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COVID-19 and Vestibular Symptoms and Assessment: A Review

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Abstract
Background: The current pandemic of COVID-19, caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), has resulted in significant morbidity and mortality primarily associated with respiratory failure. However, it has also been reported that COVID-19 can evolve into a nervous system infection. The direct and indirect mechanisms of damage associated with SARS-CoV-2 neuropathogenesis could affect our sensory functionality, including hearing and balance. ***Summary:*** In order to investigate a possible association between SARS-CoV-2 viral infection and possible damage to the vestibular system, this review describes the main findings related to diagnosing and evaluating otoneurological pathologies. ***Key Messages:*** The clinical evidence shows that SARS-CoV-2 causes acute damage to the vestibular system that would not leave significant sequelae. Recovery is similar to vestibular pathologies such as vestibular neuronitis and benign paroxysmal positional vertigo. Further basic science, clinical, and translational research is needed to verify and understand the short- and long-term effects of COVID-19 on vestibular function.

Introduction

SARS-CoV-2 is part of the *Coronaviridae* family of viruses that cause pathogens in animals and humans [Alluwaimi et al., 2020]. The virion envelope contains at least three structural proteins: membrane, spike, and envelope [Boson et al., 2021]. Its transmission originates mainly from expelling small water droplets when speaking or sneezing within 2 m or encountering exposed surfaces. The replication cycle of SARS-CoV-2 begins with the spike protein and the host cell receptor, binding to structures such as angiotensin-converting enzyme 2 (ACE-2) and the transmembrane protease CD147 [Jackson et al., 2022]. The replicated viral RNA is translated to produce polyproteins, forming a replication-transcription complex. The new virions are assembled by budding at the endoplasmic reticulum and Golgi apparatus, released by exocytosis [Ortiz-Prado et al., 2020].

Since the coronavirus pandemic to date, there have been many case reports, original research, and systematic reviews relating to the impact of SARS-CoV-2 on otoneurological dysfunction [Almufarrij et al., 2020; Aljasser et al., 2021]; however, there have been several comprehensive studies reporting alterations in the auditory system but few in the vestibular system. In addition, information on the most frequent vestibular pathologies associated with coronavirus infection and the results of vestibular evaluations in subjects infected with SARS-

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CoV-2 still need to be fully described and structured. In this review, we want to address the specific evidence between SARS-CoV-2 and vestibular pathologies.

Neurological Manifestations in SARS-CoV-2

There is strong evidence that SARS-CoV-2 can directly infect structures of the central nervous system because the ACE-2 receptor has been found in neurons and glial cells [Ellul et al., 2020; Montalvan et al., 2020; Baig, 2022]. Spread can occur through the neuronal spread, systemic vascular spread, or through the cribriform plate of the ethmoid bone in the early or late phase of infection [DosSantos et al., 2020; Reza-Zaldívar et al., 2021]. The most frequent neurological manifestations in patients with SARS-CoV-2 infection are headache, confusion, dizziness, anosmia, ageusia, cerebrovascular accidents, and reduced consciousness [Hu et al., 2021]. SARS-CoV-2 has strongly impacted otorhinolaryngological care due to alterations in the loss of smell and taste [Pierron et al., 2020]. For some time, it has been reported the affections that this infection would have on the auditory and vestibular systems; however, in the studies associated with vestibular alterations, mainly there have been reports of symptoms (dizziness, tinnitus, or vertigo) in subjects affected by SARS-CoV-2, case reports, or casuistry carried out in hospital centers [Basu et al., 2021; Gallus et al., 2021; Deva et al., 2022].

Vestibular Manifestations in SARS-CoV-2

Although the link between COVID-19 and vestibular alterations is not elucidated, there is much preliminary evidence associating SARS-CoV-2 infection with vestibular pathologies. Along with the auditory manifestations already described in previous studies [Almufarrij et al., 2020; Ricciardiello et al., 2021], some vestibular symptoms such as dizziness, vertigo, and tinnitus are described as the most common in patients with COVID-19. Several studies and self-reports have reported that dizziness is a prevalent neurological symptom after a COVID-19 infection. However, it is difficult to determine whether the dizziness corresponds to vestibular pathology or some other dysfunction, such as the associated symptoms of so-called "post-COVID fatigue syndrome" [Bornstein et al., 2021; Yuan et al., 2022]. These symptoms may persist in more than 70% of the population 3–4 weeks after having COVID-19, and then this percentage decreases over the weeks. The main symptoms are headache, unexplained muscle or joint pain, and nonspecific sensation of dizziness or unsteadiness [Lopez-Leon et al., 2021; Nalbandian et al., 2021]. Due to this last point, it is necessary to separate the cases effectively diagnosed with some vestibular pathology from the studies or self-reports that only described a sensation of dizziness or imbalance. This review investigated the most prevalent vestibular pathologies associated with SARS-CoV-2 infection are vestibular neuronitis, benign paroxysmal postural vertigo (BPPV), and acute vestibular syndrome. There is also a case report of bilateral vestibular dysfunction following SARS-CoV-2 infection. Table 1 shows the studies reporting vestibular pathologies and manifestations in subjects diagnosed with COVID-19.

Otoneurological Findings in SARS-CoV-2 Patients

In this review, we have found eighteen papers reporting some diagnosed vestibular dysfunction (Table 1). The primary pathology described within the reports has been BPPV [Abdelrahman and Shafik, 2021; Maslovara and Košec, 2021; Picciotti et al., 2021; Cetin, 2022] and vestibular neuronitis [Malayala and Raza, 2020; Vanaparthy et al., 2020; Aasfara et al., 2021; Giannantonio et al., 2021; Shinde et al., 2021; Mat et al., 2023]. Some cases of labyrinthitis [Gerstacker et al., 2021; Perret et al., 2021], acute vestibular syndrome [Zaffina et al., 2021], and 1 case of acute bilateral vestibulopathy have also been reported [Lee et al., 2022]. The evaluation and reporting of nystagmus in these reports were scarce: four horizontal cases of spontaneous nystagmus and twelve positional nystagmus (all with peripheral characteristics) were described in these series. In general, there was no further characterization of the rest of vertigo in these patients (most of the studies have been case reports). In terms of vestibular evaluation by objective tests, video-oculography (VOG), video head impulse test (VHIT), vestibular evoked myogenic potentials (VEMPs), and dynamic posturography have been performed. Taha and Ali [2021] characterized the VOG responses in patients diagnosed with COVID-19. This study reported that 8 patients had unilateral caloric weakness. Tan and Deniz [2022] reported alterations in the following evaluations of patients with COVID-19: saccades, optokinetic nystagmus, spontaneous nystagmus, and head shake nystagmus. Lee et al. [2022], Tan and Deniz [2022] reported bilateral vestibular areflexia in a case report of a patient with COVID-19 [Lee et al., 2022]. As for studies using VHIT, we can mention case report studies and some studies

Table 1. Summary of the main papers reporting the relationship between SARS-CoV-2 and vestibular pathologies

Author	Type of study	Type of vestibular evaluation	Vestibular relevant findings	Otoneurological diagnostic
Aasfara et al. [2021]	Case report	VOG	• Left-beating spontaneous nystagmus • Right vestibular areflexia	Bifacial weakness, paresthesia, and vestibulocochlear neuritis
Abdelrahman and Shafik [2021]	Cross-sectional study	• Audiometry • VOG	65% of patients with abnormal positional test 40% of patients with abnormal unilateral weakness	No
Basu et al. [2021]	Retrospective clinical study	• Audiometry • VHIT	vHIT significant The gain was seen in the right anterior canal	No
Gallus et al. [2021]	Retrospective clinical study	• Audiometry • VHIT • SHIMP	Normal VHIT	No
Gerstacker et al. [2021]	Case report	• VOG • OEA • ABR	• Tinnitus is in the left ear and vertigo • Audiometry: the hearing thresholds on the right side of 70 dB between 1 kHz and 4 kHz, and anacusis in the left ear • VOG: under-excitability on the left side	Labyrinthitis probably caused by SARS-CoV-2
Giannantonio et al. [2021]	Case report	• Head impulse test • Nystagmus evaluation	• Spontaneous, horizontal-torsional grade III nystagmus • Altered HIT	Vestibular neuritis
Lee et al. [2022]	Case report	• Audiometry • VOG	Bilateral vestibular areflexia	Bilateral vestibular dysfunction
Magrouni et al. [2022]	Case report	Dix-Hallpike maneuver	Nystagmus with the rapid component to the right	BPPV
Malayala and Raza [2020]	Case report	Only physical examination	Only persistent Vertigo	Probably, vestibular neuritis
Maslovara and Kosec [2021]	Case report	Dix-Hallpike maneuver	Two different patients with right positional nystagmus	BPPV
Mat et al. [2023]	Case report	VHIT	Decrease of the VOR gain and catch-up saccades for the left anterior and lateral semicircular canals	Vestibular neuritis
Perret et al. [2021]	Case report	Clinical and nystagmus evaluation	• Vertigo • Left-beating spontaneous nystagmus	Labyrinthitis, probably caused by SARS-CoV-2 (vestibule, semicircular canals, and cochlear on the right appeared hyperintense on FLAIR images in brain magnetic resonance)
Picciotti et al. [2021]	Retrospective study	Dix-Hallpike maneuver	Eight reported cases of BPPV (five of posterior canal and three of horizontal canal)	BPPV

Table 1 (continued)

Author	Type of study	Type of vestibular evaluation	Vestibular relevant findings	Otoneurological diagnostic
Shindle et al. [2021]	Case report	• HIT • Nystagmus evaluation	Head impulse test: positive gaze-induced nystagmus	Vestibular neuritis
Tan and Deniz [2022]			• VOG: saccades, optokinetic nystagmus, spontaneous nystagmus, and head shake nystagmus altered • VHIT: significant difference in vHIT lateral gain, LARP gain, and RALP gain measurements in both the right and left ears • cVEMP: significant difference between left ear P1 latency and N1 latency • oVEMP: statistically significant difference was found between the mean asymmetry values	No
Vanaparthy et al. [2020]	Case report	• Nystagmus evaluation • Dix-Hallpike maneuver	• Vertigo • Spontaneous nystagmus	Vestibular neuritis
Yilmaz et al. [2022]	Case-control study	• CDP • VEMPS • VHIT	• CDP: general scores of the patients were significantly lower than controls • VEMPS: decreased P1/N1 amplitudes and elongated N1 latencies were found (cVEMPS) • VHIT: gains decreased in the vertical semicircular canals	No
Zaffina et al. [2021]	Case report	• Dix-Hallpike maneuver • Nystagmus evaluation	Subjective vertigo, associated with vomiting and bilateral aural fullness Dizziness during the Dix-Hallpike maneuver on the right side	SARS-CoV-2 infection with neuro-vestibular involvement

CDP, computerized dynamic posturography; VEMP, vestibular evoked myogenic potential; cVEMP, cervical VEMP; oVEMP, ocular VEMP; VHIT, video head impulse test; VOG, video-oculography; SHIMP, suppression head impulse test; OEA, otoacoustic emissions; ABR, auditory brainstem response.

involving more patients [Basu et al., 2021; Gallus et al., 2021; Tan and Deniz, 2022]. On the other hand, using the VHIT technique, the case reports have mainly described an alteration in the gain in the anterior and lateral canals, in addition to the presence of corrective overt and covert saccades [Giannantonio et al., 2021; Mat et al., 2023]. In other studies, a decrease in the gain of the inferior semicircular canal has been mainly described [Basu et al., 2021; Gallus et al., 2021; Tan and Deniz, 2022]. Finally, about the VEMPs and dynamic computerized posturography tests, Yilmaz and collaborators describe the results obtained in 30 patients with COVID-19 using these tests. On dynamic computerized posturography, the composite and visual general scores of the patients were significantly lower than the controls. There was a significant difference between patients and controls in the

absence of ocular VEMPs, while amplitudes and latencies were similar between the groups, whereas in the cervical VEMPs, decreased P1/N1 amplitudes and lengthened N1 latencies were found [Yılmaz et al., 2022].

As for vestibular evaluation, an exhaustive vestibular examination could not be performed early in the pandemic due to sanitary conditions. This meant that an adequate assessment was not obtained with the available battery of tests. Ideally, future studies should contrast the performance of different evaluations (VOG, vHIT, VEMPs, posturography) in the same group of subjects before and after infection with COVID-19. To date, and to our knowledge, only one paper has conducted case-control studies [Tan and Deniz, 2022].

Mechanisms of SARS-CoV-2 Damage in Vestibular Pathologies

The analysis of this review showed that, despite having a low incidence, both auditory and vestibular function could be affected by SARS-CoV-2 infection. Among the potential routes of entry of the SARS-CoV-2 virus into the inner ear, three routes are postulated: (I) central nervous system via the olfactory bulb, (II) via the endolymphatic sac, and (III) by hematogenous dissemination through the *stria vascularis* [Jeong et al., 2021] demonstrated that adult human ear tissue expresses ACE-2 and TMPRSS2 receptors (necessary for the virus entry), which would be its potential mechanism of infection to the vestibular system. According to this same study, three mechanisms of action are proposed for vestibular symptoms in patients with SARS-CoV-2: first, the virus may directly infect and eventually destroy cochlear hair cells, vestibular hair cells, vestibular nerve cells, and primary auditory afferent fibers [Kalcioglu et al., 2020; Mustafa, 2020; Jeong et al., 2021]. Second, direct viral infection of inner ear cells may trigger innate interferon-mediated responses, followed by the release of proinflammatory cytokines. This resulting inflammatory response may result in damage to inner ear cells [Jafari et al., 2021; Jeong et al., 2021]. Third, damage to the *stria vascularis* would alter the endocochlear potential and potassium homeostasis of endolymph potassium within the cochlear duct, leading to hearing loss [Jeong et al., 2021; Mannan Baig, 2021]. Di Mauro and collaborators propose the following mechanisms of action for SARS-CoV-2 damage in the auditory and vestibular systems: (I) inflammation of the cochlea, vestibule, or vestibulocochlear nerve [Lang et al., 2020]; (II) immune response, such as proinflammatory cytokine production and vasculitis events [McGonagle et al., 2021]; (III) antibody or T-cell cross-reactions, which may identify inner ear antigens such as the virus [Bovo et al., 2009]; (IV) endothelial dysfunctions (one of the main pathophysiological processes of SARS-CoV-2 [Otifi and Adiga, 2022]; and (V) vascular disorders in the cochlea and semicircular canals [Trune and Nguyen-Huynh, 2012].

Inflammation due to viral infection is one of the most critical damage mechanisms. It is postulated that the virus could infect and directly damage cochlear hair cells, vestibular hair cells, and primary cochlear and vestibular afferent cells [Gedik et al., 2021; Jeong et al., 2021] and could also trigger innate immune responses or a hematogenous response where the virus could cause the appearance of microthrombi in the inner ear [Jafari et al., 2021; Umashankar et al., 2022]. In this sense, the possibility of a thrombus in the audio-vestibular artery could alter blood flow in both the cochlea and vestibule [Trune and Nguyen-Huynh, 2012]. The microthrombi in the inner ear could produce cochleovestibular infarcts, generating pathologies such as sudden sensorineural hearing loss, vestibular neuronitis, or BPPV.

Another damage mechanism would be the involvement of the immune and circulatory systems. Human studies show that SARS-CoV-2 can spread throughout the body through the circulatory system (due to abundant expression of ACE2 in arterial and venous endothelial cells) and in the arterial smooth muscle cells of many organs. Because of this, the virus can damage the blood-labyrinth barrier and invade the inner ear structure by activating monocytic cells, which can attack the vascular system [Mannan Baig, 2021].

Other mechanisms of damage would correspond to an immunological response or generation by thrombi in the microcirculation of the inner ear [Trune and Nguyen-Huynh, 2012]. However, the pathophysiological mechanisms still need to be studied more extensively in animal models and humans. It is suggested that the presentation of symptomatology could be a specific mechanism or a combination of one or more pathophysiological alterations. Still, studies with more patients are needed to confirm this.

Conclusions

This review concludes that vestibular signs, symptoms, and pathologies would be related to subjects with COVID-19. While more than one mechanism of pathophysiological damage has been proposed for SARS-CoV-2 in the vestibular system, the most obvious hypothesis is that the virus is directly targeting the structures of the inner ear through the ACE-2 and TMPRSS2 receptors present in some tissues

[Jeong et al., 2021]. Most selected studies (except for vestibular bilateral dysfunction and two cases of labyrinthitis [Gerstacker et al., 2021; Perret et al., 2021]) show that vestibular type involvement would be transient. All the evidence collected so far indicates that SARS-CoV-2 would cause transient inner ear involvement that resolves in most cases spontaneously. On the other hand, although these are not the most common symptoms reported in symptomatic or asymptomatic patients, it is suggested to have an index of suspicion for SARS-CoV-2 infection in patients who report the sudden and unique onset of these symptoms. In conclusion, we can state that the vestibular damage generated by COVID-19 would not be permanent and would not leave significant sequelae, as it could recover spontaneously. However, more basic science, clinical, and translational studies are needed to verify and understand the short- and long-term effects of SARS-CoV-2 on the vestibular system.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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Author Contributions

Cristian Aedo-Sanchez: conception and study design, critical review of the literature, drafting of the first version, drafting of the final version; Gabriela Gutierrez: a critical review of the literature, synthesis of the information collected, drafting of the final version; Enzo Aguilar-Vidal: conception and study design, critical literature review, manuscript edition, and drafting of the final version. All authors approve of the final version of the manuscript.

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