Case Report

SAGE Open Medical Case Reports

A case of sudden mixed hearing loss in SARS-CoV-2

SAGE Open Medical Case Reports
Volume 10: 1–5

DOI: 10.1177/2050313X221080642
journals.sagepub.com/home/sco
SAGE

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Abstract

The current global pandemic caused by coronavirus has uncovered multiple symptoms, including sudden hearing loss in either one or both ears, with different outcomes. We present a 68-year-old female with sudden onset bilateral hearing loss, fever, generalized body weakness, and gastrointestinal disturbances. She tested positive for severe acute respiratory syndrome coronavirus 2, was isolated, and was treated with a variety of medications for 5 days before being discharged home to self-isolate. The audiological assessment revealed both sensorineural and conductive hearing loss with a progressive favorable outcome on follow-up visits, with resolution occurring approximately 2 months after the onset. The majority of cases have reported sensorineural hearing loss. However, this case is one of the few that has reported mixed hearing loss. This report highlights an alternate clinical feature of the coronavirus that requires a comprehensive audiological examination by clinicians to ensure proper identification and treatment.

Keywords

COVID-19, SARS-CoV-2, hearing loss

Date received: 25 September 2021; accepted: 27 January 2022

Introduction

The current global pandemic caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) has produced an abundance of published literature regarding the clinical presentation and treatment of the disease. One of the uncommon clinical presentations includes hearing loss (HL). Sudden onset HL is a rapid-onset hearing impairment in either one or both ears. HL is categorized as conductive (CHL), sensorineural (SNHL), or mixed (MHL). CHL is a result of the pathology of the outer or middle ear disrupting sound transmission to the inner ear. SNHL is a result of impaired cochlea, auditory nerve, or central auditory perception or processing function triggered by a viral, autoimmune, or idiopathic cause.[1,2] This report describes one of the few sudden MHLs in a patient with SARS-CoV-2.

Case presentation

A 68-year-old female presented with a sudden onset of HL 3 days before the hospital visit. Four days before the onset of HL, the patient presented with generalized weakness, nausea, and a few episodes of loose stool. A day before the hospital visit, she presented with a fever. There was no tinnitus, ear pain, or ear discharge reported. She did not present with a cough or difficulty breathing. She denied taking any ototoxic medication. A year before the current onset of symptoms, she had reported dizziness, which was linked to uncontrolled hypertension, and after the initiation of antihypertensive medications, her dizziness resolved. She did not report any history of other chronic diseases or head and neck surgery. The family history was negative for HL or dizziness. On examination, she had a body temperature of 38.6°C, a blood pressure of 138/89 mm Hg, a pulse rate of 92 beats per minute, a respiratory rate of 22 breaths per minute, and an oxygen saturation of 91% on room air. The ear, nose, and throat examinations were normal. Lung auscultation revealed bilateral infrascapular crepitations.

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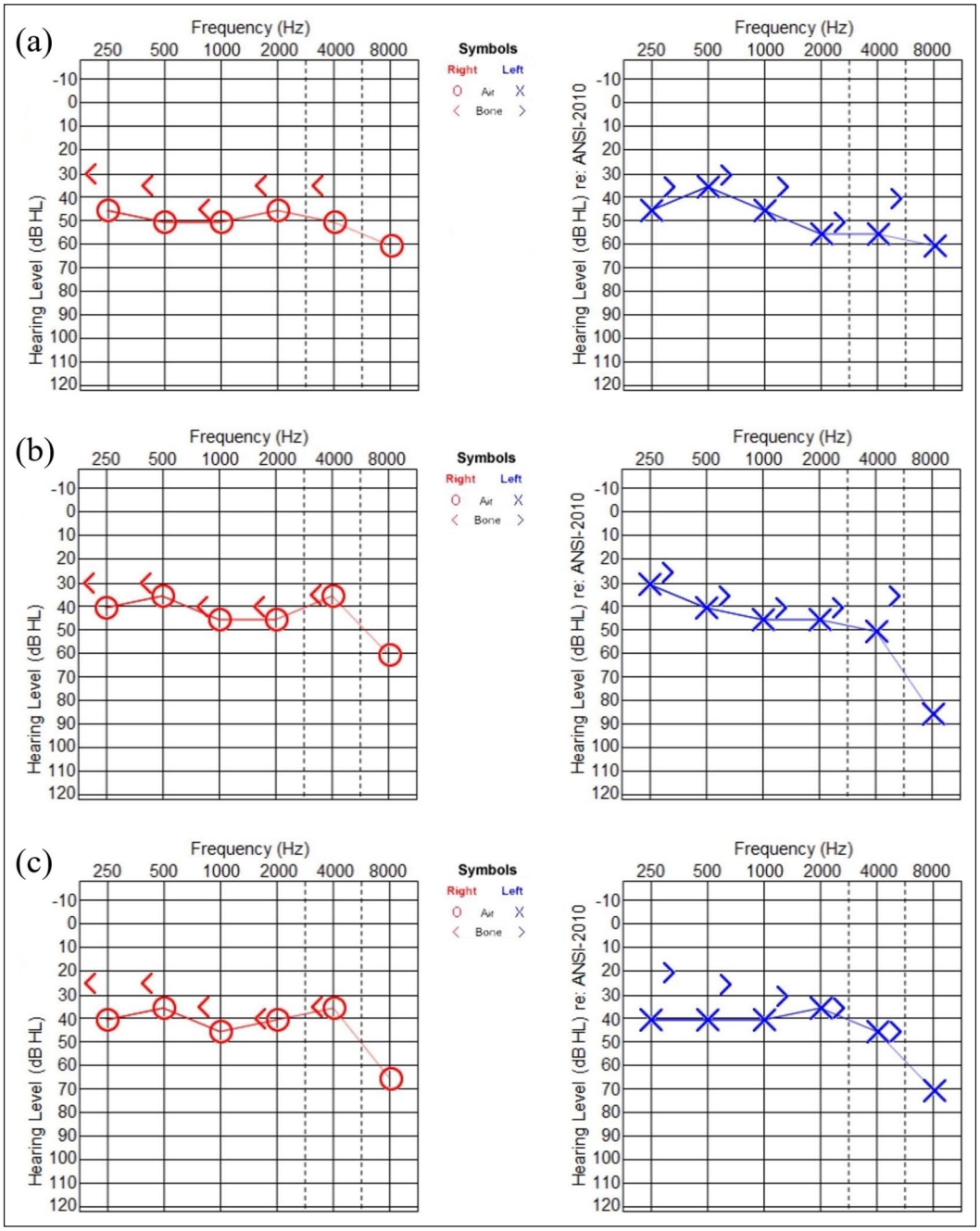

Figure 1. (a) Baseline audiometry on first encounter, SNHL at 1 kHz on the right, 500 Hz and 2 kHz on the left. Rest of frequencies with CHL. (b) Second audiograms after 2 weeks, SNHL at 500 Hz and 8 kHz on the right and 8 kHz on the left. Rest of frequencies with narrow air-bone gap concluding CHL. (c) Third audiograms after 4 weeks, SNHL at 2, 4, and 8 kHz on the right and 2, 4, and 8 kHz on the left. Rest of frequencies with CHL.

A chest X-ray revealed bilateral peripheral opacifications with cardiomegaly, and a diagnosis of pneumonia was established. The patient was isolated and tested for SARS-CoV-2 by reverse transcription-quantitative polymerase chain reaction (PCR) via nasopharyngeal and oropharyngeal swabs. The laboratory findings showed a leukocyte count of 7.4×10^3/L with a neutrophil to lymphocyte ratio of 9. Serum ferritin, C-reactive protein, and D-dimer were 583.1 ng/mL, 19.4 mg/L, and 4.6 mg/L, respectively. Within 72 h, the result came back positive. Once the swab test was taken, she was initiated on oxygen and a multitude of medications over 5 days consisting of intravenous dexamethasone 8 mg qd, intravenous ceftriaxone 1 g qd, subcutaneous enoxaparin 40 mg qd, oral azithromycin 500 mg qd, oral ivermectin 12 mg qd, oral colchicine 0.5 mg qd, oral vitamin C 1 g qd, oral vitamin D 5000 IU qd, and oral zinc 40 mg qd.

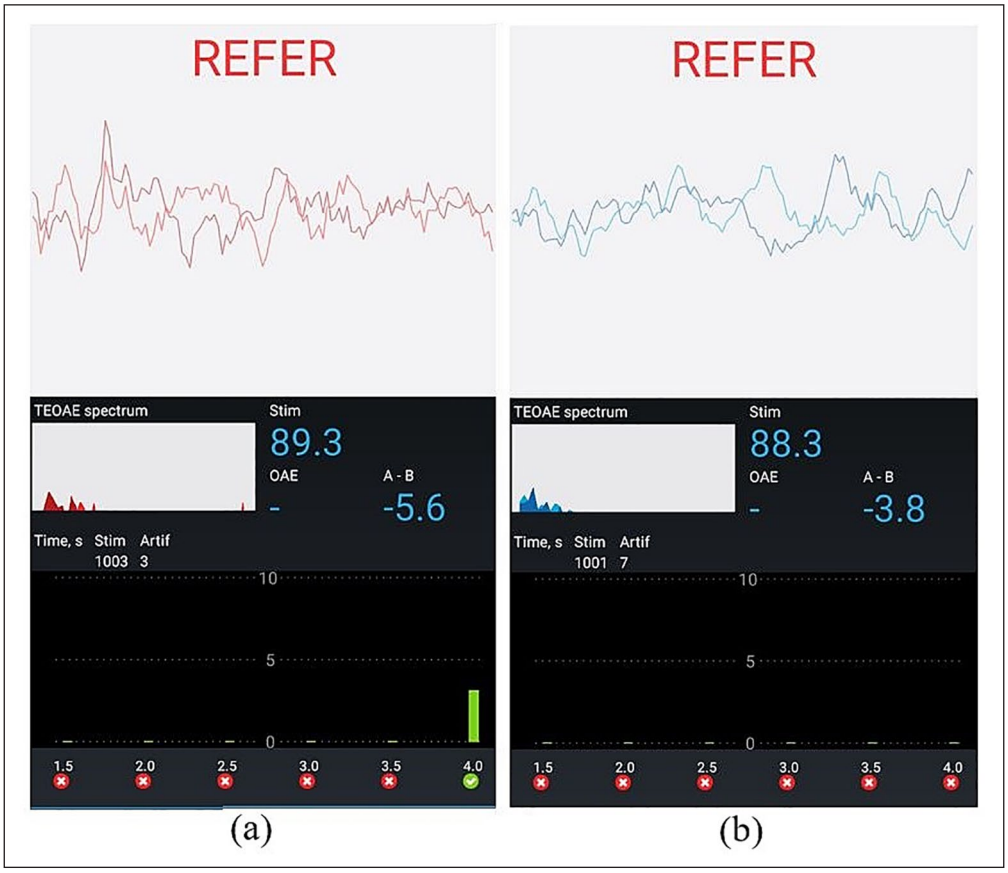

Figure 2. Transient evoked otoacoustic emissions of (a) right ear and (b) left ear quantified bilaterally outer hair cells of the cochlea could not sufficiently respond to stimuli.

Her clinical course of fever and generalized weakness steadily improved, and she was discharged home after a 5-day hospital stay to continue with self-isolation at home. She was reviewed again after 7 days with persistent HL. On otological evaluation, bilateral otomicroscopy revealed mild retraction of the tympanic membrane with an insignificant reduction in mobility, and no lesion or effusion was appreciated. On audiological assessment, pure tone audiometry revealed bilateral moderate MHL, with the left being more affected (Figure 1(a)). On Eustachian tube assessment, flexible nasopharyngoscopy revealed no noticeable pathology in the nasopharyngeal mucosa or at the opening of the Eustachian tube. Computed tomography of the paranasal sinuses revealed no pathology. She was prescribed xylometazoline nasal spray 0.1% tid for a week and fluticasone furoate 100 mcg qd for 2 weeks. She was also encouraged to perform the Valsalva maneuver to encourage the opening of the Eustachian tube and, hence, ventilation of the middle ear.

After a 2-week follow-up, she reported a significant improvement, although she still had some degree of HL. A repeat audiological assessment revealed bilateral MHL (Figure 1(b)). Inner ear assessment by an otoacoustic emission quantified that bilaterally outer hair cells of the cochlea could not sufficiently respond to stimuli, hence the negative test (Figure 2). One month after the initial assessment, there was evidence of MHL persisting (Figure 1(c)). The patient reported gradual improvement of HL with time and reported resolution approximately 2 months after the onset of the SARS-CoV-2 infection.

Discussion

Viruses cause mild to severe HL, either unilaterally or bilaterally. They typically cause SNHL, with the varicella-zoster virus causing most of the acquired HL. Secondary bacterial infection through immunosuppression by the human immunodeficiency virus can lead to CHL.[4] In this case, we suspect the sudden HL may be linked to SARS-CoV-2.

From the published literature, most cases observed SNHL, but Degen et al.,[5] Chern et al.,[6] and Gunay et al.[7] observed bilateral MHL in their patients, as shown in Supplementary Table 1. Proposed criteria may observe the relationship between SARS-CoV-2 and HL, which includes a positive PCR with radiological evidence; HL occurring at the peak of infection; associated audiological signs and symptoms; and ruling out other causes of HL.[8] In this case, a positive PCR and chest X-ray findings were suggestive. However, the HL did not occur at the peak of infection, and some published

articles observed HL occurring without fever or before the peak of infection, as seen in Supplementary Table 1. The audiological assessment of our patient occurred 2 weeks after onset and 1 week after steroid use, which may have diminished other clinical features as differentials were not identified.

In previous theories, hypotheses have been formulated based on direct viral invasion of the cochlear nerve or the fluid space, reactivation of latent viruses within the inner ear tissue, and immune-mediated response.[9] Current theories are based on SARS-CoV-2 gaining entry into erythrocytes and infecting tissues by binding with angiotensin-converting enzyme 2 present in the brain and releasing inflammatory markers that affect the temporal lobe, likewise deoxygenating the erythrocytes, causing hypoxic damage.[3] Another theory is that cochlear ischemia is caused by microvascular thrombosis caused by fibrin formation, platelet and endothelial cell activation due to hypoxemia, and cytokine activation due to septicemia.[10] Likewise, plasma serotonin is a marker of microvascular dysfunction, interrupting vascular blood flow to the cochlea.[11] These eventually disrupt the inner ear, causing SNHL and probably causing HL before or during the peak of infection. However, high-grade fever probably destroys the blood–labyrinth barrier, while an immune-complex reaction may cause HL during the downward slope of infection,[8] resulting in HL occurring during or after the peak of infection.

Corticosteroid therapy for SNHL has an unproven verdict based on the existing randomized control trials as there is insufficient evidence to conclude the treatment is ineffective. As shown in Supplementary Table 1, not all patients with HL improved with steroid therapy. Considering the profound effect on the quality of life, it is best to treat patients with corticosteroids within 2 weeks of presenting symptoms.[2]

The conductive hearing pathway may be affected by ascending infection from the nasopharynx, leading to middle ear effusion.[12] This interrupts the transmission of sound waves from the external auditory canal, eardrum, middle ear, and ossicular chain. The management is pharmacotherapy with or without surgery depending on the cause, as nasal decongestants relieved the inadequate transmission of sound waves in the patient. It is likely that the worsening of CHL due to middle ear effusion and negative pressure or tympanic membrane retraction over the baseline SNHL made the patient more aware of the HL at normal speech levels (40–60 dB). Other cases without baseline SNHL may not be as affected by the short-term CHL associated with middle ear effusion and congestion.

From the multitude of medications that have been used during the pandemic to treat SARS-CoV-2, some have been linked to causing otological symptoms. In this case, azithromycin and ivermectin were used, which have been studied to cause ototoxic effects, which are often reversible once stopped. Although the mechanism is not understood, azithromycin use, even at a standard dose, has been linked to HL, and in some cases, causes irreversible damage.[13] Ivermectin, however, causes disequilibrium, which has been reported as self-limiting.

Conclusion

This case report highlights one of the few cases reported presenting with both SNHL and CHL, with a favorable outcome from the current pandemic and sudden HL. MHL could be an upcoming symptom of SARS-CoV-2, even in mild to moderate manifestations. This case highlights that prompt recognition of uncommon symptoms may allow all patients to receive early treatment. Clinicians must conduct a thorough audiological assessment on all patients who tested positive for SARS-CoV-2 to ensure appropriate identification and treatment.

Acknowledgements

The authors thank the patient and family for their co-operation.

Declaration of conflicting interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Ethical approval

Our institution does not require ethical approval for reporting individual cases or case series.

Funding

The author(s) received no financial support for the research, authorship, and/or publication of this article.

Informed consent

Written informed consent was obtained from the patient for their anonymized information to be published in this article.

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Supplemental material

Supplemental material for this article is available online.

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