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CASE REPORT

Unilateral sudden sensorineural hearing loss in post-COVID-19 patients: Case report

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Abstract

Sudden sensorineural hearing loss (SSNHL) is an otologic emergency. Sensorineural hearing loss needs to be distinguished from conductive when patients present with sudden hearing loss at the primary care level. Prompt diagnosis of sensorineural hearing loss with pure tone audiometry and immediate treatment by an otolaryngologist can improve the hearing outcome. To date, few case reports exist about SSNHL among post-COVID-19 patients, and none were reported in Malaysia. Here, we present two cases of SSNHL in patients after COVID-19 infection. We wish to highlight the association of SSNHL following COVID-19 infection for timely referral towards better audiology outcomes. Permanent hearing loss will lead to another negative impact on the long-term quality of life of COVID-19 patients.

Introduction

Sudden-onset sensorineural hearing loss (SSNHL) is hearing loss of at least 30 dB in at least three consecutive audiometric frequencies that has developed within 72 hours.[1-4] SSNHL is usually unilateral.[5-7] The main causes of SSNHL include viral infections, vascular occlusion, abnormal cellular stress responses within the cochlea, and immune-mediated mechanisms.[8] Sensorineural hearing loss can be distinguished at the primary care level by examination using an otoscope and testing using a 512-Hz tuning fork.[4,9]

COVID-19 is a new pandemic caused by a novel coronavirus, SARS-CoV-2. A wide range of complications following COVID-19 infection have been studied, and primary care physicians are directly involved in the rehabilitation of post-COVID-19 patients. SSNHL is a rare complication and should be identified by the primary care team and referred to an otolaryngologist urgently.[4,9]

Case Presentation

Case 1

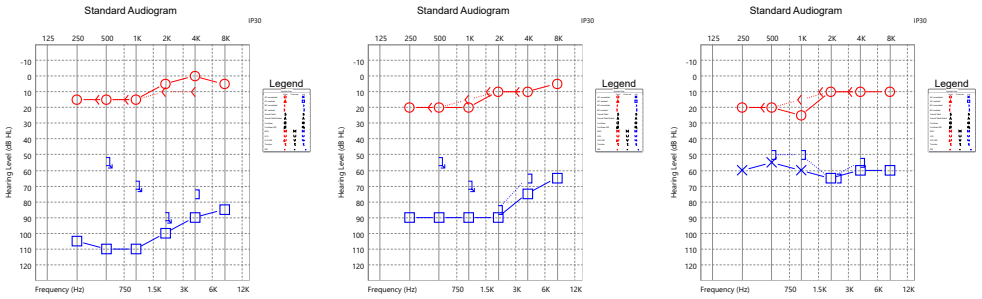
A 35-year-old Chinese gentleman presented to the health clinic 8 weeks after contracting COVID-19. He had tested positive for COVID-19 by reverse transcription polymerase chain reaction. He was asymptomatic during the initial diagnosis and classed as Category 1. He had anosmia on day 5 of his illness. He completed his home quarantine with no complications. He presented with sudden hearing loss in the left ear, which had occurred 3 days prior, on awakening. This was associated with vertigo and left aural fullness. He denied any history of trauma, upper respiratory infection, exposure to loud noise, recent air travel or taking any regular medication before the onset of hearing loss. He had worked as an engineer in the same factory for 11 years. He had no history of smoking or other substance use.

On examination, the patient was afebrile and normotensive with a regular pulse. Otoscopic examination revealed intact tympanic membranes. Bedside testing showed a positive Rinne's test over the affected ear, with Weber's test lateralised to the opposite ear. His gait was normal with no cerebellar sign. The Dix–Hallpike manoeuvre was performed, which was negative. He was referred urgently to the ENT department for further evaluation and treatment.

The patient proceeded with an audiology assessment. Tympanometry was normal. Pure tone audiometry (PTA) showed a unilateral profound sensorineural hearing loss on the left side (**Figure 1A**). He was treated with oral prednisolone 1 mg/kg/day (60 mg daily) for 10 days in a tapering dose, betahistidine 24 mg twice daily and mecobalamin 500 mcg thrice daily. He was scheduled to be reviewed after 10 days along with PTA. However, his vertigo worsened and he was admitted

to a private hospital, where investigations were carried out to look for the cause of SSNHL. A baseline blood investigation was performed, and the result was unremarkable. Autoimmune screening and VDRL were negative. A brain MRI showed no neurovascular lesion or tumour. The facial and vestibulocochlear nerves were normal. He was additionally given tanakan 40 mg thrice daily on discharge.

During follow-up, distortion product otoacoustic emission was performed, and the report showed reduced emission of the left ear. Subsequently, his hearing showed improvement clinically (**Figures 1B and 1C**).



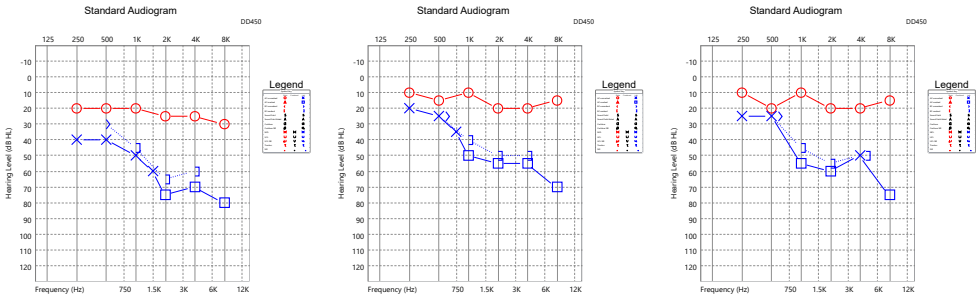
A B C

Figure 1. Interval audiograms. A. Initial on post-COVID-19 day 56. B. Day 17 of SSNHL (post-COVID-19 day 72). C. Day 39 of SSNHL (post-COVID-19 day 94).

Case 2

A 45-year-old Malay gentleman who had tested positive for COVID-19 by RTK-Ag (swab test) was admitted to the Low-Risk COVID-19 Quarantine & Treatment Centre for 4 days for breathlessness and classed as Category 2. On post-COVID-19 day 20, he presented with a new onset of left otalgia, vertigo and vomiting for 4 days. He also had left ear discharge, aural fullness and sporadic fever for 2 days. His symptoms did not improve with oral and topical antibiotics taken from a GP clinic. Before COVID-19 infection, he had been systematically well. On examination, his vital signs were within normal ranges. Left tragal tenderness was observed, with an absence of mastoid or pinna tenderness. Otoscopic examination revealed pus discharge at the left ear, and the tympanic membrane was not visible. The nose and throat examinations were unremarkable. He was referred to the otolaryngology department as left otitis media and reviewed on the same day by the on-call medical officer. Left ear toileting was performed, and otoscopic examination revealed a vesicle at the left pinna. The patient was treated as left otitis externa with a differential diagnosis of herpes zoster oticus and discharged with oral antibiotic and antiviral medication.

At follow-up after 1 week, his symptoms had slightly improved, but he had developed left-sided facial weakness and left ear tinnitus 2 days before the appointment. The assessment of facial nerve function on the left side was House–Brackmann Grade II. Audiologic assessment showed a normal tympanogram bilaterally, and PTA showed left moderate-to-severe sensorineural hearing loss (**Figure 2A**). The diagnosis was revised to left Ramsay–Hunt syndrome (RHS). Oral acyclovir was continued with the addition of Neurobion 1 tablet daily and oral prednisolone 30 mg twice daily for 1 week. During follow-up, he was given another course of oral prednisolone 30 mg twice daily for 1 week, then tapered over 9 days. Subsequently, he was treated with intratympanic dexamethasone 0.5 mL. Little clinical or audiologic improvement was seen after 2 months, although the left facial nerve palsy and vertigo had resolved.



A B C

Figure 2. Interval audiograms. A. Initial on day 7 of SSNHL (post-COVID-19 day 27). B. Day 18 of SSNHL (post-COVID-19 day 38). C. Day 48 of SSNHL (post-COVID-19 day 68

Discussion

Hearing loss following COVID-19 infection is not fully reviewed, despite this symptom having been seen in patients with both COVID-19 and influenza.[1] A systematic review revealed a direct association between COVID-19 and SSNHL, as in other viral infections. It postulated three pathophysiology mechanisms:

(1) Coronavirus could damage the auditory centre in the temporal lobe.
(2) Coronavirus could change the microvascular structure functions of the inner ear, resulting in a thrombus or an embolus that interrupts the blood flow to the inner ear.
(3) Any multi-organ involvement could occur, or a direct peripheral injury could occur to the sensory cells of the cochlea due to neurotropism of coronavirus.[6]

Kilic *et al.* reported five patients with SSNHL as the sole symptom of COVID-19 infection and highlighted that it could be a non-specific symptom of this viral infection.[10] COVID-19 infection has been shown to cause delayed effects on both the central and peripheral nervous systems via an immune-mediated mechanism.[11] Several cases have reported unilateral as well as bilateral SSNHL in post-COVID-19-infected patients.[12-15] A case report suggested screening for hearing loss for inpatients to avoid missing the treatment window.[1] The latest CPG recommended corticosteroids may be offered to patients presenting within 2 weeks of symptom onset. Treatment decisions, including the mode of administration of steroids, are made based on shared decision-making with patients after benefits–harm assessment.[4]

Though both patients presented with vertigo, benign paroxysmal positional vertigo and vestibular neuritis had been ruled out in the presence of sensorineural hearing loss.[16] Vestibular neuritis occurs due to a viral or post-viral inflammatory disorder affecting the vestibular portion of the cranial nerve VIII. The patient will present with severe vertigo, nausea, vomiting and gait imbalance. When these symptoms are combined with auditory problems, it is called labyrinthitis.[17] Labyrinthitis following COVID-19 infection has been reported, and the use of corticosteroids is controversial because it is a self-limiting condition.[16,18]

A systemic review revealed the association of audio-vestibular disorder with new SARS-CoV-2 infection, which manifested as sensorineural hearing loss, vertigo and tinnitus.[14] However, audio-vestibular dysfunction is a rare consequence of COVID-19 infection.[16]

We have presented two relatively young patients with no known comorbid nor otological pathology who developed SSNHL after COVID-19 infection. Our first case was a Category 1 COVID-19 patient who developed SSNHL 8 weeks following COVID-19 infection. All investigations looking for the cause of his SSNHL were negative. Hence, the association of COVID-19 infection causing the SSNHL is likely. Prompt treatment with oral prednisolone showed improvement in this case.

Our second case developed Ramsay Hunt syndrome (RHS), which is also known as herpes zoster oticus, following COVID-19 infection. RHS is caused by the reactivation of the varicella zoster virus (VZV) in the geniculate ganglion of cranial nerve VII. It is characterised by a classic triad of auricular pain, ipsilateral facial nerve palsy and vesicles at the auditory canal. Three types are described based on the complexity of the clinical presentation. Type I manifests as

otalgia and herpetiform rash. Type II adds ipsilateral peripheral facial palsy to Type I. Type III, the most severe form, which was seen in our second case, affects the ipsilateral vestibulocochlear nerve along with Type II manifestations.[19] A study has shown that VZV has around a 5 times higher risk among COVID-19 patients, though the exact pathophysiology is yet to be elucidated.[20] However, RHS is rarely reported.[19]

Conclusion

SSNHL is an otologic emergency similar to stroke and myocardial infarction in primary care. Hence, awareness of SSNHL following COVID-19 infection should prompt primary care physicians to refer patients with SSNHL, especially post COVID-19 infection, to a tertiary centre for audiology assessment. Early diagnosis and intervention may be possible in improving the hearing outcome.

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Conflicts of interest

The authors report no conflict of interest.

Patients' consent for use of images and content for publication

Written consent was obtained from both patients.

How does this paper make a difference to general practice?

- Sudden sensorineural hearing loss can occur following COVID-19 infection.
- Urgent referral of post-COVID-19 patients who present with hearing loss or herpes zoster oticus for audiology assessment and treatment by an otolaryngologist is crucial.

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