



Original Article

Temporal Bone Posterior Wall Defect, a New Clinic Entity?

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OBJECTIVE: To define clinical and audiological findings in patients with temporal bone posterior wall defects (TBPWD) and to investigate possible relationships between these findings and the characteristics of the defect.

MATERIALS and METHODS: The computed tomography (CT) views of 1198 patients with vestibulocochlear symptoms between 2007 and 2012 were retrospectively evaluated, and TBPWD and associated anomalies were investigated. Patients who had TBPWD were called back, and clinical and audiological examinations (tympanometry, pure tone audiometry, acoustic reflexes, and otoacoustic emission) were performed.

RESULTS: Twenty-eight (2.34%) patients had TBPWD. Twenty-three of them were eligible for the study. Size of the defect was significantly correlated with the presence of tinnitus and/or vertigo ($p < 0.005$). The cut-off values for the largest size of TBPWD were 1.65 mm [sensitivity: 0.67 and specificity: 0.77 (95% CI: 0.58–0.97); $p = 0.04$] in case of the presence of tinnitus and 1.85 for vertigo (sensitivity: 0.78 and specificity: 0.86 (95% CI: 0.67–0.99); $p = 0.006$). In pure tone audiometry tests, mixed-type hearing loss was present in four (17%) patients, sensorineural hearing loss was present in three (13%) patients, and conductive-type hearing loss was present only in one (4%) patient. Otoacoustic emission tests revealed significant differences in signal/noise ratios at frequencies of 500, 750, 1000, and 6000 Hz.

CONCLUSION: For the first time in the literature, we defined clinical and audiological findings in patients with TBPWDs. These defects seem to cause more prevalent symptoms of vertigo and tinnitus and disturb the audiological characteristics of patients.

KEYWORDS: Temporal bone, vertigo, tinnitus, audiometry

INTRODUCTION

The posterior wall of the temporal bone is thick, and it is rid of natural dehiscences when compared with the petrous bone, which is located in the middle cranial fossa. In the literature, arachnoid granulations originating from the posterior wall of the temporal bone, which result in temporal bone defects because of pulsations of the arachnoid villi, have been defined ^[1]. These bony defects may be overlooked by the clinicians, and rare but important complications may occur in the presence of this anomaly.

In two studies investigating temporal bone posterior wall defects (TBPWD), only histological and radiological data were available ^[2,3]. For the first time in our study, we aimed to define clinical and audiological findings in patients with TBPWD and to investigate the possible relationship between these findings and the characteristics of the defect.

MATERIALS and METHODS

Computed tomography (CT) views of 1198 patients admitted to our outpatient clinics with vestibulocochlear symptoms between 2007 and 2012 were retrospectively evaluated. TBPWD was found in 32 patients. After the exclusion of patients who had a history of ear surgery or cholesteatoma causing bone destruction, 28 (2.34%) patients were enrolled.

The local ethics committee approved the study protocol and informed consent was obtained from all participants.

Computed Tomography Imaging

A 16-detector CT machine (GE Lightspeed; GE Healthcare, Wisconsin, USA) was used for CT imaging. Imaging protocol parameters were as follows: scan time of 1 s, 0.6-mm section thickness, 140-kVp peak kilovolt, 260-mAs milliampere-second value, matrix of 512×512, 26-cm field-of-view (FOV), mean number of slices was 140, and mean volume computed tomography dose index (CT-DIvol). All CT images were produced in an axial plane without gantry angle. CT views of patients who had TBPWD were examined in a dedicated double-monitored workstation with 3-megapixel resolution (Advanced Workstation 4.2 GE Healthcare, USA). All images were retrieved from hospital-based digital radiological image archive (Centricity, GE Healthcare, USA). Two radiology residents examined CT images in high-resolution algorithm and at a window width of 4000 HU and a window level of 400 HU. They

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performed measurements together, and a final decision was reached regarding the existence of a bony defect and its size. Location of the defect in the posterior fossa on an axial plane was not mentioned in the evaluation. Bony defect measurements were taken in two planes perpendicular to each other. One experienced radiologist reviewed abnormal cases and made corrections. Other temporal bone anomalies, such as high jugular bulb, jugular dehiscence, and superior semicircular channel dehiscence, were also investigated.

Clinical and Audiological Evaluation of Patients

The hospital records of 28 patients were retrospectively evaluated, and patients were phoned and called back for physical and audiological examination. Five (17.8%) of the patients were unavailable; thus, they were excluded. Rest of the patients were invited to our clinics for detailed examination.

The presence or absence of tinnitus and vertigo was reported. After the physical examination of the ear, patients underwent audiological tests, including tympanometry (AZ 26 Clinical Audiometer; Interacoustics Assens, Denmark), pure tone audiometry (AC 33 Clinical Audiometer; Interacoustics Assens, Denmark), and otoacoustic emission [distortion product otoacoustic emission, DPOAE; (Integrity V500; Vivosonic, Toronto, Canada)].

In a completely isolated cabin using hearing thresholds that were obtained between the frequencies of 250–8000 Hz pure tone averages were determined. Audiogram results were classified into the three following groups: conductive-type hearing loss (CHL), mixed-type hearing loss (MHL), and sensorineural-type hearing loss (SNHL).

Acoustic stapes reflex (ASR) and tympanograms of patients were obtained. ASR was separately interpreted as present or absent at 500, 1000, and 2000 Hz. Tympanogram curves were interpreted as type A, B, C, and As^[4].

DPOAE measurements for both ears were made in an acoustically isolated room. DPOAEs (2f1-f2) were evoked by the system using an f2/f1 ratio of 1.2. For L1, the amplitude of the stimulus was 65 dB SPL, and for L2, it was 55 dB SPL. At a constant stimulus amplitude and time, distortion-dependent otoacoustic emission data were saved at different frequency zones from low-to-high frequency range of f2=0.5–8.0 kHz, and distortion dependent audiogram was attained. In a patient with CHL who had sclerosis at the bilateral mastoid bone, no acoustic reflexes, and a type B curve on the tympanogram, a middle ear disease was also thought to be accompanied. Except this patient, all patients underwent the DPOAE test. In patients with unilateral TBPWD, signal/noise ratio (SNR) values were also calculated.

Statistical Analysis

Data analysis was performed using SPSS for Windows, version 15.0 (SPSS Inc.; Chicago, IL, USA). Whether continuous variables were distributed normally or not was determined using the Kolmogorov–Smirnov test. Data were shown as mean±standard deviation and median range (minimum–maximum). Normally distributed variables were compared with t-test and non-normally distributed variables with Mann–Whitney U-test. For categorical variables, chi-square test was used. Degrees of associations between continuous variables were calculated by the Spearman's correlation coefficient. Receiver-operating characteristics analysis was used to determine the cut-off values. A p value of <0.05 was considered to be statistically significant.

RESULTS

After the exclusion of patients who are ineligible for the study, 23 patients [12 (52.2%) women; mean age: 52.0±16.6] were enrolled. TBPWD was present at the right side in eight (34.8%) patients, at the left side in 13 (56.5%) patients, and bilaterally in two (8.7%) patients. All TBPWDs were non-lytic circular defects with irregular borders, and they did not have calcification or bony particles inside (Figure 1). The mean largest diameter of defects was calculated as 1.51±1.15 mm.

Two (8%) patients had high jugular bulb at the same side with the defect. Other two (8%) patients have jugular dehiscence, and one (4%) patient had carotid dehiscence ipsilaterally located. In all, four (17%) patients had superior semicircular channel dehiscence (SSCD) accompanying TBPWD, with three patients having ipsilaterally located and one having bilaterally located SSCD.

A significant positive correlation was found between age and size of the TBPWD ($r=0.581$; $p=0.004$; Figure 2).

The symptomatology of patients was as follows: three (13.0%) of the patients had vertigo, six (26.1%) had tinnitus, and three (13.0%) had both symptoms. Out of 23 patients, 11 (47.8%) were asymptomatic by means of vestibulocochlear symptoms. The size of the TBPWD was higher in patients who had tinnitus (2.33 ± 1.21 vs. 0.98 ± 0.74 mm; $p=0.006$) and in patients who had vertigo (2.12 ± 1.15 vs. 1.29 ± 1.10 mm; $p=0.048$). As other anomalies in the temporal bone may be responsible for the symptoms, we also separately investigated 16 patients without additional anomalies and found similar results in patients who had tinnitus (2.40 ± 1.28 vs. 1.05 ± 0.83 mm; $p=0.022$). However, size of the defect did not differ between patients with and without vertigo (1.75 ± 0.95 vs. 1.43 ± 0.31 mm; $p=0.38$).

The cut-off values of the maximum size of TBPWD were 1.65 mm [sensitivity: 0.67 and specificity: 0.77 (95% CI: 0.58-0.97); $p=0.04$] for the presence of tinnitus and 1.85 for vertigo [sensitivity: 0.78 and specificity: 0.86 (95% CI: 0.67-0.99); $p=0.006$; Figures 3A and B]. When patients with additional anomalies were excluded, cut-off values of the max-



Figure 1. CT view of an 81-year-old woman with a defect of size 39 mm in the posterior wall of the temporal bone (arrow) located at the left side

imum size of TBPWD for tinnitus were 1.65 mm [sensitivity: 0.80 and specificity: 0.82 (95% CI: 0.68-0.99); $p=0.023$; Figure 3c]. However, no relationship between the size of TBPWD and the presence of vertigo was found after the exclusion of patients with additional anomalies.

One patient had a type B curve in the tympanogram, and ASR was not found. All the other patients had type A curves and ASRs were present. In pure tone audiometry tests, MHL was present in four (17%) patients, SNHL was present in three (13%) patients, and CHL was present only in one (4%) patient.

In 21 patients with unilateral DPTWB, we compared our study parameters between defective and non-defective sides. In pure tone audiometry tests, hearing thresholds did not differ between patients with and without TBPWD. However, at 1000 Hz and 1500 Hz DPOAE measurements were significantly lower at the defective side (17.48 ± 5.9 dB vs. 19.41 ± 6.5 dB; $p=0.006$ and 18.63 ± 3.9 dB vs. 20.81 ± 4.8 dB; $p=0.003$, respectively).

In 14 patients with unilateral TBPWD without additional anomalies, hearing thresholds at 2000 Hz were significantly higher for bone conductions at the defective side (25.71 ± 16.4 dB vs. 21.07 ± 12.43

dB; $p=0.046$). In these patients, we also investigated SNR at different DPOAE frequencies and found significant differences between the defective side and anatomically normal side (Table 1).

DISCUSSION

Defects are rarely seen in the thick, posterior portion of the temporal bone. Ear infections, cholesteatoma, surgical procedures, trauma, tumors, radiotherapy, and congenital defects can be counted in etiology. Besides, arachnoid granulations, which may cause spontaneous otorrhea, can also play a role in the formation of bony defects [5].

Arachnoid granulations are formed by the overgrowth of the arachnoid membrane to the dural sinuses [6]. In addition to spontaneous otorrhea, these lesions may also cause meningeal tumors [7]. The defects formed by arachnoid granulations are usually lobulated with irregular borders, and the radiologic appearance of the defect is almost always sufficient to diagnose arachnoid granulations [8]. In our study, the radiologic appearances of all defects were compatible with arachnoid granulations. Although unlikely, they could not be differentiated from the emissary veins of the posterior fossa because magnetic resonance and/or CT angiography views were not available. Endolymphatic sac tumors may be another cause of TBPWD. However, in our study, none of the patients had calcification at the lesion borders, bony particles inside the lesion, and abnormal localization of the endolymphatic sac. As a result, the diagnosis of endolymphatic sac tumor was ruled out.

Scarce data in the literature have shown that TBPWDs are usually associated with serious life-threatening complications such as spontaneous otorrhea and meningitis [3]. In our study, we disclosed that these lesions may also cause common otological symptoms, i.e., tinnitus and vertigo. It is important because an underestimation of these common symptoms may cause the clinician to miss out this potentially life-threatening disease.

SSCD was present in four (17%) patients in our study. The incidence of dehiscence of the superior semicircular channel, which completes its ossification first, is between 3.6%–4.9% [9, 10]. The higher incidence of this anomaly in patients with TBPWD may support the hypothesis, implying that multiple defects may occur in case of discontinuation of ossification in the temporal bone at several steps in the postpartum period [11]. However, if we consider that TBPWD may occur because of arachnoid granulations or idiopathic increases of the intracranial pressure at older ages, it may be said that erosion of the thin

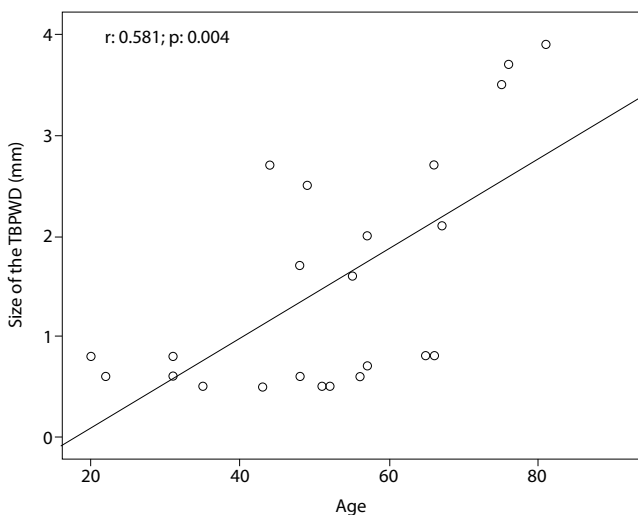


Figure 2. Correlation between age and size of the TBPWD

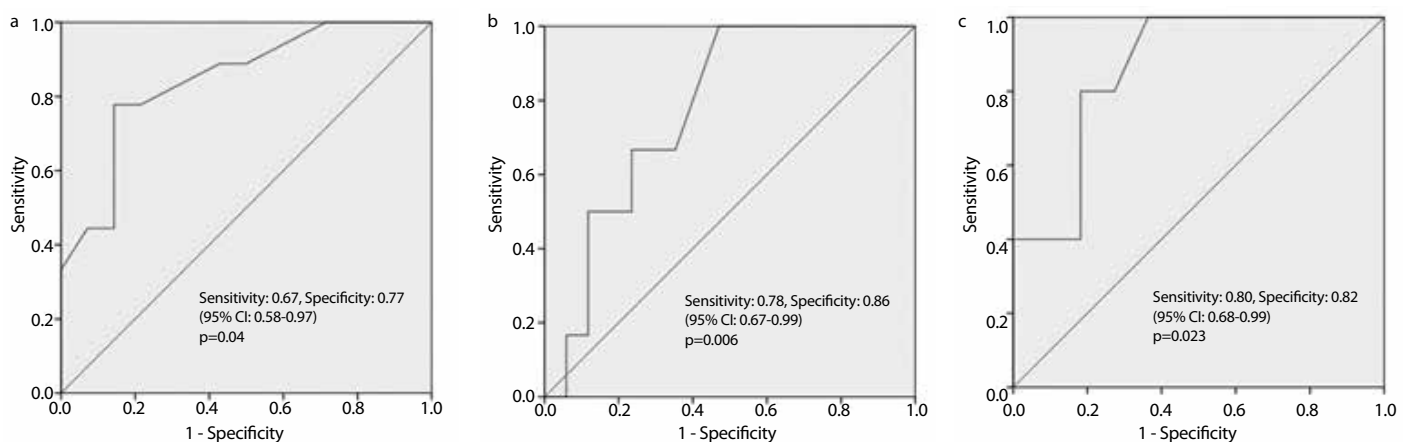


Figure 3. a-c. Receiver-operating characteristics (ROC) curve analysis of the size of the defect and symptoms tinnitus (a), vertigo (b), and tinnitus (c) in patients without additional anomalies

Table 1. Signal noise ratio (SNR) comparisons between defective and non-defective sides at different DPOAE frequencies in patients with defect in the posterior wall of the temporal bone

DPOAE frequency	TBPWD (n=14)	SNR	p value
500 Hz	Absent	7.6 (2.7–19.8)	0.018
	Present	5.4 (1.2–14.4)	
750 Hz	Absent	16.9 (10.2–20.5)	0.023
	Present	11.6 (–2.4–21.0)	
1000 Hz	Absent	21.1 (11.5–30.1)	0.001
	Present	19.9 (5.1–21.8)	
1500 Hz	Absent	19.4 (12.7–27.4)	0.036
	Present	17.8 (11.3–24.1)	

Median (min–max) values are given for SNR values.

DPOAE: distortion product otoacoustic emission; TBPWD: temporal bone posterior wall defect; SNR: signal/noise ratio

tegmen tympani with time has an increased possibility in causing TBPWD^[12–15]. In our study, older patients had larger TBPWDs in size, which was consistent with this theory.

The phenomenon of conductive hyperacusis has been defined by Stenfelt et al.^[16] in subjects with SSCDs. Patients with this disorder may hear their eyeball movements, their heart beat at night, the movement of their joints while jogging, or their own voice too loudly in the ear with superior semicircular canal dehiscence. This symptom can be very disturbing as well as a hindrance to the patients' life. The cause of conductive hyperacusis is the negative bone conduction in certain subjects with SSCDs, which creates a higher sensitivity to bone-conducted sound at frequencies below 2 kHz. Interestingly, in our study, patients with TBPWD did not have conductive hyperacusis. A second defect in the bony conduction may prevent the development of conductive hyperacusis.

In our study, high jugular bulb was defined when the jugular bulb was more superior than the inferior annulus of the tympanic membrane. The incidence of high jugular bulbs varies among authors and studies because of the variation in its definition. It was found to be approximately 3% when the inferior annulus had been selected as the reference structure^[17]. Again, we have found an increased incidence of high jugular bulbs (8%) in patients with TBPWD, which supports the general knowledge implying that temporal bone anomalies commonly accompany each other.

In our study, the pattern of DPOAE decrease was similar to the pattern of patients who had tinnitus after exposure to noise^[18, 19]. We thought that this phenomenon may be due to the lack of a barrier at the posterior temporal bone, which may cause an increased effect of the inner voice on the external hairy cells in the cochlea. However, further studies should be conducted to explain this finding.

For the first time in the literature, we defined clinical and audiological findings in patients with TBPWDs. These defects seem to cause more prevalent symptoms of vertigo and tinnitus and disturb the audiological characteristics of the patients.

Ethics Committee Approval: The approval of local Institutional Review board has been obtained (04.09.2013/2).

Informed Consent: Written informed consent has been obtained from all participants.

Peer-review: Externally peer-reviewed.

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