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Unusual regression of a sudden-onset sensorineural hearing loss in a patient with cerebellopontine angle pathology

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Abstract
This is a report of a patient with small internal acoustic canal pathology presenting as a sudden-onset sensorineural hearing loss. Initially the patient received non-specific empirical medical therapy and the sudden-onset sensorineural hearing loss recovered rapidly. Regression of the hearing loss, despite no change in magnetic resonance imaging (MRI) findings after the medical treatment is documented and discussed.

Key words: Hearing Loss, Sensorineural; Neuroma Acoustics

Introduction

Sudden sensorineural hearing loss (sudden SNHL) has been described as 'sensorineural hearing loss with at least a 30-dB decrease in threshold in three contiguous test frequencies occurring over a 24 to 72 hour period'[1] and may necessitate medical or surgical intervention. We report on a patient who presented with sudden SNHL and received non-specific medical treatment. Despite the consistency of the internal acoustic canal pathology in terms of magnetic resonance imaging (MRI) parameters before, and after the treatment, sudden SNHL partially recovered during the treatment, as demonstrated by subjective and objective audiological test results. Rare occurrence of the regression of sudden SNHL in a patient with cerebellopontine angle pathology is reported and the case is discussed.

Case report

A 48-year-old lady presented to our out-patient department on 20 December 1999, complaining of tinnitus and hearing loss in her left ear, 28 hours before presentation. She had no balance problems at all. Her ear, nose and throat examination and systemic physical examination were unremarkable. Past medical history was non-contributory and the patient denied any previous viral illness, flying, diving, strenous physical activity, or head trauma. Pure tone audiogram performed on the same day revealed a left-sided (cookie-bite type) severe mid-frequency sensorineural hearing loss with an average of 67 dB HL (average of 0.5/1/2 kHz pure air-conduction thresholds, i.e. the lowest curve in Figure 1). The speech recognition threshold was 65 dB HL, and the speech discrimination score was 48 per cent, that was considered to be suspicious in terms of retrocochlear pathology. Hearing thresholds on the right ear were within normal levels. No transiently evoked oto-acoustic emission was recorded on the left ear, while normal response was obtained on the right one. Both of the tympanograms were type A. Acoustic reflex was

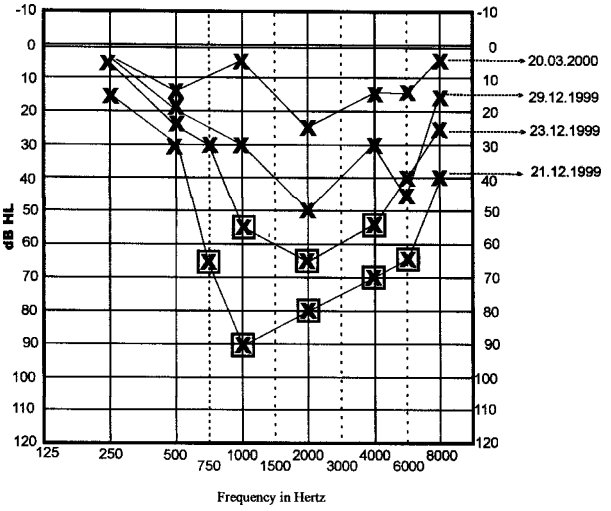

Fig. 1
Pure tone air-conduction thresholds of the left ear obtained on consecutive days.

recordable in the left ear only on response to a 500 Hz tone and the acoustic reflex decay test was positive at this frequency. Thyroid hormone levels were normal, fluorescent treponemal antibody absorption serum (FTA-ABS), and toxoplasmosis, rubella, cytomegalovirus and herpes simplex (TORCH) screening test was negative. The bithermal water caloric test revealed symmetrical and normal response.

On the day of admission, she was hospitalized and an infusion protocol treatment was commenced targeting idiopathic sudden SNHL: a combination of high-dose of prednisolone (starting with 100 mg daily) and acyclovir 250 mg flagon (10 mg/kg). Additionally oxpentifylline 400 mg tablet, per os in two divided doses, was also used.

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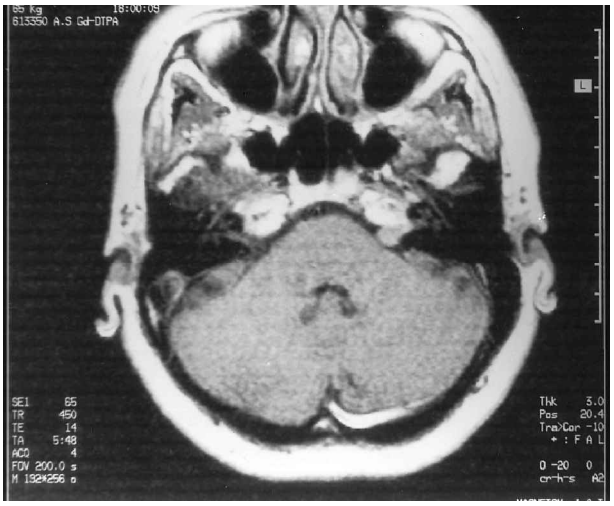

FIG. 2

Axial T1 Gd-MRI demonstrating enhanced lesion within the left internal acoustic canal and extending into the CPA.

Two days after admission (22 December 1999), ABR recordings showed abnormal findings on the left ear: absence of Wave I, an abnormally long Wave V latency (6.42 ms, which is longer than five standard deviation (STD) of our normative data), and a very long interaural Wave V latency difference (1.09 ms) in response to rarefaction clicks at 85 dB nHL.

On 23 of December, 1999, the pure tone threshold average shifted from 67 dB to 48 dB HL (hearing thresholds of the left ear in consecutive dates are presented in Figure 1). Similarly, the speech discrimination score was increased from 48 to 80 per cent. On the same day, a second ABR recording was performed. ABR parameters were slightly improved but were still abnormal: Wave I emerged within the normal range (1.42 ms), the interaural Wave V difference was reduced (0.71 ms), and both Wave V latency (6.01 ms) and I-V inter-peak interval (4.59 ms) were still beyond 2 STD.

On the seventh day of admittance (27 December 1999), magnetic resonance imaging (MRI) was performed and a gadolinium-enhanced lesion measuring $10 \times 5 \times 6$ mm, that was isointense with gray matter on T1 and T2 weighted images was detected in the left cerebellopontine angle arising from the left internal acoustic canal (IAC) (Figure 2). Its typical appearance suggested it to be a vestibular schwannoma (VS).

Medical therapy protocol was continued for 10 days as scheduled before. Hearing thresholds of the left ear gradually recovered almost to normal hearing (curve on the top of Figure 1). Re-evaluation of the pathology with MRI technique after the treatment (5 January 2000), showed an identical lesion in the left IAC.

Discussion

Sudden SNHL is mostly associated with cochlear pathologies but not with CPA and IAC pathologies. Even though progressive hearing loss is a well known major complaint in patients with CPA pathology, sudden SNHL was found to be evident in 22 per cent of 132 patients with surgically proven unilateral VS.[2] In the latter review, the characteristics of sudden SNHL cases with VS were summarized as follows: 1) a small tumour, 2) short duration after onset, 3) low incidence of associated symptoms, 4) trough configuration of audiogram, and 5) normal caloric response of the affected ear. In the literature, theories for the cause of sudden SNHL in VS are suggested: i) direct pressure on the cochlear nerve, ii) pressure on the vessels supplying the labyrinth, iii) the conduction block of the cochlear nerve fibres.[3] The incidence of VS in all sudden SNHL cases is varied. For instance, in Shaia and Sheehy's series of 1220 sudden SNHL patients, VS accounted for 0.8 per cent of all the cases, thus these authors recommend that all the patients with sudden SNHL should be evaluated to rule out VS.[4] However, in another study, the probability of VS in sudden SNHL is estimated to be relatively high, five to 30 per cent and thus, MRI is proposed as a 'cost-effective tool' to evaluate the patients suspected of having sudden SNHL due to VS.[5]

Furthermore, in rare instances, sudden SNHL reported to be the presenting symptom in a patient with VS in an only hearing ear. In such a case report, surgery had not been performed and hearing loss recovered after steroid therapy on four separate occasions.[6]

Surgery was not performed for the case reported herein, therefore no pathological diagnosis was obtained. However, the MRI parameters described above indicate that the lesion is an intracanalicular tumour, i.e. VS. After the medical therapy, abnormal ABR parameters were found to be slightly improved. Interestingly, the regression of the ABR parameters correlated well with the similar regression in pure tone thresholds. It is likely that the conduction block in the cochlear nerve, which was demonstrated by abnormal ABR parameters, could be partially released due to high-dose steroid therapy. On the other hand, oxpentifylline could also contribute to the regression by increasing the blood supply of the labyrinth.

Based on the unusual regression of the sudden SNHL of the case reported here, it is suggested that patients with sudden SNHL initially should be evaluated with ABR and then cases with high suspicion of CPA pathology further investigated utilizing MRI, even if the sudden SNHL recovers.

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