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(CASE STUDY)

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Sudden sensorineural hearing loss as a sequela after recovery from post-COVID pneumonia on an atherosclerotic patient: A single case study

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Abstract

It has recently been discovered that COVID-19 is a major contributor to ear disorders and hearing loss such as high-frequency hearing loss and sudden sensorineural hearing loss (SSNHL). Several causes of SSNHL are cited in the literature, including loud noise, ototoxicity, trauma, metabolic diseases, and viral infections. SSNHL could be sequelae of post-COVID-19 infections in people who have metabolic problems or chronic illnesses and this could be ruled out in previously normal hearing-healthy persons. A 57-year-old male reported to the department of Audiology with the complaint of sudden commencement of reduced hearing sensitivity & ringing sensation in his left ear 2 weeks after a post-COVID history of pneumonia. The onset of hearing loss was sudden and progressive. The client's medical history revealed that he has CAD -Acute inferio posterior wall Mayo cardial Infarction and has had H/o hyperlipemia for the previous 10 years. A battery of audiological tests was administered over the course of a year, and a diagnosis of severe unilateral sensory neurological hearing loss was made. We attempt to understand the potential mechanism of sudden hearing loss in post-COVID pneumonia patients with atherosclerosis through this single case study. SARS-CoV-2-induced inflammation may influence atherosclerotic plaques which cause prothrombotic alterations in the blood and endothelium of the cochlear region as a result of COVID-19 infection. During COVID pneumonia attack increased blood viscosity with atherosclerosis of the cochlear vessels reduces the blood perfusion of the cochlea and results in hearing impairment.

Keywords: COVID-19; Atherosclerosis; Sudden hearing loss; Hyperlipidemia

1. Introduction

The coronavirus disease 2019 (COVID-19) epidemic was deemed a global pandemic by the World Health Organization (WHO), which can occasionally be lethal [1]. The respiratory system is the one that is most frequently impacted by the virus, along with the cardiovascular, renal, neurological, and audio-vestibular systems [1]. Viral infections have been associated with hearing loss for a long time, with different viruses having various effects [2]. The virus may directly infect the inner ear or impact the auditory center in the temporal lobe, disrupting the auditory system [3, 4]. Sudden sensorineural hearing loss (SSNHL) is most often defined as a sensorineural hearing loss of 30dB or greater over at least three contiguous audiometric frequencies occurring over 72 hours [5]. Several causes of SSNHL are cited in the literature, including loud noise, ototoxicity, trauma, metabolic diseases (hypertension, diabetes, and hyperlipidemia), and viral infections [6]. Several case studies have documented post-coronavirus disease sudden sensorineural hearing loss (SSNHL) and its adverse effects on hearing mechanisms [7, 8]. Knowing the pathogenesis of COVID-19-induced SSNHL is crucial because it gives the clinician the best alternatives for treating hearing loss while reducing adverse effects. SSNHL could be sequelae of post-COVID-19 infections in people who have metabolic problems or chronic illnesses and this could be ruled out in previously normal hearing-healthy persons [9]. The potential effects of the COVID-19 virus infection on chronic sickness conditions such as atherosclerosis are discussed in the current case study.

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The audiological profile of a patient with CAD-Acute infero posterior wall Mayo cardial Infarction following COVID-19 infection is shown here as a single case study.

2. Case presentation

2.1. History

A 57-year-old male reported to the department of Audiology with the complaint of sudden commencement of reduced hearing sensitivity & ringing sensation in his left ear 2 weeks after post-COVID history of pneumonia. The subject also had complaints of difficulty in understanding speech both in quiet and with noise in the left ear. The onset of hearing loss was sudden and progressive in nature as reported by the client. According to the client's medical history, he has CAD (Coronary artery Disease)-Acute inferio posterior wall Mayo cardial Infarction (coronary artery disease/atherosclerosis) and has had H/o hyperlipemia for the previous 10 years. The client is under anti-coagulation medications (statins group of drugs) for CAD for 1.5 years. During the COVID infection, the client had taken self-prescribed paracetamol and amoxicillin along with previous drugs till the symptoms subsides. The client reported to the audiology clinic after two weeks of mandatory home quarantine.

2.2. Audiological Assessment

The routine audiological evaluation which included pure tone and speech audiometry with immittance evaluations was performed on the client. Bilateral DPOAEs were also carried out to check the OHC functioning. The audiological evaluation was carried on successive intervals between February 2021 to 2022. An audiometric test was performed utilizing a MAICO MA 42 clinical audiometer and supra-aural headphones. The result showed that the left ear had a minimal degree of hearing loss with >90 % speech identification scores and the right ear had a moderately severe degree of hearing loss with 70% speech identification scores, and >100 dBHL uncomfortable levels in both ears (figure 1).

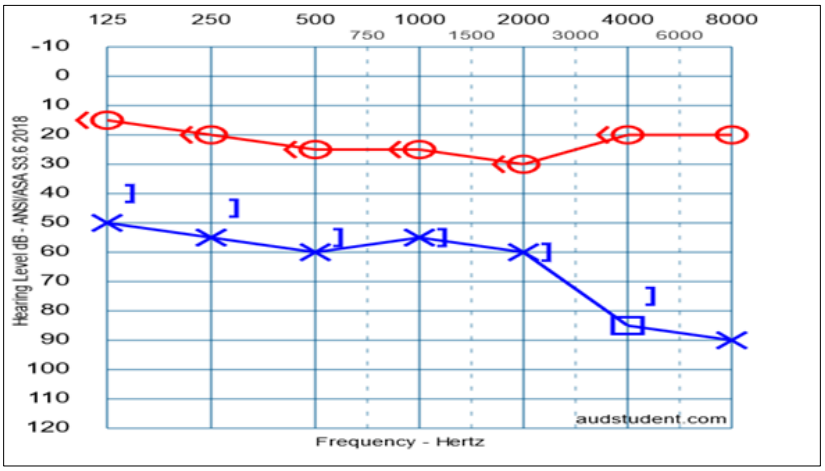

Figure 1 Audiogram of the client during the initial evaluation on 18/03/2021

A GSI Tympstar pro instrument was used to assess immittance, and the results revealed "A" type tympanogram for both ears, with ipsilateral reflexes in the right ear. As a result, a provisional diagnosis of minimal hearing loss in the left ear and moderately severe sensorineural hearing loss in the right ear was made. Further, MAICO ERO-SCAN was used to measure bilateral DPOAEs to check outer hair cell function; the absence of OAEs in the left ear indicated OHC dysfunction. Retro cochlear involvement was ruled out using Tone Decay Tests (TDT) and Supra Threshold Adaptation Tests (STAT). The client was recommended for a hearing aid trial in the right ear, an ENT consultation, and a follow-up after 1 month. A consultation with an ENT specialist found that his bilateral tympanic membranes appeared to be normal, and he refused to undergo steroid drug therapy because he was currently seeking treatment for cardiovascular disease.

The audiological evaluation was repeated on his second visit, 4 months after post-COVID pneumonia and the provisional diagnosis of the client remained the same, he was fitted with ReSound Enya 2 CIC hearing aid in the left ear. A third follow-up visit was scheduled after 6 months and the result revealed a significant change in the hearing thresholds of the left ear. Pure tone thresholds were significantly worsened to 82 dBHL with 60% SIS & >100 dBHL UCL levels (figure 2). Hence the provisional diagnosis became a severe sensorineural hearing loss.

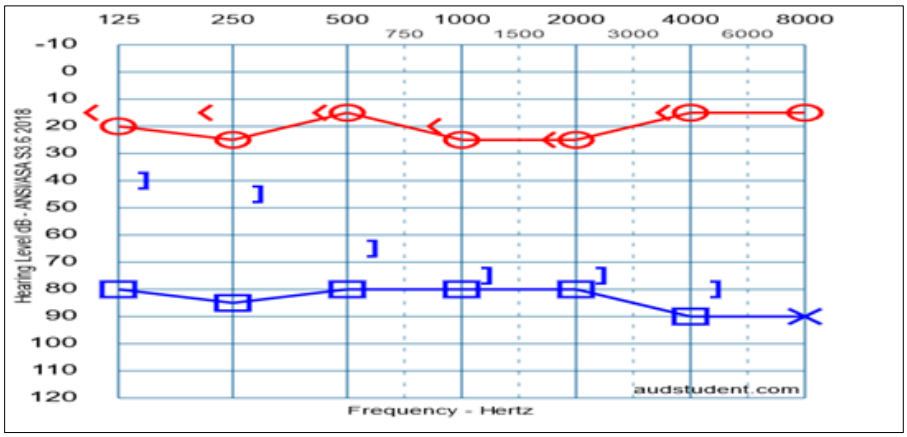

Figure 2 Follow-up Audiogram was done on 09/02/22

3. Discussion

Sudden sensorineural hearing loss with chronic illness is being reviewed in the literature still its pathophysiology is ambiguous [10,11]. We attempt to understand the potential mechanism of sudden hearing loss in post-COVID pneumonia patients with atherosclerosis through this single case study, despite the fact that SSNHL is a rare complication of post-COVID 19 in individuals who had previously normal hearing. Our client has a history of hyperlipidemia, which over time led to atherosclerosis. Hyperlipidaemia is a common metabolic disorder and one of the risk factors for cardiovascular disease by promotes the development of atherosclerosis, non-ischemic heart failure, systolic function, and cardiac electrophysiological responses [12]. Atherosclerosis is defined as an inflammatory process occurring as a response to the accumulation of lipids within the arterial wall [13]. In elderly persons, carotid atherosclerosis is linked to worse hearing and the associations are mostly discovered unilaterally [14].

Inadequate blood and oxygen supply brought on by arteriosclerosis, which reduces blood flow of the labyrinth arteries to the stria vascularis, blocks adenosine triphosphate (ATP) consumption of the Na+-K+-ATP enzyme in the stria vascularis. As a result, the endo-cochlear Potential (EP) could not be maintained and was sharply reduced [15]. The decrease of EP leads to ciliary cell death and decreased hearing sensitivity [16]. The cochlea's apical regions, where low-frequency sound is conveyed and the blood supply is farthest away, are particularly susceptible to ischemia [17]. SARS-CoV-2-induced inflammation, may influence atherosclerotic plaques, generate prothrombotic changes in the blood and endothelium, and ultimately cause their instability, which results in myocardial infarction (MI) [18].

Many pieces of evidence suggest that, at least in some patients, vascular atherosclerotic alterations may play a significant role in the pathogenesis of SSNHL [19]. Our client, who has been dealing with myocardial infarction for the past 1.5 years, was at risk of experiencing sudden sensory neural hearing loss, which was aggravated by COVID 19 infections. During COVID pneumonia attacks increased blood viscosity with atherosclerosis of the cochlear vessels reduces the blood perfusion of the cochlea and results in hearing impairment [20]

4. Conclusion

The client in this study had coronary artery disease/atherosclerosis with hyperlipidemia and was under medication for the past 1.5 years. The client experienced sudden hearing loss in the left ear followed by post COVID pneumonia. The etiology and pathophysiological mechanism of SSNHL have not been completely clarified. Local and systemic factors such as metabolic disease, viral infection, and vascular disease may cause SSNHL. In this case, the onset of hearing loss was reported after the viral attack which could result in microcirculation disorders in the auditory systems. Also, immediate steroid drug therapy could not be administered as the client was reported to the audiology department after mandatory home quarantine. Hence, sudden unilateral hearing loss in the left ear could be caused by atherosclerotic plaques, which cause prothrombotic alterations in the blood and endothelium of the cochlear region as a result of COVID 19 infection.

Compliance with ethical standards

Acknowledgments

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Disclosure of conflict of interest

The authors affirm that they have no known financial conflicts of interest or close personal ties that could have impacted the research presented in this study.

Statement of ethical approval

Non-invasive methods were used to complete the study which adhered to the "AWH ethical committee, compiled with the Declaration of Helsinki"

Statement of informed consent

The individual who was a part of the study provided his informed consent.

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