Auditory and Vestibular Research

Bilateral Sensorineural Hearing Loss as a Manifestation of Toxic Encephalitis: A Comprehensive Audiological and Neurological Case Report

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Highlights

Toxic encephalitis can cause bilateral sensorineural hearing loss
Audiological tests revealed cochlear and brainstem dysfunction in the patient
Early multidisciplinary evaluation aids timely diagnosis and rehabilitation

ABSTRACT

Background and Aim: Toxic encephalitis, a rare form of neuroinflammation induced by toxic agents, can lead to multifaceted neurological dysfunction, including auditory impairment. Sensorineural hearing loss (SNHL), a reduction in hearing sensitivity due to inner ear or auditory nerve damage, is an uncommon but clinically significant manifestation in such cases. This study aimed to present a comprehensive audiological and neurological evaluation of a patient with toxic encephalitis to highlight the auditory consequences and emphasize the importance of early detection and intervention.

Case presentation: We report a 44-year-old woman with progressive bilateral SNHL over one year and vertigo. Neurological examination showed cerebellar dysfunction, and Magnetic resonance imaging revealed multiple acute infarcts with basal meningeal enhancement and vasculitic changes suggestive of toxin-induced neuroinflammatory injury. Occupational history confirmed prolonged exposure to industrial solvents, a recognized neurotoxic factor. Audiological testing demonstrated bilateral moderate SNHL with absent otoacoustic emissions and abnormal auditory brainstem responses, supporting the diagnosis of toxic encephalopathy with encephalitic features.

Conclusion: This study highlights the significant impact of toxic encephalitis on auditory pathways, revealing vulnerabilities in cochlear and brainstem structures. By integrating behavioral, physiological, and electrophysiological evaluations, it provides insights into hearing loss associated with the condition. Early diagnosis and intervention are crucial to improving outcomes, and further research is needed to explore the mechanisms linking toxic encephalitis features and auditory dysfunction.

Keywords: Toxic encephalitis • audiological assessment • sensorineural hearing loss •early identification • early intervention

Introduction

Encephalitis is a severe, acute inflammation of the brain that leads to significant neuropsychological dysfunction, either in a diffuse or focal manner. When it occurs alongside meningitis, it is referred to as meningoencephalitis. Although relatively rare, encephalitis poses a serious health risk, particularly for children, the elderly, and immunocompromised individuals.

Toxic encephalopathy, a lesser-known but clinically significant subtype, results from exposure to neurotoxic agents such as heavy metals, organic solvents, and pesticides. These agents impair brain function and auditory structures by inducing oxidative stress, disrupting mitochondrial activity, and damaging cochlear hair cells, ultimately contributing to cognitive, motor, and sensory impairments, including hearing loss [1]. Toxic encephalopathy manifests through a broad spectrum of neurological deficits, including memory loss, motor incoordination, mood disturbances, sensory abnormalities, and Parkinsonian features. Chronic exposure to neurotoxic substances has been linked to syndromes such as toxic encephalopathy with encephalitic features—a term sometimes applied when toxin-induced injury produces inflammatory or vascular changes on neuroimaging,

whereas “toxic encephalopathy” more broadly refers to non-inflammatory toxic brain injury [2]. Patients often experience progressive memory loss, impaired coordination, tremors, mood disturbances, and auditory dysfunction, all of which significantly diminish their quality of life.

Epidemiologically, encephalitis remains an important public health concern. The Global Burden of Disease Study estimated an age-standardized global incidence of 19.3 per 100000 people years in 2019, a decrease from 23.2 per 100000 in 1990 [3]. Supporting this, a national surveillance effort in Kushinagar District, Uttar Pradesh, reported a comparable annual acute encephalitis syndrome incidence of 20.2 per 100000 population in 2011 [4].

Hearing impairment is a frequently overlooked but significant complication of neurotoxic exposure. Epidemiological studies in humans have established that exposure to heavy metals (particularly lead and cadmium) and organic solvents (notably toluene and styrene), even at moderate levels or in combination with noise, adversely affects cochlear and central auditory pathways, manifesting as sensorineural hearing loss (SNHL) and auditory processing dysfunction [5].

Individuals exposed to organic solvents have been reported to experience hearing loss, with some also reporting tinnitus. Such exposure can impair both auditory and central nervous system functions, emphasizing the need for evaluations. Manganese exposure contributes to both peripheral and central auditory dysfunction by damaging outer hair cells and auditory neurons, and when combined with noise, its ototoxic effects are amplified—leading to elevated hearing thresholds and suggesting a potential mechanism for manganese-induced neurodegeneration in auditory pathways. Occupational exposure to heavy metals such as lead and mercury causes measurable auditory changes, including delayed Auditory Brainstem Response (ABR) interpeak latencies specifically prolonged Wave I–V latencies indicating central auditory dysfunction when compared with unexposed controls. These findings underscore the importance of early auditory assessments in patients with toxic encephalopathy, as timely identification of hearing deficits can facilitate appropriate interventions and rehabilitation [6-8].

Diagnosing toxic encephalopathy requires a multidisciplinary approach that integrates behavioral, physiological, electrophysiological, and radiological assessments, including computed tomography and Magnetic Resonance Imaging (MRI). Given the established link between neurotoxicity and auditory dysfunction, audiological evaluations—such as pure-tone audiometry (PTA), otoacoustic emissions (OAEs), and ABR testing—are essential for early detection of auditory pathway involvement. Workers exposed to organic solvents exhibited central auditory processing deficits and abnormal ABR, even in the absence of significant peripheral hearing loss, highlighting the importance of detailed auditory assessments in individuals with toxic encephalopathy [9]. In this context, the present study aimed to conduct an extensive auditory assessment of a single case of toxic encephalitis using a multidisciplinary approach. By integrating behavioural, physiological, electrophysiological, and radiological evaluations, the investigation sought to delineate the extent and nature of hearing loss associated with toxic encephalopathy with encephalitic features and to provide deeper insights into its auditory implications.

Case Presentation

A 44-year-old woman presented with progressively worsening bilateral hearing loss over the past year and persistent giddiness for 20 days. She had no family or medical history of hearing impairment, and no prior audiological records were available for comparison. ENT examination showed a normal right tympanic membrane and a retracted left tympanic membrane without infection, while neurological assessment revealed a swaying gait toward the right and cerebellar dysfunction. Occupational history indicated prolonged exposure to industrial solvents in a shoe manufacturing unit, a recognized neurotoxic risk factor. Brain magnetic resonance imaging demonstrated multiple tiny acute infarcts in the genu and bilateral centrum semiovale with diffuse basal meningeal enhancement and vasculitic changes—findings suggestive of toxin-associated vascular and neuroinflammatory injury. Audiological evaluation confirmed bilateral moderate SNHL with absent otoacoustic emissions and abnormal ABRs, reflecting combined cochlear dysfunction and impaired brainstem conduction. In the absence of biochemical toxicology data, these clinical, imaging, and occupational findings were most consistent with toxic encephalopathy presenting with encephalitic features.

This study followed a single-case design, and hence, inclusion or exclusion criteria were not applicable. Ethical approval was obtained from the Institutional Human Ethics Committee, and written informed consent was collected from the participant. Neurological assessment was conducted prior to audiological testing. MRI showed multiple tiny acute infarcts involving the genu and bilateral centrum semiovale, along with basal meningeal involvement suggestive of vasculitis, which guided subsequent audiological investigations.

A case history was collected regarding hearing and communication difficulties, followed by otoscopic examination using a Heine 3000 otoscope (Heine Optotechnik GmbH & Co. KG, Germany). All assessments were conducted in a sound-treated room. Pure-tone audiometry (PTA) was performed using an AD528 Interacoustics audiometer (Interacoustics A/S, Denmark) to determine air and bone conduction thresholds. Speech audiometry evaluated the speech recognition threshold, speech identification score, and uncomfortable loudness levels.

Tympanometry and acoustic reflex threshold measurements were carried out using a MAICO MI24C tympanometer (MAICO Diagnostics GmbH, Germany) with a 226 Hz probe tone, utilizing a pressure sweep from −200 to +200 daPa. Ipsilateral and contralateral acoustic reflexes were recorded at 0.5, 1, 2, and 4 kHz at stimulus levels of 80–110 dB. Outer hair-cell function was evaluated using distortion product otoacoustic emissions (DPOAEs) with a Path Medical Sentiero instrument (Germany) using an F2/F1 ratio of 1.2, with tone pairs set at 65 dB SPL and 55 dB SPL for L1 and L2, respectively.

ABR testing was performed in a quiet environment using a Neuro-Audio system (Neurosoft, Russia). Click stimuli (0.1 ms duration, 11.1/s) were delivered in rarefaction polarity through ER-3A insert earphones. Responses were recorded using a vertical electrode montage with gold cup electrodes employing a bandpass filter of 100–3000 Hz within a 12-ms time window. A total of 2,000 sweeps were collected, maintaining electrode impedance below 5 kΩ for optimal signal quality.

Otoscopic examination showed a normal right ear canal and tympanic membrane, while the left ear canal appeared normal but with a retracted tympanic membrane. Tuning fork tests revealed Rinne positive bilaterally, indicating no conductive component, and the Weber test lateralized to the right ear, consistent with an SNHL pattern. The audiogram (Figure 1) revealed bilateral moderate SNHL, with masked bone conduction thresholds closely aligned with air conduction thresholds, confirming the sensorineural origin. Speech reception thresholds were consistent with audiometric findings, and speech identification scores ranged from fair to good at 40 dB above the threshold. No evidence of PB rollover was observed, indicating no retrocochlear pathology. Uncomfortable loudness levels exceeding 100 dB indicated a normal dynamic range.

Tympanometric evaluation (Figure 2) demonstrated normal middle ear mobility bilaterally (Type A tympanogram). Reflexometry showed bilateral absence or elevation of acoustic reflex thresholds for ipsilateral stimulation. Figure 2 and Table 2 depict these outcomes.

DPOAEs were largely absent bilaterally, although some residual emissions with low signal-to-noise ratios were noted. These findings support the diagnosis of SNHL. The frequency-specific DPOAE responses are presented in Figure 3 and Table 3.

Electrophysiological evaluation using ABR (Figure 4) showed the absence of Wave V responses below 70 dBnHL. At 70 dBnHL and above, Wave V was present but delayed, supporting central auditory pathway involvement. A follow-up assessment after one month revealed no significant changes in hearing thresholds or diagnostic findings. Figure 4 and Table 4 present the ABR waveforms and latency data.

The patient was referred to a neurologist for further evaluation and treatment. Audiological rehabilitation was initiated, with recommendations for annual hearing assessments.

Discussion

The findings of this study reveal bilateral moderate SNHL, confirmed through a detailed audiological evaluation. Otoscopic examination showed a normal tympanic membrane in the right ear and a retracted tympanic membrane in the left ear, with no signs of infection or other abnormalities. PTA demonstrated bilateral moderate SNHL, consistent with the patient's clinical complaints. Immittance evaluation revealed Type A tympanograms bilaterally with absent or elevated acoustic reflexes, indicating normal middle ear mobility but reduced reflex activity. The absence of DPOAEs bilaterally suggests significant cochlear outer hair cell dysfunction. Additionally, ABR testing revealed absent Wave V responses at intensities below 70 dBnHL, with delayed latencies at higher intensities, indicating impaired neural conduction along the auditory brainstem pathways. Together, these findings point to combined cochlear and brainstem dysfunction, both of which have been reported in neurotoxic conditions such as toxic encephalopathy with encephalitic features.

Toxic encephalopathy can lead to widespread neuroinflammatory changes and vascular compromise that affect the auditory system. The cochlea, due to its high metabolic demand, is especially vulnerable to oxidative stress and mitochondrial dysfunction. Neurotoxic substances may damage cochlear structures and auditory neural pathways either directly or through secondary immune-mediated inflammation. Patients with chronic solvent-

induced encephalopathy have been reported to exhibit persistent neuropsychological and sensory impairments, including signs consistent with cortical and subcortical damage [10]. These findings support the vulnerability of both central and peripheral auditory pathways to neurotoxic exposure, consistent with our patient's absent OAEs and delayed ABR wave latencies.

Inflammatory mediators released in toxic encephalitis can disrupt neural transmission within the auditory brainstem. Consistent evidence from neuroinflammatory conditions such as multiple sclerosis and neurodegeneration demonstrates impaired auditory processing and abnormal ABR findings, suggesting that inflammation-related neural disruption may impair synaptic efficiency in central auditory pathways [11]. Our patient's delayed ABR wave latencies align with these mechanisms, indicating possible central auditory involvement.

Several occupational and clinical studies reinforce this association between toxic exposure and hearing impairment. Co-exposure to organic solvents and heavy metals has been shown to significantly exacerbate noise-induced hearing loss, as evidenced by greater elevations in pure-tone thresholds compared to noise exposure alone, supporting the ototoxic synergy of these chemical agents on peripheral auditory structures [12]. Even chronic, low-level exposure to organic solvents—especially when combined with occupational noise—can lead to progressive cochlear and neural degeneration [13]. This often manifests as delayed-onset SNHL and central auditory processing deficits, mechanisms highly relevant to toxic encephalopathy and reflective of the current patient's findings.

In a broader clinical context, approximately 20% of children recovering from acute encephalitis syndrome have been reported to suffer hearing loss, with factors such as prolonged seizures, high fever, and reduced consciousness significantly correlated with auditory dysfunction highlighting the need for early and routine audiological assessments in encephalitis survivors, especially when toxic or inflammatory etiologies are suspected [14]. A case of a 66-year-old woman with progressive encephalopathy and bilateral SNHL revealed delayed interpeak latencies in ABR testing, with absent otoacoustic emissions, supporting concurrent cochlear and brainstem involvement and underscoring the potential for toxic encephalopathy to affect both peripheral and central auditory pathways [15].

In the present case, although the patient's hearing thresholds remained stable over a one-month follow-up, ongoing monitoring is necessary due to the risk of delayed or progressive auditory decline. Long-term effects of toxic exposure on the auditory system may not always be immediate, and fluctuating or progressive SNHL has been documented in similar cases.

This case emphasizes the importance of a multidisciplinary management approach involving neurologists, audiologists, and otolaryngologists. In-depth evaluation, including PTA, OAEs, tympanometry, and ABR are essential in detecting early signs of auditory involvement. Early identification allows for timely audiological intervention, rehabilitation, and counselling, ultimately improving patient outcomes and quality of life.

However, the present study has certain limitations. The absence of previous audiological records limited objective confirmation of progressive hearing loss. No patient-specific toxicological or biochemical evidence was obtained to confirm toxin exposure. Additionally, special tests for central auditory processing disorder, including speech-in-noise testing, were not performed.

Conclusion

This study highlights bilateral moderate sensorineural hearing loss with audiological evidence of cochlear dysfunction and auditory brainstem abnormalities in a patient with toxic encephalopathy with encephalitic features. These findings underscore the vulnerability of auditory structures to neurotoxic injury and the need for extensive audiological evaluation. Early detection and intervention are crucial to preserving communication and quality of life, and multidisciplinary care involving audiology, neurology, and otology is recommended. Further research is required to elucidate the mechanisms linking toxic encephalitis and auditory dysfunction.

Acknowledgments

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Ethical consideration

This study was approved by the Institutional Human Ethics Committee of Vinayaka Missions Research Foundation (Ethical Code No: VMRF/IHEC/ASLP/2025/032). Written informed consent was obtained from the participant for clinical evaluation and publication.

Conflict of interest: The authors declare no conflicts of interest

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Table 1: Audiological findings indicating cochlear and brainstem involvement

Ear	PTA (dBHL)	SRT (dBHL)	SIS (%)	UCL (dBHL)
Right ear	41.6	50	90	>100
Left ear	50	60	90	>100

Note: PTA – Pure Tone Average; SRT – Speech Reception Threshold; SIS – Speech Identification Score; UCL – Uncomfortable Loudness Level; dBHL – decibel hearing level.

Table 2: Tympanometric measures including static admittance, peak pressure, canal volume, and width

Ear	Static Admittance (mmho)	Tympanic Peak Pressure (daPa)	Ear Canal Volume (cc)	Tympanometric Width (daPa)
Right	0.9	−9	1.8	108
Left	0.8	−21	1.7	110

Note: daPa – decapascals; mmho – millimhos; cc – cubic centimeters.

Table 3: Distortion product otoacoustic emission findings for both ears

F2 (Hz)	DP (dB) Right	Noise (dB) Right	SNR (dB) Right	DP (dB) Left	Noise (dB) Left	SNR (dB) Left
889	17	9.21	7.7	11.5	3.04	8.46
988	6.55	2.33	4.2	6.56	3.75	2.81
1481	2.70	−1.69	2.0	5.28	2.78	2.5
2222	1.92	−3.58	−1.6	3.79	2.81	0.98
2963	4.23	−10.42	−6.1	0.86	−4.06	−3.2
4444	0.46	−10.23	−9.7	0.12	−4.18	−4.06
5714	−5.12	−15.00	−19.6	−10.5	−8.22	−18.7

Note: F2 – higher frequency of tone pair; Hz- hertz; DP – distortion product; SNR – signal-to-noise ratio; dB – decibel.

Table 4: Auditory brainstem response wave V latency data and interaural latency difference

ABR Component	Intensity (dBnHL)	Right Ear Latency (ms)	Left Ear Latency (ms)	Interaural Latency Difference (ms)
Wave V	90	6.11	6.54	0.4
Wave V	70	6.52	7.10	0.6

Note: ABR – Auditory Brainstem Response; dBnHL – decibel normalized hearing level; ms – milliseconds

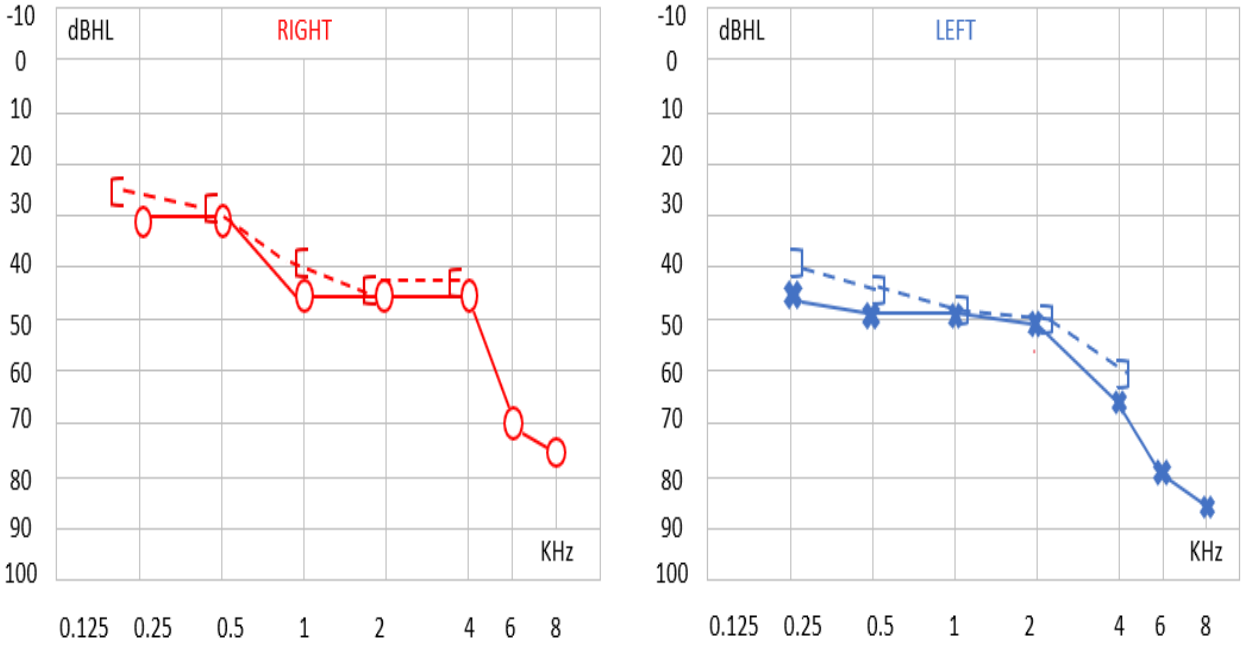

Figure 1: Pure tone audiogram showing bilateral moderate sensorineural hearing loss

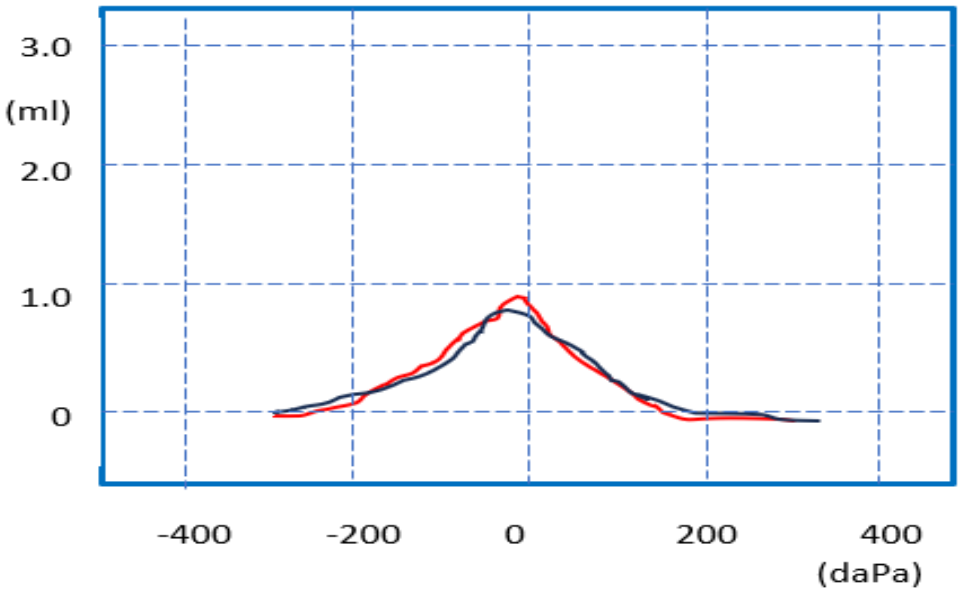

Figure 2: Tympanometry showing type A bilaterally, indicating normal middle ear function (right ear: red, left ear: blue)

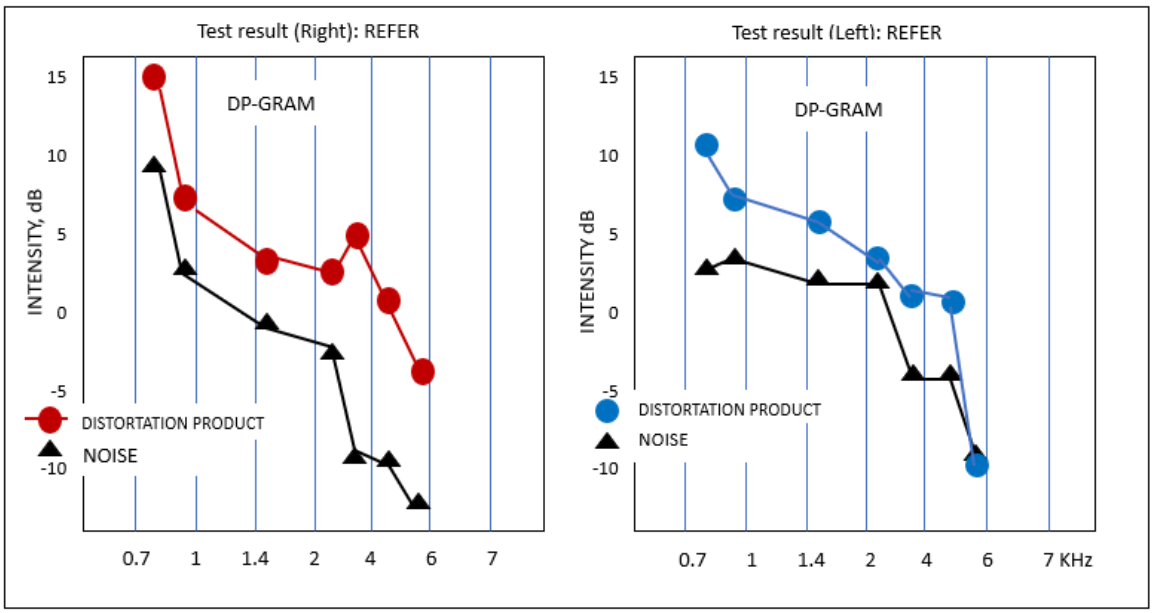

Figure 3: Distortion product otoacoustic emissions absent bilaterally, indicating outer hair cell dysfunction

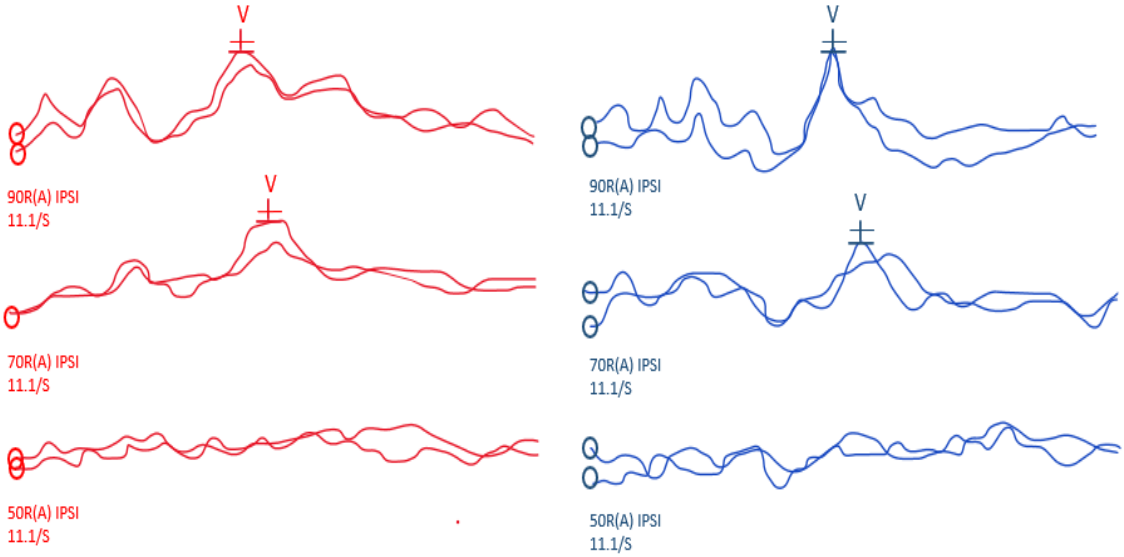

Figure 4: Auditory brainstem response showing Wave V latencies, suggesting brainstem impairment