

CASE REPORT

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Audio-vestibular manifestations in Kawasaki disease (KD): a rare atypical case presentation

R. Mayuri Rao, E. R. Rajils Raveendran, K. Nidha, K. Namitha, P. Prashanth Prabhu and K. V. Nisha*

Abstract

Background: Kawasaki disease (KD) is an acute febrile, usually self-limiting illness of infancy and childhood, that can show audiological manifestation of sensorineural hearing loss (SNHL, 36% of patients), usually bilateral in nature.

Case presentation: We report a 12-year-old female diagnosed with KD at 6 years, showing atypical manifestation of asymmetrical SNHL since 1.5 years after the episode of febrile illness. The patient was administered Immune Globulin intravenously and aspirin, which were tapered to a low dose. The patient later reported reduced hearing sensitivity, ear pain, blocking in both ears, and tinnitus (intermittent) in the left ear. The audio-vestibular manifestations in the case using test battery approach (Pure tone audiometry, speech audiometry, tympanometry, reflexometry, otoacoustic emissions, auditory brainstem response, and behavioral and objective vestibular tests) is discussed. The rehabilitative measures taken, and the need for sensitization of school teachers for early identification of hearing loss in children with KD is highlighted.

Conclusions: This case presentation concentrates on the rare asymmetrical SNHL and vestibular finding post-KD and emphasizes the need for health care professionals to refer patients with KD to an audiologist to trace the possible sequelae including hearing loss and vestibular disorders. The atypical manifestations of KD in hearing domain is suggestive of the need for early identification, adequate profiling of not just hearing but also balance-related manifestations, instigation of appropriate rehabilitative measures, effective counselling of parents and school teachers to facilitate better academic learning, and communication opportunities.

Keywords: Kawasaki disease (KD), Auditory tests, Vestibular tests

Background

Kawasaki disease (KD) is an acute febrile, usually self-limiting illness of infancy and childhood that affects the small- and medium-sized blood vessels. It was first described in 1967 by Kawasaki in Japan [1]. The predominance of Kawasaki disease is more in children and over 75% of cases occur in children less than 5 years of age [2, 3]. Incidence of Kawasaki disease is 4 to 216 per 100,000 children less than 5 years of age worldwide [3, 4]. Diagnosis of Kawasaki disease is purely clinical and there is no specific diagnostic test available to diagnose it. So, it

is based on persistent fever for at least 5 days and further four out of five diagnostic criteria: limb changes, such as hand and/or foot erythema or swelling during the acute stage and fingertip scaling over convalescence; polymorphous exanthema; bilateral conjunctivitis with no purulent discharge, lip erythema and cracking, raspberry tongue, throat redness and anterior cervical adenopathy with a 1.5-cm or larger size [5].

The exact cause of KD remains unknown. According to Goodman and Fuller [6], the current theory is that children may be born with genetic predisposition to KD. On environmental triggering, they can develop into a syndrome. However, the triggers remain to be unknown. It is likely to be a virus or unknown infectious agent which may trigger KD. When children develop KD, the arterial walls become inflamed. The endothelium protects

*Correspondence: nishakv@aishmysore.in

Department of Audiology, All India Institute of Speech and Hearing, Manasagangothri, Mysuru 570 006, India

the blood vessel wall thereby preventing the blood cells from leaving. KD causes the endothelial cells to become activated and causes cell of immune system to stick to the endothelium and leave through the blood vessel wall. Inflammation in the vessel wall is caused by immune cells gathering in the wall and liberating chemical signals that recruit more cells and further damage the vessel wall. If this inflammation is treated late, damage to the arterial wall can be permanent. Sometimes, this causes ballooning of the wall called an aneurysm which increases the risk of blood clot formation which can be prevented with low dose aspirin and other medications. If the blood clot continues to grow, this may result in blockage of blood vessel and heart attack. In many cases, scar tissue will begin to form in aneurysm over time and the blood vessel will heal. However, the blood vessel is never completely normal and after many years of KD, scarring continues to form resulting in narrowing of blood vessel.

The audiological manifestations of the KD are scanty. Out of the few reports available in literature [7], bilateral sudden sensorineural (SNHL) is a rare complication of Kawasaki Disease. Knott et al [8] reported 13 cases (out of 62 patients) who developed SNHL over the first 30 days of onset of KD, while two patients (out of 36) developed SNHL in a second audiological assessment 10 days later. The bilateral SNHL in previous studies can be reflective of the vascular involvement of the cochleovestibular artery supplying blood to the inner ear, although anatomical evidence linking same has to be established. In the present study we report a 12-year-old adolescent female, who exhibited atypical post-complication of KD with asymmetrical bilateral SNHL and discuss the audio-vestibular profile of her.

Case presentation

Present complaints

A 12-year,10-month-old adolescent female diagnosed with KD at the age of 6 years reported to the Out Patient Department at All India Institute of Speech and Hearing, Mysore, India, on October 22, 2021, with the complaints of reduced hearing sensitivity, ear pain, and blocking sensation in both the ears. Additionally, the patient also reported tinnitus (Intermittent and ringing type) in the left ear since 1.5 years.

Case history

A detailed case history was obtained with the child's caregiver regarding the prenatal, perinatal, and post-natal history. Post-natal history revealed birth asphyxia and delayed birth cry. The child was kept in neonatal intensive care unit (NICU) for 4–5 h (less than a day). Difficulty in understanding speech in noisy situations and frequent cold were the problems as reported. History of

bilateral ear discharge with a frequency of 2–3 times/year at age of 5 years was reported. The ear discharge was diagnosed as secretory otitis media, which was treated using antibiotics (amoxicillin). History of sudden and fluctuating spinning sensation after the attack of KD was reported with one episode of vertigo lasting for 10–20 s with the frequency of occurrence of 1/2 times per week, at the age of 11 years. At the time of audiological assessments, child did not complain of any giddiness episodes.

Medical reports

The medical history includes the diagnosis of KD with no coronary artery involvement, at the age of 6 years. The clinical features observed during the attack of KD consisted of lymph node enlargement, bilateral cervical lymphadenopathy, congested oral cavity, and skin peeling of the palm. Hence, the child was treated with intravenous immune globulin (IVIG) and aspirin tapered to low dose, as per the recommendations of pediatrician, to treat KD at onset.

Reports by allied medical professionals. Speech and language as well as psychological evaluations results were found to be normal. The examination of the ear by an otorhinolaryngologist revealed a clear external auditory canal with an intact tympanic membrane and cone of light being visualized in both the ears.

Audio-vestibular profiling

A detailed audio-vestibular profiling was performed to assess the status of outer, middle, and inner ear. The audio-vestibular test battery comprised of Pure tone audiometry, speech audiometry, tympanometry, acoustic reflexometry, otoacoustic emissions (OAEs), auditory brainstem response (ABR), and vestibular tests (ocular VEMP (oVEMP), cervical (cVEMP), and vestibulogram).

Immittance audiometry

A calibrated immittance equipment (Inventis Clarinet, Inventis Inc., Padova, Italy) was used to evaluate the middle ear functioning. Tympanometry was done bilaterally using a probe tone of 226 Hz. Ipsilateral and contralateral acoustic reflex threshold measurement at 0.5, 1, 2, and 4 kHz was carried out in both the ears. The results revealed 'As' type tympanogram [9] with absent acoustic reflexes bilaterally with tympanometric peak pressure of 11 and – 3 daPa, static admittance of 0.30 and 0.31 mmho, ear canal volume of 0.66 and 0.71 cm³ in right and left ear, respectively. This indicated a possible middle ear pathology in both ears.

Pure tone and speech audiometry

Pure tone and speech audiometry were performed in a sound treated room with ambient noise level in

adherence to ANSI S3.6-2018. Pure tone threshold, Speech detection/recognition threshold, speech identification score, speech perception in noise and uncomfortable loudness level were estimated using a calibrated audiometer, Inventis Piano (Inventis Inc., Padova, Italy). Air conduction and bone conduction thresholds were determined with Telephonics dynamic supra-aural Headphones-49 (Telephonics, Farmingdale, NY, USA) and Radioear B-71 bone vibrator (RadioEar New Eagle, PA, USA) respectively. The AC and BC thresholds were tracked using modified Hughson-Westlake method [10] at octave frequencies from 0.25 to 8 kHz and 0.25 to 4 kHz respectively. Pure tone thresholds indicated a 4 frequency pure tone averages (0.5, 1, 2, 4 kHz) being 91.25 and 33.75 dB HL in the right and left ear respectively. The degree of hearing loss was inferred from the average of hearing thresholds at 0.5, 1, 2, and 4 kHz and interpreted using Clark classification [11]. The air and bone conduction thresholds across octave frequencies is shown in Fig. 1. Based on the audiometry, child was diagnosed as profound mixed hearing loss in the right ear and mild mixed hearing loss in the left ear. Conductive component seen in both ears in the child can point to unresolved middle ear issues, which were reported at the age of 5 years (discussed in the case history).

Speech audiometry was carried out using Malayalam wordlist (Spondee wordlist and PB word list) and speech reception threshold (SRT) in the left ear was found to be 40 dB HL. Speech Identification Score (SIS) was found to be 100% in the left ear. Speech perception in noise (SPIN) scores were 68% at 0 dB SNR and 72% at 5 dB SNR in the

left ear. Speech detection threshold (SDT) in the right ear was found to be 90 dB HL.

Otoacoustic emissions (OAEs)

Transient evoked otoacoustic emissions (TEOAEs) and Distortion product otoacoustic emissions (DPOAEs) were recorded using Otodynamics ILO V6 DP Echoport (Otodynamics Ltd., Hatfield, Herts, England). TEOAEs were recorded for each ear separately for non-linear click trains presented at 80 dB peak equivalent SPL. On other hand, DPOAE was done across the frequency band of 1000 Hz to 6000 Hz with F2:F1 ratio being 1.22:1 and constant stimulus intensity level of L1 = 65 dB SPL and L2 = 55 dB SPL. For TEOAE's/DPOAE's to be considered as present, a signal-to-noise ratio of at least 6 dB at 3 adjacent frequencies with wave reproducibility of > 80% is required. Both TEOAEs and DPOAEs were absent bilaterally across all the frequencies, suggestive of bilateral outer hair cell dysfunction.

Auditory brainstem response (ABR)

Natus Biologic Navigator pro (Natus Medical Incorporated, San Carlos, CA, USA) Auditory Evoked Potentials (AEP) system using Eartone 3A insert phones (Etymotic Research, Elk Grove Village, IL, USA; electrode impedance < 5000 Ω) was used to perform ABR testing. ABR threshold estimation using click stimuli in rarefaction polarity at 11.1 s⁻¹ stimulus rate with 90 dB nHL as the starting intensity level was carried out for threshold estimation. ABR site of lesion was done at 11.1 s⁻¹ and 90.1 s⁻¹ stimulus repetition rate. High pass filter setting of 30

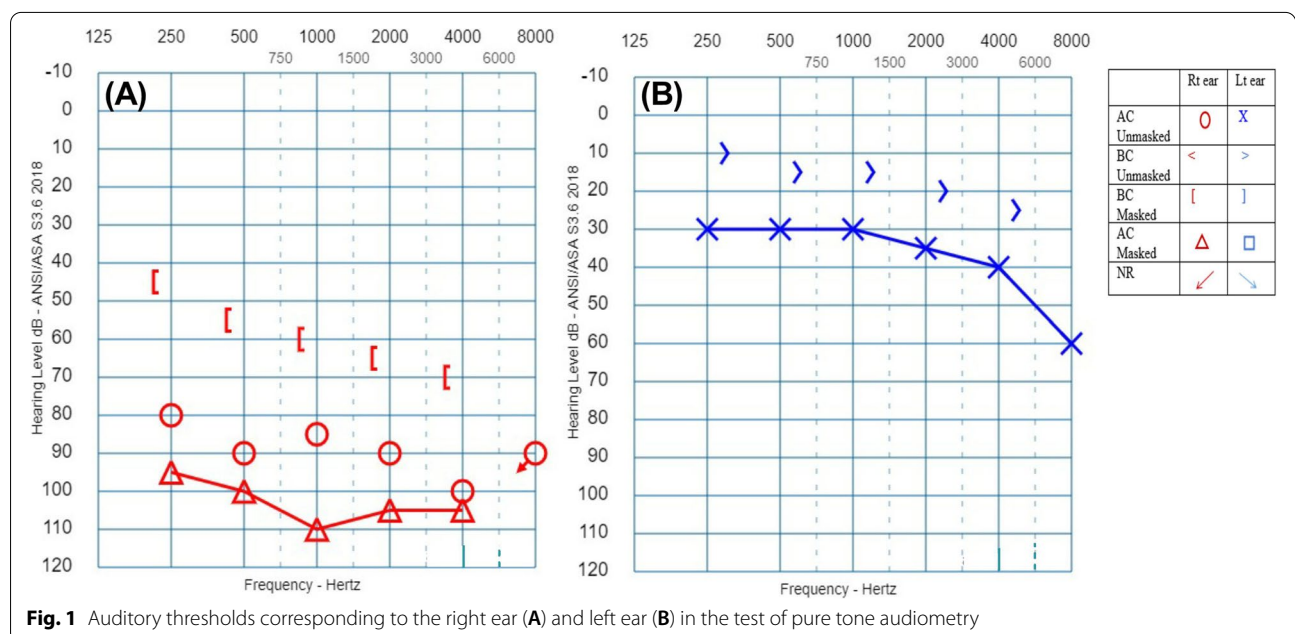


Fig. 1 Auditory thresholds corresponding to the right ear (A) and left ear (B) in the test of pure tone audiometry

Hz and low pass filter setting of 1500 Hz was preset. To avoid electrical artifacts, a notch filter was kept at 50 Hz. For click evoked ABR, analysis window of 10.1 ms was used. To account for the replicability, at least two recordings of the waveform were recorded.

Waveforms were not traceable in the right ear, indicating severe to profound hearing loss. Waves could be traced up to 50 dB nHL in the left ear indicating mild hearing loss. Absolute latencies of wave I, III, and V were determined in the left ear. At 90 dB nHL, the peak latencies of I, III and V peaks were found to be 1.35, 3.76, and 5.35 ms respectively. The ABR waveforms of both ears are shown in Fig. 2 for the left ear (blue tracings) and right ear (red tracings) respectively, indicative of mild hearing loss in the left ear and severe-profound hearing loss in right ear.

Vestibular assessment

Behavioral vestibular tests which included Romberg test, sensitive Romberg test, Fukuda stepping test, Finger to nose test, Diadochokinetic test, bedside head impulse test, head shake nystagmus, and skew deviation were performed [12]. The child could maintain the posture up to 30 s in Romberg test and up to 11 s in sensitive Romberg test. Around 10° angle of deviation with less than 1 m of displacement in forward direction was observed in Fukuda stepping test. No tremors were seen in finger to nose test. The child was able to perform alternate task in

DDK test. No nystagmus, saccades and skew deviations were noted.

Objective vestibular tests, cVEMP, and oVEMP were performed with the instrument Neurosoft-Audio (Neurosoft, Ivanovo, Russia) as the child failed in the behavioral vestibular tests and the no responses were noted in both the ears for both cVEMP and oVEMP. The test outcome were suggestive of bilateral sacculo-collic and utriculo-ocular pathway dysfunction.

Rehabilitative outcomes

The child underwent a hearing aid trial for both ears. Unaided thresholds were determined in the free field at 40 dB HL intensity using questions and wordlist (Malayalam wordlist). The unaided and aided thresholds evaluated for speech stimuli and pure tones using hearing aids in each ear is shown in Table 1. A close visual inspection of the table shows that hearing aid is not beneficial for the right ear. Hence, Phonak Audio P30 R RIC hearing aid was recommended for the left ear.

The child and the caregivers were counseled regarding anticipatory and repair strategies to make the communication easier along with the hearing aid. Few of the anticipatory strategies suggested were to talk about the problem, select appropriate seating, adequate lighting, avoid noisy situations, anticipate conversations, request for speaking naturally, request for clues about the topic, be informed, etc. The repair strategies recommended

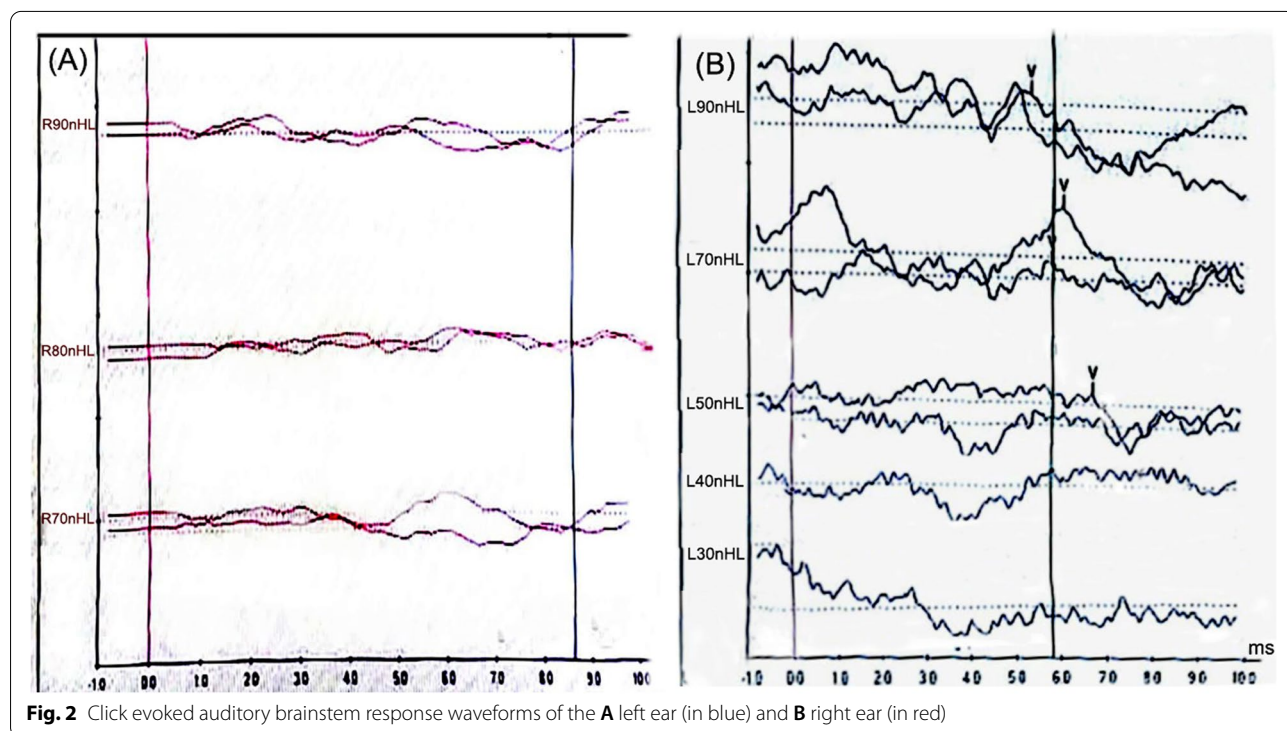


Fig. 2 Click evoked auditory brainstem response waveforms of the **A** left ear (in blue) and **B** right ear (in red)

includes repetition, remove visual distraction, pointing to subject matter, writing, sign language/gestures/finger spelling etc. Recommendations for better school communication includes the child to be seated in such a way that better ear is towards the teacher and poorer ear towards the window/corridor/noisy area/playground, etc., to ensure that better ear is not facing the noisy hallway or air conditioner, to provide pictures and handouts with verbal lessons, monitor the child's progress, to look at the child when speaking to her, to get child's attention before talking to her and to teach child to take care of hearing aid and to make sure that she doesn't turn the volume up too loud when using headphones. These communicative strategies were counselled to the parents to optimize the child's capabilities in the school and daily listening environments. The parents were also asked to report for a follow-up visit after 6 months.

Discussion

With the use of audio-vestibular test battery, the study highlighted the need for a thorough and systematic profiling in KD. Although literature documents of bilateral hearing loss of sensorineural deficits in children with KD [8, 13–15], the present case report of a female adolescent diagnosed with KD shed light on a rare manifestation of the asymmetrical loss with severe-profound mixed right ear and mild conductive HL in the left ear. The age at which the patient in our study first time reported of symptoms of KD (febrile fever, strawberry tongue, peeling of skin from the palm), which later were confirmed by lab tests was at the age of 6 years. Literature suggests that approximately 85% of the children affected with KD are younger than 5 years [16]. Although Indian data suggests that almost a third of these patients are older than 5 years [2, 17], the age reported in our present study well correlates with the conventional age of onset reported in other studies.

The diagnosis of sensorineural hearing loss recently has been reported as a frequent complication of KD. The time gap between the diagnosis of KD and perception of hearing loss as reported is from 10 days to 5 years [18]. In our client, hearing loss was suspected and diagnosed after 5 years of diagnosis of KD. The delay in detection of hearing loss could be because of near normal hearing through the better ear (left ear).

The atypical feature in our KD is the presence of mild conductive HL in one ear and severe mixed HL in other ear. This diagnosis is well correlated with the findings on auditory brainstem response test. Although conductive hearing loss can be owing to any middle ear condition, the presence of SNHL in the right ear to such a higher degree is indicative of the involvement of inner ear. Although the results of otoscopic examination indicated

intact tympanic membrane in both ears, immittance evaluation in the present case revealed 'As' type tympanogram in both ears and absent acoustic reflexes, both of which are suggestive of middle ear dysfunction. Complementing the same, OAEs were also absent in both ears. The possible cause for conductive component in our present study could be due to history of ear pain and ear discharge that the client reported to have since childhood. Acute inflammation of middle ear blood vessels can also be a factor [19], although the study is the first of its kind to report mixed hearing loss post-KD.

However, reports of SNHL in children with KD are available [7, 8] and could be due to vasculitic neuropathy (damage to the blood vessels supplying nerves) of acoustic nerve, systemic inflammation thereby affecting the membranes of labyrinth and the osmotic balance in endolymph and/or perilymph in cochlear vessels [20] and/or secondary abnormalities of the vasa nervorum and perineural vessels affecting the acoustic nerve [21]. It could reflect infection of the inner ear, similar to the cytopathic effects on labyrinth and cochlea [22]. An overlying conductive component in the same ear could be attributed to conductive pathology, which are most frequent in school-going children. Any neurological assault unlike few reported cases with KD [23] is ruled out by the neurologist opinion in the present case. Similarly, coronary artery lesions were also ruled out in the present case using ECG, although it is otherwise reported as serious complication of KD [24]. According to the few studies, ototoxicity of the high-dose aspirin used for the treatment of acute phase might cause SNHL in KD [8, 25]. Low dose of aspirin has been tapered to our client which could rule out the cause of aspirin ototoxicity. Hence, the potential role of salicylate ototoxicity can be also ruled out from the possible cause of sensorineural hearing loss [26]. Thus, the cause of sensorineural component in this case may be attributed to the vascular neuropathy. This atypical finding sheds light on to the importance of KD as a progressive condition, where the SNHL can manifest in one ear and later appear with delayed onset in the other ear. Hearing loss manifested during the treatment of acute phase of KD usually involves bilateral mild sensorineural hearing loss which recovers spontaneously [27]. In the present case, it could be hypothesized that hearing loss that began as mild in one ear progressed to severe-profound in the right ear, but due to treatment effect (intravenous gamma globulin, IVIG given after KD diagnosis in the present child) the hearing would have returned to normal in the left ear. IVIG treatment which is thought to interact with the blood cells suppressed the adverse auto-immune response. However, it remains to be answered why IVIG treatment would differentially affect the recovery patterns and the disease progression.

Table 1 Hearing aid trial and outcomes on testing digital hearing aids in aided and unaided conditions

Unaided/aided	Ear	A/V	Intensity (dB HL)	Questions	Word lists
Unaided	B	A	40	3/5	14/25
Unaided + Lt ear plug	R	A	40	0/5	0/25
Aided Audio P30 RIC	L	A	40	5/5	20/25
Aided Adhere + Lt ear plug	R	A	40	0/5	0/25
Unaided/aided	SDT	500 Hz	1000 Hz	2000 Hz	4000 Hz
Unaided (B)	50	95	85	85	105
Adhere:aided (Rt ear)	35 dB	60	40	80	75
Danavox Klar 398 BTE	45 dB	80	80	70	75

In the light of these findings, we recommend a close monitoring and regular follow-up for understanding the pathophysiology in a multi-professional setup.

Detailed vestibular assessment including both subjective and objective were carried out and dysfunction of both sacculo-collic and utriculo-ocular pathway was revealed, which propound the earlier studies which has reported of ataxia in KD patients due to involvement of vestibular acoustic nerve [3]. Due to the underestimated vestibular functions in KD, it could be an important complication associated with SNHL in children with KD. Another unique finding in the study is the bilateral absence of VEMP, indicative of compromised vestibular functioning, although the child did not demonstrate any external symptoms. The absence of VEMP could not be related to severity of hearing loss in right ear, as oVEMP/cVEMP can be even recorded in individuals with profound hearing loss [28]. Similar results are also seen in the left ear, with both cVEMP and oVEMP being compromised, reflective of vascular insult to the vestibular system. However, central compensation substituting the peripheral vestibular dysfunction by visual and somatosensory regulation could be the reason for regression in the episodes of vertigo and absence of symptoms related to disequilibrium [29]. Vestibular rehabilitation will be recommended in case the client reports of vestibular problems in future. Vestibular exercises for general coordination, balance training, eye-hand coordination, visual-motor training etc., will be taught if required, and parents are asked to monitor and report any balance related issues as and when they manifest.

For auditory rehabilitation, the child was prescribed with Audio P30 R RIC hearing aid for the left ear (better ear) as she did not benefit with hearing aid for the right ear. The prognosis with auditory rehabilitation measures was fairly good. The speech and language in our child was age-adequate, as measured in general

interview. For normal speech and language development, normal hearing is an essential component, especially in the first 5 years of life. Even mild SNHL may interrupt with normal speech and language development [30]. KD in our client is a late onset disease, greater than 6 years, which is past critical age of speech and language development, and thus our client is having normal speech and language skills.

In the view of asymmetrical hearing loss, the problems that the child might face in daily life includes trouble in localizing the sound, problem in understanding speech in noise, difficulty in understanding speech in noise, hampered social and peer interaction, need for repetition, etc. Few characteristics of children with hearing loss in classroom includes social skills that are not grade appropriate, seeking reassurance more often than other peers, feel fatigue at the end of the day due to the effort and energy they give to listening, frustration about academic success leading to behaviors such as apathy, disrespect, refusal to wear amplification etc. To overcome these problems, parents were suggested to teach the child anticipatory, repair and corrective strategies [31] to facilitate effective communication in schools and everyday listening environments.

Conclusions

We conclude that SNHL being a rare complication of KD can be in varying severities. Based on our present study, it is highly recommended to counsel the caregivers of the patients with KD about this possible sequel of hearing loss. Children with sequela of SNHL in KD may be referred to hearing evaluation long after the disappearance of signs and symptoms of KD. Health care professionals should also be aware of this possible combination of hearing loss and vestibular manifestation in KD and should perform audiometric screening in

suspected patients. This study also indicated the need for detailed case history, evaluation, and effective management and counselling in children with KD.

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Authors' contributions

MY and RJ wrote the case report. NKV and PP edited it. NI and NM performed audiological tests under guidance of NKV and PP. MY edited the photographs. All authors read and approved the final manuscript.

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Availability of data and materials

Not applicable.

Declarations

Ethics approval and consent to participate

This study was approved by the ethical committee of Institutional ethical board (REF/AUD4/2020-21) of All India Institute of Speech and Hearing, Mysuru, India. Written informed consent was obtained from the parent of the patient for the publication of this case report.

Consent for publication

The parent of the patient included in this case report gave written informed consent for the publication of the data and materials contained within this study.

Competing interests

The authors declare that they have no competing interests.

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