms of COVID-19 show remarkable variation. The most common symptoms include high fever, cough, and shortness of breath, while additional symptoms such as diarrhoea, nausea, vomiting, headache, sore throat, fatigue, muscle pain, and smell and taste disturbances have also been reported.³ Mao et al. reported neurological symptoms in 214 patients diagnosed with SARS CoV-2 and reported that the symptoms were central, peripheral, and musculoskeletal. The authors detected neurological symptoms in 36% of patients, all of which were found to be correlated with the severity of the disease. Moreover, imbalances were detected in 16.8% and headache in 13.1% of the patients with central nervous system (CNS) symptoms (24.8%), while hypogeusia (5.6%) and hyposmia (5.1%) were the most common symptoms related to the peripheral nervous system (PNS) (8.9%). The authors concluded that the incidences of stroke, altered consciousness, impaired general condition, and muscle injury were correlated significantly with disease severity, though laboratory workup showed no significant difference between patients with and without PNS symptoms and no relationship was found between the symptoms and disease severity⁴ Yan et al. detected influenza-like symptoms and loss of smell and taste in 71% of COVID-19-positive patients.⁵ One theory posits that CNS involvement reflects focal meningitis/encephalitis, particularly of the rhino-gustatory-cortical pathway. This hypothesis was supported by the detection of viral RNA in

cerebrospinal fluid (CSF).⁴ A previous report described ocular involvement in two cases in whom MRI revealed abnormal perineural or cranial nerve findings.⁶ Although the authors indicated that it remained uncertain whether the cranial neuropathies were associated with SARS-CoV-2 infection in those two cases, we suggest that SARS-CoV-2 infection may underlie the symptoms of our patient since they preceded respiratory symptoms. For these reasons, further investigation is required. In one case report, vestibular neuronitis developed in a 29-year-old patient with COVID-19.⁷

Vestibular neuronitis ranks third among the causes of peripheral vestibular vertigo, characterized by nausea, vomiting, and dizziness that develop within minutes or hours. Although the etiopathogenesis remains poorly known, neuronitis is generally considered to be attributable to viral or post-viral inflammation of the vestibular branch of the eighth cranial nerve.⁷ In the case presented, coexistence of diarrhoea and a COVID-19 positivity, points to the presence of vestibular neuritis. However, the postmortem detection of COVID-19 in vestibular nerve ganglia will be required to confirm this possibility.

Conclusion

Although cranial nerve involvement after SARS-CoV-2 infection has not been clearly established, the virus may potentially be neurotrophic, attacking the brainstem, as in other coronaviruses. Some COVID-19-infected patients may exhibit cranial nerve involvement. Viral infection is the most common cause of vestibular neuronitis. SARS-CoV-2 should be kept in mind in patients presenting to the emergency service with vertigo during the COVID-19 pandemic.

Consent: A verbal consent was obtained from the patient regarding the publication of this report.

Disclaimer: None.

Conflict of Interest: The author does not have any commercial or other association that might pose a conflict of interest.

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