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Association of Sudden Hearing Loss with Vertebrobasiler Insufficiency: A Case Report

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Abstract

Aim: Sudden hearing loss can occur due to viral and vascular causes. Vertebrobasilar insufficiency, which is one of the vascular reasons, commonly cause vestibular problems in middle-aged and older patients, with or without hearing impairment. However, little is known about the association between sudden hearing loss and vertebrobasilar insufficiency.

Methods: A 51 years old male who had sudden onset hearing loss in his left ear and vertigo is presented. Initially, he was diagnosed of left-sided sudden idiopathic sensorineural hearing loss. Suspicion of vertebrobasilar insufficiency raised following completion of vestibular evaluations and diagnosed with imaging studies.

Results: It should be kept in mind that vertebrobasilar insufficiency can be an etiologic factor in cases presenting with sudden hearing loss.

Keywords: Dizziness; sudden hearing loss; Vertigo; Vertebrobasiler insufficiency; Vestibular diseases; Rehabilitation

Ani İşitme Kaybının Vertebrobaziler Yetmezlikle İlişkisi: Bir Olgu Sunumu

Öz

Amaç: Ani işitme kaybı, viral ve vasküler nedenlere bağlı olarak ortaya çıkabilir. Vasküler nedenlerden biri olan vertebrobaziler yetmezlik, daha çok orta yaş ve üzerindeki hastalarda vestibüler problemlere neden olabilir. Ani işitme kaybı ve vertebrobaziler yetmezlik arasındaki ilişki az bilinmektedir.

Yöntem: Bu yazıda sol kulağında ani başlangıçlı işitme kaybı ve vertigo nedeniyle başvuran bir erkek olgu sunulmuştur. Hasta başlangıçta ani idiyopatik sensörinöral işitme kaybı tanısı almıştır. Vertebrobaziler yetmezlik şüphesi, vestibüler değerlendirmeler sonucunda oluşmuş, tanısı radyolojik görüntülemeler ile koyulmuştur.

Sonuç: Vertebrobaziler yetmezliğin, ani işitme kaybı etiyolojisinde rol oynayabileceği akılda tutulmalıdır.

Anahtar Kelimeler: Baş dönmesi; Ani işitme kaybı; Vertigo; Vertebrobaziler yetmezlik; Vestibüler hastalıklar; Rehabilitasyon

1. Introduction

Sudden hearing loss (SHL) is defined as the occurrence of sensorineural hearing loss (SNHL) of 30 dB and more, at least three consecutive frequencies, in less than three days [1]. The incidence of SHL varies between 5-27/100.000 population [2]. There is no complete clarity about the etiology of SHL, which is seen in different configurations and usually unilateral [1]. It is known that SHL is mostly caused by viral and vascular problems. Vascular causes include high viscosity syndromes, hypercoagulopathies, vasospasm, thromboembolism, carotid artery

pathologies and vertebrobasilar insufficiency (VBI) [3].

VBI is one of the common causes of vestibular disorders in middle-aged and older patients [4]. The arterial supply to the inner ear and vestibulo-cochlear nuclei in the brainstem are provided by vertebrobasilar arterial system (VAS). Blood flow insufficiency in VAS or episodic decrease in blood flow can cause vertigo and hearing loss [3, 4]. In addition to vertigo and hearing loss, symptoms such as nausea, vomiting, numbness in the limbs, muscle weakness and headache may accompany VBI. The frequent anatomic variation of vertebral arteries, their fusion to form a basilar artery and the presence of collateral circulation make difficult to the diagnosis of VBI [4]. The clinical diagnosis of VBI is made by the presence of objective symptoms such as visual impairment, vertigo, position-related nystagmus, balance disorders, and supported by imaging methods such as magnetic resonance imaging (MRI), magnetic resonance angiography (MRA) and doppler ultrasonography (USG) of the vertebrobasilar system [4]. Hypertension, various heart diseases, carotid artery disease, diabetes and advanced age are among the risk factors for VBI [3]. In this case report, an adult with complaints of SHL and vertigo in the left ear and who had VBI as one of the causes of SHL is presented.

2. Case Report

51 years old male presented with complaints of SHL, humming and concurrent vertigo in his left ear, nausea, vomiting, imbalance. He had a history of heart disease and hypertension for years and was using a pacemaker. He had no other known chronic disease or neurological problem. In the otoscopic examination of the patient, both external auditory canals and tympanic membranes were normal. He was referred to Ankara University, Faculty of Medicine, Ibni Sina Hospital, Department of Otorhinolaryngology, Audiology, Speech and Vestibular Disorders Diagnosis and Rehabilitation Center for further audiological and vestibular tests. Informed consent was obtained verbally from this patient before the study and the principles in the Declaration of Helsinki were followed.

Table 1. Results of Audiologic Evaluation

	Left Ear	**Right Ear**
Before medication/first audiologic evaluation	Mild SNHL, configuration with a sudden drop from 2 kHz.	Normal hearing, SNHL configuration with a sudden drop from 2 kHz.
3rd day of complaints/second audiologic evaluation	Moderate SNHL, configuration increasing towards high frequencies	Same as previous hearing thresholds
5th, 9th and 11th day of complaints/other audiologic evaluation	Severe SNHL, flat type configuration	Same as previous hearing thresholds
1 MONTH OF SHL TREATMENT		
Post-treatment 6th week	Bilateral normal hearing, SNHL configuration with a sudden drop from 2 kHz.	

2.1. Audiological Evaluation

The audiological evaluation results are presented in *Table 1*. Grason-Stadler AudioStar Pro (USA) audiometry was used for audiological evaluation. Pure tone average (PTA) referred to the average of hearing threshold levels at a set of specified frequencies: 500, 1000, 2000 and 4000 Hz typically. The patient had high dose systemic (starting with 250 mg prednisolone, tapered within 12 days) and intratympanic dexamethasone treatment (4 mg/ml every other day for three times) with the diagnosis of sudden idiopathic SHL. The course of his hearing is summarized in *Table 1* and consequent pure tone audiograms representing the progression of hearing loss despite medical treatment are presented in *Figure 1*.

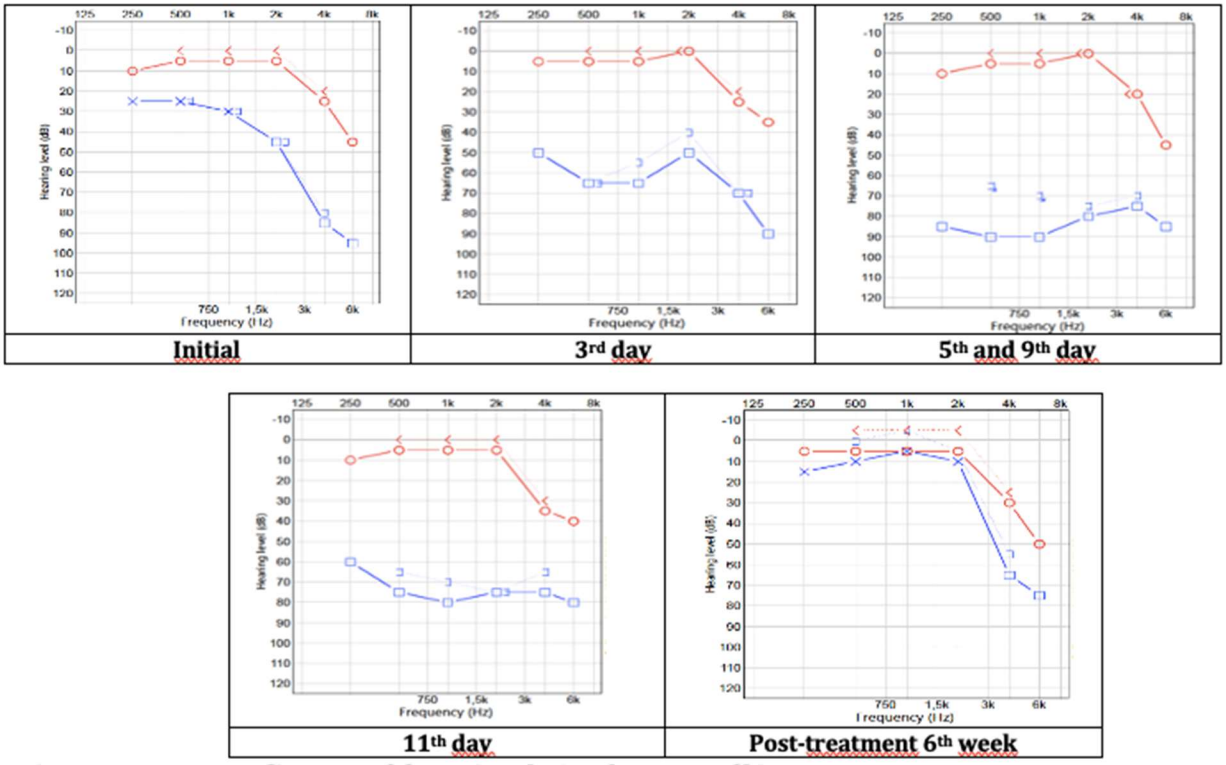

Figure 1: Pure tone audiograms of the patient during the course of the treatment

2.2. Vestibular Evaluation

Gaze, saccade, pursuit, optokinetic tests, spontaneous nystagmus, Dix Hallpike and Roll-head maneuver, head shake and positional body test were applied with Micromedical Video-nystagmography (VNG) device. Sensor Organization Test (SOT) was applied with NeuroCom Dynamic Posturography device. Lateral Video Head Impulse Test (L-VHIT) was applied with Interacoustics (EyeSeeCam) VHIT device. In *Table 2*, vestibular evaluation results of this case were presented, the sensation of vertigo and fatigue did not occur during the positional tests. The first vestibular evaluation was performed before SHL treatment.

After one month of VR dizziness improved and repeated SOT was within normal limits *(Table 3).* In VHIT, bilateral lateral canal vestibulo-ocular reflex (VOR) gain was low (right gain: 0.66, left gain: 0.55), gain asymmetry was 9% in favor of weakness on the left, and covert saccades were observed on the left. L-VHIT after positional body tests arose suspicion of VBI on the left side and imaging studies were recommended for the

patient. Carotid Doppler ultrasonography (USG) showed bilateral intimal thickening and non-stenotic plaques. Vertebral Artery Doppler USG showed that the diameter of left vertebral artery was 2.9 mm on the left side, compared to 4.5 mm on the right side, and the flow rate of the left vertebral artery was 35 ml/min, compared to 105 ml/min on the right side. Total flow rate of both vertebral arteries was 140 ml/min, which was less than the normal lower limit of 200 ml/min. VBI caused the dizziness and this feeling was compensated by VR. It was recommended that this case should continue VOR exercises for improving quality of daily life. After SHL treatment, hearing thresholds of this case in the left ear recovered and stabilized, sensation of vertigo recovered *(Figure 1).* Since dizziness persisted, vestibular evaluation was repeated *(Table 2).* He did not describe sensation of vertigo during the positional tests. The disappearance of the nystagmus on the left in positional body test suggested the suspicion of VBI on the left side. Because of the dizziness, the case was performed SOT *(Table 3).* Somatosensory (SOM), visual (VIS), vestibular (VEST) and composite scores of this case were observed to be significantly decreased and the center of gravity (COG) was lateralized to the right. Vestibular rehabilitation (VR) was planned with VR exercises.

3. Discussion

A certain etiological cause cannot be found in SHL and it is commonly considered idiopathic. In this report, a case whose diagnosis of VBI was reached by imaging methods based on the audio-vestibular evaluations was presented.

Demographic studies of SHL show a wide age distribution, with an average of 50–60 years and no gender preference [5]. It is well known that 25% of elderly population and especially over age of 50 may experience imbalance as a result of VBI. VBI is more common in males [6]. The prevalence of VBI was 0.5% of patients with SHL and 0.2% of patients with not SHL in a study [3]. Our case was 51 years-old who was within the decade with the highest incidence of SHL and in the gender group where VBI is seen more frequently.

Documentation of moderate to severe SNHL in SHL is diagnostic. Hearing loss can be progressive, occurring only low or high frequencies as well as all frequencies [5]. Our case had mild SNHL which occurred in high frequencies in the left ear initially, then it progressed into severe HL with a flat type configuration. After SHL treatment, it was thought that the presence of bilateral sudden deterioration above 2 kHz may also be due to existing presbyacusis.

The hearing complaints improved with systemic and intratympanic dexamethasone treatment after about 2 months from the beginning SHL. Although the first vestibular evaluation results suggested a peripheral problem, the continuation of dizzy feeling required a more detailed second vestibular evaluation after SHL treatment. In addition to the initial vestibular evaluation, positional body test raised the suspicion of unilateral vestibular hypofunction. Nystagmus was not observed in the body-left position in the positional body test, but the presence of right upbeat torsional nystagmus in the body-right position. It has been shown that rotation and hyperextension of the neck compress the vertebral artery in the opposite direction of the rotated side in postmortem studies [7]. In a study, transient occlusion in the left vertebral artery was shown in the angiography of a case with vertigo and horizontal nystagmus that appeared when head to right position [8]. This situation was the most important finding leading to distinctive diagnosis. It suggested transient ischemia of the common cochlear artery of the left labyrinth and consequently the suspicion of VBI on the left. Position related transient ischemia causes a change in the blood flow of the vestibulocochlear system. It results in temporary hyperactivity of the vestibular nerve and/or hair cells, it is reasonable to have hearing loss associated with nystagmus. Our roadmap was radiological imaging due to suspicion of VBI on the left with this assumption in this case

Table 2: Results of Vestibular Evaluation

	Before SHL treatment	After SHL treatment
VNG	Gaze, saccade, pursuit tests were normal. In optokinetic test, right gain: 0.85 (normal), ***left gain: 0.58 (low)***	Gaze, saccade, pursuit, optokinetic tests were normal.
Spontan nystagmus	No nystagmus with fixation. ***Presence of left torsional nystagmus in no fixation***	No nystagmus with fixation. ***Presence of minimal left horizontal nystagmus in no fixation***
Head-shake test	In passive state-in first 5 seconds, right downward torsional, then continuous left horizontal and minimal downbeat nystagmus	-
Dix-Hallpike maneuver	Head to right lying: >60 seconds lasting right upbeat torsional, head to left lying: >60 seconds lasting left upbeat torsional nystagmus, nystagmus more severe on the left.	Head to right lying: >60 seconds lasting right upbeat torsional, head to left lying: >60 seconds lasting left upbeat torsional nystagmus
Roll-head maneuver	***Head to right position: >60 seconds lasting right geotropic nystagmus, head to left position: >60 seconds lasting left geotropic nystagmus***	***Head to right position: >60 seconds lasting right geotropic nystagmus, head to left position: >60 seconds lasting left geotropic nystagmus***
Positional body test	-	***Body-left: no nystagmus*** ***Body-right: presence of right upbeat torsional nystagmus***
Lateral-VHIT	-	Bilateral lateral canal vestibulo-ocular reflex (VOR) gain: Low (right gain: 0.66, left gain: 0.55) ***Gain asymmetry was 9% in favor of weakness on the left.*** ***The presence of covert saccades on the left.***

Table 3: Results of Sensor Organization Test (SOT) Before and After Vestibular Rehabilitation (VR)

	Before VR	After VR
SOM	79↓	97
VIS	100	78
VEST	21↓	71
PREF	98	99
Composite	67 (%4)↓	76
COG	Lateralized to the right-up	medial

VR: Vestibular rehabilitation; SOM: Somatosensory; VIS: Visual; VEST: Vestibular; PREF: Preference; COG: Center of Gravity

VBI could cause discrete symptoms, which are related to the ischemia of the inner ear circulation, atherosclerosis of VAS and decreased blood flow in the VAS may cause damage to the inner ear [9]. Degenerative changes were found in the cochlea and superior vestibular labyrinth of a case with sudden hearing loss and vertigo in a postmortem study. It was concluded that the internal auditory artery infarction developed due to decreased perfusion in the vertebrobasilar system [1]. VBI was investigated using MRI in 51 patients with SHL in a study. Vertebrobasilar blood flow was detected slow in 21% of all cases (12 cases), and all patients had vertigo [10]. It was thought that vertigo complaint in our case might be a resourced of VBI and the presence of VBI on

the left might have triggered SHL in the left ear. The cervico-ocular reflex (COR), one of the proprioceptive reflexes of the neck, terminates the information which receives from the proprioceptives in the upper cervical vertebral joints and neck muscles in the contralateral vestibular nucleus [11]. It shows a synergistic effect with the VOR in abnormal situations such as increased muscle tension in the neck. Asymmetric neck proprioception induced by COR causes optokinetic after nystagmus, imbalance and vertigo [11]. It was thought that low left optokinetic gain in the first VNG evaluation during the initial period of SHL may be caused by a possible problem on the left neck region in our case. It supported VBI suspicion on the left. Computerized dynamic posturography (CDP) is known to objectively show effect of the neck on balance and postural performance in patients with suspected cervical problems and VBI. The depression of the cervical canals can affect postural control by forced to the spinal cord and tract, where proprioceptive information is transmitted. It was reported that balance exercise was benefit to the patients with vertigo induced by VBI [12]. The obvious decrease was seen in SOM and VEST scores according to the first SOT results in our case. The right lateralization of the COG suggested hypofunction on the left. After one month of VR, SOT scores which between normal limits may suggest that the VBI problem was not required surgical intervention. It was very promising that the improvement in SOM and VEST scores, his complaints and the quality of daily life with even one month of VR.

It is known that the presence of thromboembolism causing vascular occlusion can cause SHL [3]. SHL has been shown to be associated with several circulatory system diseases, such as stroke and myocardial infarction. SHL was an independent risk factor for the development of myocardial infarction in a study [13]. It was thought that presence of cardiovascular problems and using pacemakers in our case may be a risk factor for SHL. In addition, hypertension and advanced age are known to be risk factors for VBI [3]. The prevalence of hyperlipidemia, diabetes mellitus and hypertension were higher among patients with SHL [3, 14]. The presence of hypertension in our case might have increased the risk of VBI. The first limitation of this report was that L-VHIT results could not be compared before/after VR. L-VHIT was not applied in the first vestibular evaluation and the possible improvement could not be seen in L-VHIT results. Another limitation was that VHIT is not applied for all semicircular canals (SSC). In the validity and reliability studies of VHIT, it was reported that the lateral canal has higher sensitivity and specificity compared to vertical canals [15]. It was seen that gain asymmetry was 9% in favor of weakness on the left, presence of low VOR gain and covert saccades on the left in L-VHIT in this case. These findings also supported VBI suspicion on the left. It was thought no necessity to continue VHIT for vertical canals in clinical practice. The reason why the SHIMP test could not be performed is that the device is not available in our clinic. It would be appropriate to perform before and after treatment "limits of stability" and "rythmic weight shifting" tests to provide more information about the individual's adaptation to daily life by simulating challenging dynamic balance conditions. However, these tests in posturography could not be applied to this patient due to the lack of time allocated during the evaluation and rehabilitation process in the clinical routine. The rehabilitation process continued by monitoring this patient on SOT, one of the basic tests of posturography. In our clinic, the vestibular rehabilitation process of such patients is monitored with the SOT test. In brief, VBI which develops secondary to sudden blood pressure change due to stress or cardiac problems, had existed for a long time. SHL has been treatment with systemic and intratympanic dexamethasone, VBI may have triggered SHL,

although it is not a direct cause of SHL. This patient was unknowingly compensating for VBI, but the diagnosis was made by investigating the cause of the SHL and vertigo. If SHL was occurred due to acute decrease in blood supply, SHL might be improved with steroid support given to cochlear cells. The absence of feeling of vertigo and fatigue during positional tests before and after SHL treatment but the presence of geotropic nystagmus in all positions excluded benign paroxysmal positional vertigo. Positional body test and L-VHIT results suggest unilateral (left) hypofunction/pathology suspicion.

Based on our recent phone call with the patient who could not meet face to face due to the Covid-19 pandemic; he is describing dizziness in intermittent sudden head turns for one year. When he feels dizziness, he is doing the VR exercises that were given before. He has no auditory and/or vestibular complaints for the last 3 months. Vasodilator was given to this patient after the VBI diagnosis. He has used it regularly for 10 months, he is thinking that this treatment is very beneficial.

4. Conclusion

Vertebrobasilar insufficiency, although rare, is an etiologic option which should be considered in patients with SHL. Further research is needed to investigate the association among the severity of VBI and the risk of SHL. This case report was demonstrated in clinical practice the value of comprehensive audiological and vestibular evaluation, accurate interpretation of the findings in diagnosis and the importance of follow-up in patients who applied to Audiology clinics with complaints of SHL and concurrent vertigo, and also drew attention to the importance of VR in VBI.

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Informed consent: Informed consent was obtained verbally from this patient before the study and the principles in the Declaration of Helsinki were followed.

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