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Emerging reports of hearing loss during the COVID-19 pandemic and the importance of accessibility to audiological services

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Abstract:

Covid-19 symptoms range from respiratory distress and fever to completely asymptomatic cases. Several viral infections are known to affect the auditory system and recent case studies propose that SARS-CoV-2 is no exception. Emerging reports indicate the manifestation of both sudden sensorineural hearing loss as well as milder levels of damage during a SARS-CoV-2 infection. Inflammation, ischemia, and thrombosis have, amongst others, been identified as possible causes. Despite the current directives for home isolation and closure of certain services, accessibility to early audiological evaluation and monitoring upon suspicion or detection of sensorineural hearing loss remains essential.

A review of emerging case reports and studies suggests that injuries to structures in and around the peripheral and central auditory systems may occur following the infection of SARS-CoV-2; the virus which causes the response more commonly known as COVID-19. Of interest, is the occurrence of hearing loss and other related deficits in both asymptomatic patients and those exhibiting symptoms related to COVID-19. While these reports and studies do not establish causality, various attributions of cause have been proposed and will be highlighted in this article.

The most apparent observations of hearing loss are from confirmed cases of infection of SARS-CoV-2 along with the timely onset of sudden sensorineural hearing loss (SSHL). In March 2020, Sriwijitalai and Wiwanitkit were early in reporting the occurrent of unresolved SSHL in a patient with COVID-19 related respiratory care and recovery. Similarly, Degen, Lenarz and Willenborg (2020) reported the case of a 60-year old man with reported deafness and loud white noise tinnitus bilaterally. Audiologic testing confirmed anacusis on right side and profound sensorineural loss on left side. MRI results for this patient

were indicative of an inflammatory process in the right cochlea and meningeal contrast enhancement was detected at the base of the right temporal bone. Cochlear implantation was conducted preventatively on the right side in anticipation of ossification. The left ear was treated with intratympanic injections of triamcinolone without any noted results. This patient also received azithromycin and furosemide during hospitalization, both of which have documented ototoxic effects, but could not have caused the pathological results observed with MRI, or the asymmetrical audiologic findings. The authors highlight the association between Covid-19 acute respiratory distress syndrome and encephalopathy, with more than half of all cases showing meningeal contrast enhancement which subsequently can cause sensorineural hearing loss. Koumpa, Forde and Manjaly (2020), also reported a case of SSHL with accompanying tinnitus without abnormal MRI findings. The 45-year old COVID-19 patient was intubated for 30 days to treat bilateral pulmonary emboli and no ototoxic medications were administered. Bedside testing suggested 65-85 dB descending hearing loss with thresholds at 2, 3, 4, and 6kHz being most affected. A 7-day course of oral prednisone followed by intratympanic steroid injection were administered. Partial improvements of thresholds were subsequently observed. Similarly, Lamounier et al. (2020) reported on a case of predominantly unilateral loss with accompanying disabling tinnitus showing some partial low-frequency improvement following the administration of combined oral and intratympanic corticosteroid therapy. As contemplated, delayed therapy has a limited potential of hearing threshold recovery, such as was observed in an unresolved case of unilateral profound high-frequency SSHL with accompanying tinnitus (Lang, Hintze & Conlon, 2020). Their 30-year old patient did not show any improvement following a course of oral prednisone which was commenced seven weeks after the positive result of SARS-CoV-2 infection and three weeks post onset of hearing loss. The authors highlight the importance of a low index of suspicion of SSHL in hopes of providing a time-sensitive recovery with use of corticosteroid therapy.

Interestingly, cases where SSHL is the only condition related to infection of SARS-CoV-2 have also been noted. Rhman and Wahid (2020) reported a 52-year old male patient with a positive SARS-CoV-2 swab who was otherwise asymptomatic but was subsequently referred for sudden sensorineural hearing loss on the left side preceded by a gradually increasing tinnitus. Hearing thresholds revealed a severe sensorineural hearing loss for the left ear. No intracranial abnormalities could be detected with MRI and no other attributable causes for the sudden hearing loss could be identified. Intratympanic injection of methylprednisolone provided a partial recovery of hearing. Suspecting an emergence of SSHL following the SARS-Cov-2 outbreak, and the known viral etiology of SSHL, Kilic et al (2020) performed a polymerase chain reaction (PCR) test on adults who presented to an ENT clinic with the sole complaint of SSHL. One of the five patients was found to be positive for SARS-CoV-2, and otherwise did not exhibit any symptoms of Covid-19.

A widely cited mechanism to explain the manifestation of SSHL revolves around the same receptor to which SARS-CoV-2 binds to enter the body; the angiotensin-converting enzyme 2 (ACE2). SARS-CoV-2 is known to enter the body by binding to ACE2 receptors of the alveolar epithelial cells and endothelial cells of the lungs and from there bind to other areas of the body where ACE2 receptors are found. (Cure & Cumhur Cure, 2020). While it is not known if these receptors are expressed in the human cochlea and whether or not they would permit a direct entry of SARS-CoV-2, they have been found in the mucosal epithelium of the Eustachian Tube and middle ear spaces as well as the cochlea of mice (Uranaka et al., 2020). An ensuing release of inflammatory cytokines would then likely result in oxidative damage of hearing structures. Other possible hypotheses to explain the pathophysiology of SSHL have been proposed, namely, but not limited to, a SARS-CoV-2-induced deoxygenation of erythrocytes leading to the hypoxia of hearing structures, or conversely a thrombosis caused by a change in vascular microstructure leading to ischemic lesions (Cure & Cumhur Cure, 2020).

The direct verification of these proposed etiologies, or others to follow, may shed light on the possible prevalence of SARS-CoV-2-related hearing loss. However, a broader hearing assessment strategy of both symptomatic and asymptomatic cases may also have significant merit. As support of this argument, using transient evoked otoacoustic emissions (TEOAE) which are sensitive to changes of cochlear function, Mustafa (2020), was able to observe differences in cochlear function between asymptomatic individuals, aged 20-50 years, with confirmed infection from SARS-CoV-2 and a control group with normal hearing. Significantly lower TEOAE amplitudes were also accompanied by significantly poorer hearing thresholds at 4, 6, and 8kHz despite no hearing impairment being noted during case history. These results are indicative that SARS-CoV-2 may also cause milder forms of hearing loss than the more apparent cases of SSHL noted above. While the study makes no mention of severity of loss or whether or not there was an asymmetric nature to their results, there is nevertheless indication that milder forms of hearing dysfunction may be measured in individuals infected with SARS-CoV-2. It may also be inferred from these results that hearing dysfunction may be a specific indicator (symptom) of infection for individuals who are otherwise asymptomatic for COVID-19.

As we commence to obtain reports which indicate the potential effects of SARS-CoV-2 on the auditory system, we acknowledge the need for systematic studies on the matter to help establish prevalence as well as other epidemiological data. Also, as part of a broader remedial scope, future studies should also evaluate if milder occurrences of hearing or auditory processing deficits are also prevalent for both symptomatic and asymptomatic cases. In this context, access to audiology services is of substantial value to allow for the adequate surveillance of hearing loss, most notably in cases of suspected SSHL where timely assessments and treatment are often of utmost importance to allow for any recovery of hearing function.

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