



## Tinnitus in the side with better hearing

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### ABSTRACT

**Objectives:** We aimed to confirm the characteristics of patients with tinnitus in the better-hearing side.

**Materials and methods:** Among the 778 patients who visited the tinnitus clinic complaining of unilateral tinnitus at a local university hospital between March 2014 and December 2017, we recruited 62 patients who showed tinnitus in the better-hearing side on pure-tone audiometry. The mean hearing threshold was calculated using the arithmetic mean of the pure tone thresholds at 1, 2, 3, and 4 kHz. In addition, patients' medical history, tinnitus questionnaires, and other audiologic test results were thoroughly analyzed together for diagnosis.

**Results:** Fluctuating hearing loss without vertigo or Ménière's disease were the most common etiologies (n = 16, 25.8%), followed by high-frequency hearing loss (n = 13, 21.0%), sudden idiopathic hearing loss (n = 6, 9.7%), and presbycusis (n = 6, 9.7%). Somatosensory tinnitus was also observed in seven patients. Neck pain was associated with tinnitus in five patients (8.1%), and two other patients (3.2%) experienced temporomandibular disorder in the same side as the tinnitus.

**Conclusion:** Tinnitus was associated with deterioration of hearing even when it occurred in the better-hearing side. Among the possible etiologies, fluctuating hearing loss in the tinnitus side was the most common audiologic finding. Assessment of hearing level at each frequency was more effective in detecting high-frequency hearing loss rather than the use of the mean hearing level. In addition, somatosensory tinnitus should not be ignored.

### 1. Introduction

Tinnitus is generally considered to be associated with auditory deprivation [1]. Reduced peripheral input induces changes in the audio-somatosensory integration in the cochlear nucleus, after which tinnitus-related neural hyperactivity is induced by an increase in gain [2,3]. Thus, cochlear function may play an important role in the tinnitus percept. In addition, functional coupling and alterations at multiple levels of the central auditory pathway are involved in the tinnitus percept by promoting abnormal propagation of tinnitus-related neural activity [4].

High-frequency hearing loss or extended high-frequency hearing loss above 8 kHz is sometimes observed in tinnitus sufferers with normal hearing [5]. Extended high-frequency audiometry along with otoacoustic emissions can elucidate the hidden high-frequency hearing loss that cannot be confirmed in usual pure-tone audiometry [6]. In this regard, other studies have noted that the tinnitus pitch is correlated with the frequency of maximum hearing loss, suggesting that tinnitus is a fill-in-phenomenon resulting from homeostatic mechanisms [7].

However, the tinnitus pitch does not completely match the audiogram pattern [8]. Contrary to the general expectation, some patients complain of tinnitus in the better-hearing side. In addition, the severity of tinnitus distress has been recently reported to be unrelated to the

hearing level [9,10]: in one study, the severity of tinnitus was not correlated with the degree of sudden hearing loss [9], whereas the other study showed that for orchestra musicians, increased lifetime sound exposure, not the high-frequency hearing loss, was associated with the tinnitus distress level [10]. Similarly, a recent study reported that tinnitus was more disturbing in patients with normal hearing than in those with hearing loss because the latter tend to perceive tinnitus only in very calm surroundings [11].

Determination of the etiologies underlying tinnitus is the most important initial step for treatment of tinnitus, since this information determines the type or order of treatments that the patients are given [12]. To our knowledge, the characteristics of patients with tinnitus in the better-hearing side have rarely been studied. Thus, management of these patients may be confusing for clinicians. In this study, we aimed to assess the patient characteristics and final diagnosis in cases showing unilateral tinnitus in the better-hearing side.

### 2. Material and methods

Between March 2014 and December 2017, 778 patients visited the tinnitus clinic at a local university hospital for treatment of unilateral tinnitus. The inclusion criteria were as follows: (1) age > 18 years, and (2) mean hearing threshold in the tinnitus side better than that on the

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contralateral side. The mean hearing threshold was calculated using the arithmetic mean of the pure tone thresholds at 1, 2, 3, and 4 kHz. Patients who did not undergo pure-tone audiometry were excluded. The institutional review board (IRB) of the university hospital approved this study. The IRB granted a waiver of written informed consent for this retrospective study.

Careful history-taking for the tinnitus as well as patient characteristics, including age, sex, laterality, accompanying symptoms such as dizziness, aural fullness, headache, and subjective feelings of anxiety/depression were documented at the initial visit. For assessment of subjective tinnitus distress, scores for the tinnitus handicap inventory (THI) and Beck depression inventory (BDI) were obtained at each follow-up. In addition, visual analogue scale (VAS) (0–10: 0: No, 10: maximal) scores for tinnitus loudness were also evaluated. For patients with sudden idiopathic hearing loss and presbycusis, brain magnetic resonance imaging (MRI) was performed to confirm whether they had central lesions. For patients with suspected accompanying hyperacusis, loudness discomfort level tests were performed. All of these described above were thoroughly analyzed together for diagnosis.

The Shapiro–Wilk test was used to assess normality. Comparison of paired numeric values was performed by the paired *t*-test and the Wilcoxon signed-rank test. All statistical analyses were performed using the SPSS for Mac software package (ver. 25.0, SPSS Inc., Chicago, IL, USA), with a *p*-value < 0.05 taken to indicate statistical significance.

### 3. Results

A total of 62 patients (29 male, 33 female) were finally recruited, and their data were analyzed. The patients' mean age was 49 years (range: 18–76 years, standard deviation [SD]: 14.36 years). With respect to laterality, 24 (38.7%) patients had tinnitus in the right side and 38 (61.3%) had left-sided tinnitus. The mean duration of tinnitus was 11.92 ± 32.07 months (range: 1–180 months). With respect to accompanying symptoms, 14 (22.6%) showed dizziness and 29 (46.8%) experienced aural fullness. In addition, 18 (29.0%) reported headaches and 20 (32.3%) experienced a feeling of anxiety/depression. The mean hearing level at the tinnitus side was 13.05 ± 12.08 dB, which was significantly better than that on the contralateral side (19.74 ± 21.15 dB) (*p* < 0.001, paired *t*-test). In questionnaire assessments, the THI and BDI scores at the initial visit were 38.24 ± 19.92 and 9.26 ± 6.75, respectively. The VAS score for loudness at the initial visit was 4.53 ± 2.23.

The possible etiologies in our patients are shown in Table 1. Two cases in which we could not find any reasonable etiologies were

categorized as “unidentified.” The remaining 60 patients had one or more main problem at least that might be associated with tinnitus percept. As a result, even among the patients who had tinnitus in the better-hearing side, 75% (45 of 60) had tinnitus related to changes in auditory perception. These etiologies included fluctuating hearing loss with or without vertigo, high-frequency hearing loss, sudden hearing loss, presbycusis, hyperacusis, and acoustic trauma. In 23% of the patients (*n* = 14), tinnitus was mainly associated with a somatosensory origin such as neck pain, temporomandibular joint (TMJ) disorder, migraine, high jugular bulb, and Eustachian tube dysfunction. The last patient (*n* = 1) had a psychiatric problem and was transferred to the department of psychiatry due to a suicidal attempt.

The initial THI was the highest in patients with hyperacusis, followed by those with presbycusis, high-frequency hearing loss, and TMJ disorder. The worst BDI score was observed in the patients with TMJ disorder, followed by those with high-frequency hearing loss and presbycusis. Patients with hyperacusis experienced the loudest tinnitus, followed by those with presbycusis and those with a venous hum.

Fluctuating sensorineural hearing loss (*n* = 16, 25.8%) was the most common audiologic finding. The patients' hearing level showed both improvement and aggravation repeatedly during the follow-up period. Among these, nine patients experienced accompanying or late-developed dizziness during the follow-up period and were finally diagnosed as showing Ménière's disease (MD). However, in the other seven patients, dizziness did not develop during the entire follow-up period. Interestingly, worsening of the final hearing threshold in the tinnitus side was observed during the follow-up period in some patients (*n* = 2, 12.5%). In these patients, the final hearing in the tinnitus side worsened in comparison with that in the contralateral side.

High-frequency hearing loss (*n* = 13, 21.0%) was the second-most common audiologic finding. Among these patients, four patients with normal hearing from 250 Hz to 8 kHz showed ipsilateral extended high-frequency lesions above 10 kHz. Although the mean difference in pure-tone thresholds was statistically significant between the tinnitus side and the contralateral side, the speech reception thresholds (SRTs) were not different (*p* = 0.655). In addition, the hearing thresholds at each frequency did not show any statistical differences related to the presence of tinnitus (*p* > 0.05).

Six patients experienced sudden idiopathic hearing loss in the tinnitus side. All of them had previously existing hearing loss in the contralateral side, and brain MRI confirmed that they had no retrocochlear lesions. Another six complained of progressive hearing loss in the tinnitus side and were finally diagnosed as having presbycusis. Similar to the patients with sudden hearing loss, all of them had previously

**Table 1**  
Various etiologies associated with tinnitus in the side with better hearing.

Causes	Number	Age	Onset (month)	Initial THI	BDI	Tinnitus loudness (1–10)	Mean PTA in the tinnitus side	Mean PTA in the contralateral side
Fluctuating hearing loss (with or without vertigo)	16	50 ± 16	14.17 ± 34.04	35.54 ± 19.99	9.08 ± 6.40	4.17 ± 0.75	11.50 ± 13.14	14.25 ± 13.65
High-frequency hearing loss	13	49 ± 11	3.67 ± 7.63	44.17 ± 25.79	11.42 ± 8.12	4.67 ± 3.28	12.69 ± 12.23	18.85 ± 17.90
Sudden idiopathic hearing loss	6	57 ± 13	1.50 ± 0.837	28.00 ± 8.485	5.42 ± 4.23	3.23 ± 0.75	18.83 ± 12.66	36.00 ± 35.46
Presbycusis	6	62 ± 11	59.20 ± 77.91	45.20 ± 5.93	11.20 ± 7.95	7.00 ± 0.82	24.17 ± 15.97	39.67 ± 36.30
Somatic tinnitus associated with neck pain	5	49 ± 15	9.00 ± 9.46	23.60 ± 12.28	7.75 ± 6.40	2.80 ± 1.10	13.05 ± 12.08	19.74 ± 21.15
Venous hum	3	46 ± 9	6.50 ± 7.78	40.67 ± 14.05	9.33 ± 1.53	5.30 ± 0.79	8.00 ± 12.17	12.67 ± 15.14
Hyperacusis	3	39 ± 16	3.33 ± 2.31	48.00 ± 11.31	10.00 ± 12.73	7.33 ± 0.58	4.51 ± 2.60	4.58 ± 2.64
Somatic tinnitus associated with TMJ disorder	2	29 ± 2	4.50 ± 4.95	37.00 ± 24.04	12.50 ± 2.12	4.50 ± 0.71	0.5 ± 0.71	1.5 ± 0.71
Migraine	2	62 ± 16	1	16.00 ± 2.83	3.50 ± 0.71	3.00 ± 0.00	8.49 ± 6.00	10.61 ± 7.50
High jugular bulb	1	35	7	16	3	1	8	11
Acoustic trauma	1	52	2	34	3	3	10	12
Eustachian tube dysfunction	1	58	1	18	3	3	12	45
Anxiety/depression	1	34	6	48	25	3	3	6
Unidentified	2	33 ± 10	3.00 ± 0.00	77.00 ± 1.41	8.00 ± 5.66	3.50 ± 0.71	4.5 ± 3.54	7.0 ± 4.24

existing sensorineural hearing loss in the contralateral side. In addition, they complained of new-onset tinnitus with concurrent ipsilateral progressive hearing loss that had worsened over time recently from several months to years ago. Brain MRI did not reveal brain lesions in any of these patients. Interestingly, the mean pure tone thresholds and SRTs of both sides in these two groups was significantly worse than those in the other etiologies ( $p < 0.05$ ).

Five patients had unilateral tinnitus associated with neck pain. Somatic modulation was possible in four of these five patients (80%). Two patients (40%) showed accompanying sleep disturbances. None of the patients had a noise-exposure history, and none of them showed subjective hearing loss either. Interestingly, the mean age of the patients with TMJ disorder was  $29 \pm 2$  years, and these patients were younger than those showing other causes. In addition, somatic modulation on the TMJ deteriorated their tinnitus shortly in both of these patients. For Eustachian tube dysfunction, the patient had a history of contralateral sudden idiopathic hearing loss. Eleven months after a partial recovery, he heard intermittent opening sounds in the other side when he yawned or swallowed something and was finally diagnosed as showing Eustachian tube dysfunction.

#### 4. Discussion

In this study, we found that about 8% of tinnitus sufferers complained of tinnitus in the better-hearing side and of these, 75% also showed deterioration of hearing. Most of the other remaining patients had somatosensory tinnitus. Fluctuating hearing loss with or without vertigo was the most common audiologic condition that caused tinnitus in the better-hearing side.

In the past, fluctuating auditory symptoms without vertigo and/or dizziness have been often referred to as cochlear MD. However, recent guidelines have included the presence of vertigo as a prerequisite for the diagnosis of MD [13]. For a diagnosis of definite MD, two or more episodes of spontaneous vertigo lasting 20 min to 12 h are needed [13]. Even probable MD requires two or more episodes of vertigo or dizziness lasting 20 min to 24 h [13]. A Belgian group suggested that patients with only auditory symptoms without vertigo represent a separate disease with Ménière-like pathophysiology or endolymphatic hydrops [14]. They used the Ménière's disease index (MDI), which assesses hearing thresholds at 125 Hz and 8000 Hz and the summing potential amplitude at 4000 Hz with tone-burst stimulation. Thus, the MDI score tends to increase from non-MD to definite MD; the MDI scores of those who had only auditory symptoms were between those for definite MD and probable MD [14].

As a pathologic marker, endolymphatic hydrops is associated with Ménière's disease irrespective of the presence of typical symptoms [15]. It was frequently found in patients with fluctuating hearing loss without vertigo by MRI [16] and especially in patients who reported tinnitus as their chief complaint [17]. In addition, resolution of endolymphatic hydrops confirmed by three-dimensional fluid-attenuated inversion recovery MRI was correlated with alleviation of symptoms in patients with MD [18]. Paparella mentioned that patients with only dizziness symptoms or hearing symptoms often develop the other symptoms over time and are finally confirmed to have Ménière's disease, and among these, up to 50% of the patients who experience tinnitus, hearing loss, and aural pressure may show improvement following endolymphatic sac surgery [19].

In fact, tinnitus is not the most representative symptom in MD. One study reported that only 19% of the patients with MD considered tinnitus as their most severe symptoms. However, tinnitus-predicting symptoms include aural fullness, hearing loss, and dizziness, suggesting that treatment of tinnitus in MD requires concomitant treatment of all audio-vestibular symptoms [20]. Herraiz et al. reported that longer duration, bilateral involvement, hearing loss, hyperacusis, and higher MD staging were associated with increased tinnitus loudness and tinnitus-related distress in patients with definite MD [21]. Moreover,

tinnitus in MD is closely related to anxiety, sleep, and depression [22]. This relevance to psychologic conditions is important because tinnitus-related distress itself may persist or be aggravated in patients complaining of acute tinnitus with depression over time, irrespective of their diseases [23].

On the other hand, fluctuating hearing loss without vertigo or dizziness can occur not only in MD but also cochlear migraine, which was proposed by a Taiwanese group [24]. According to their proposal, the other clinical features in such cases included aura, tinnitus, family history of migraine or chronic headache, unilateral neck stiffness, hypersensitivity to light, sound, or atmospheric pressure changes, and motion sickness or visual motion intolerance [24]. Although migraine usually does not affect the auditory pathway, a recent study with a large sample size reported that patients with migraines were twice as likely to develop auditory symptoms than a normal cohort [25]. Others assumed that migraine-related audio-vestibular symptoms may result from vasospasm of the posterior cerebral circulation by compromised blood flow to the inner ear [26]. One of our patients had a history of migraine and was being treated with medication to prevent relapse.

High-frequency hearing loss was the second-most common cause of tinnitus in the better-hearing side. We found many patients who met this criterion had normal hearing levels from 1 kHz to 4 kHz, but the hearing levels above 6 kHz were higher than those on the other side. This implies that the clinician should focus on the hearing level at each frequency rather than the mean value of the hearing threshold. Some patients with acute tinnitus and extended high-frequency hearing loss only above 8 kHz responded to steroid treatment, similar to those with idiopathic sudden hearing loss [27]. Other studies have reported that older patients with greater tinnitus distress tended to have extended high-frequency hearing loss above 8 kHz [28]. In addition, the hearing asymmetry and the laterality of tinnitus was correlated [28].

In fact, hearing loss including high-frequency hearing loss, sudden idiopathic hearing loss, and presbycusis may be one of the typical evidences suggesting that auditory deprivation is associated with tinnitus. Using an earplug led to artificial auditory deprivation in an experiment, and a casual relationship between auditory deprivation and the new-onset tinnitus was highly suggested by this approach [29]. The functional organization of the cortical and subcortical networks can be destabilized by auditory deprivation, and can eventually lead to dysfunctional neural circuits with neural correlates such as increased excitability, neural synchronization, and spontaneous firing rates in neurons [30]. Other researchers insisted that abnormally increased spontaneous firing rate in the auditory system results in abnormally increased neural gain, which could be spontaneous or stimulus-evoked. These changes can be recognized as tinnitus, sound intolerance, or hyperacusis [31]. Similarly, a questionnaire study with a large sample size reported that hearing loss was associated with awareness and loudness of tinnitus. In addition, anxiety sensitivity had a greater effect on annoyance in tinnitus and quality of life [32]. Interestingly, workers with a history of tinnitus in conjunction with high-frequency hearing loss showed a 25% increased acute injury risk. Hearing asymmetry showed no relation to injury risk [33].

Next, we found that some patients had concurrent neck pain or pain around the TMJ with tinnitus and somatic modulation was possible in most cases. Similarly, previous studies have reported that about 40% of tinnitus patients suffer from somatosensory changes of the cervical spine or temporomandibular area; somatic modulation is observed in up to 65%–80% of all tinnitus patients [34,35]. Somatosensory tinnitus is caused by somatosensory-auditory interactions [34–39].

In a previous study, TMJ disorder was associated with unilateral tinnitus, and TMJ + cranio-cervical dysfunction was associated with bilateral tinnitus [36]. TMJ maneuvers were reported to aggravate the tinnitus loudness [36,37]. For diagnosis of somatosensory tinnitus related to TMJ disorder, history-taking may be the most important. About 80% of patients who had a self-reported history for TMJ dysfunction plus positive somatic modulation test results for the TMJ region were

finally diagnosed as having TMJ disorder [38,39]. Female gender, younger age, modulation of tinnitus by somatic maneuvers or acoustic stimulation were predictable factors for tinnitus patients with TMJ disorders [40].

In addition to TMJ disorder, cervicogenic somatic tinnitus (CST) is sometimes used to represent the coincidence of both neck pain and tinnitus or an increase in both conditions [35]. The criteria for CST include (1) presence of head and neck trauma, (2) a history of teeth, jaw, or cervical spine manipulation, (3) recurrent pain in head, neck, and shoulder, (4) aggravation of tinnitus according to postures, and (5) bruxism [34,38]. For treatment of somatosensory tinnitus, Herraiz recommended muscular training, electrostimulation, or drugs [12]. A recent systemic review reported that cervical spine treatment (manipulations, exercises, trigger-point treatment) or TMJ treatment (splints, occlusal adjustments, jaw exercises) had positive effects on tinnitus. However, the evidence level in the studies conducted to date was too poor to draw a conclusion due to methodologic issues [41].

The limitations of our study are as follows. First, for calculation of the mean hearing level, we used the mean of thresholds at 1, 2, 3, 4 kHz. This approach may have aggravated the number of patients with high-frequency hearing loss above 6 kHz in the study. Second, some patients had multiple etiologies, but we chose the etiology that might have been most responsible because we wanted to focus on the main problems that caused the tinnitus. Thus, selection bias might have occurred. Third, the follow-up period differed according to the causes. Thus, it was difficult to compare the final results directly. As a result, we had no choice but to focus on the initial classification in this study. This resulted from the heterogeneous etiologies of tinnitus.

## 5. Conclusion

Tinnitus was associated with deterioration of hearing even in patients showing tinnitus in the better-hearing side. About 8% of patients complained of tinnitus in the better-hearing side. Fluctuating hearing loss with or without vertigo was the most common audiologic finding and some of them worsened their hearing in the follow-up. Assessment of hearing level at each frequency was effective than the mean hearing level in detecting high-frequency hearing loss. In addition, somatosensory tinnitus should be considered a relatively frequent cause of tinnitus in the better-hearing side.

## Conflict of interest

None to declare.

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