

Original Article

Clinical Features of Non-Lateralized Tinnitus

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Cite this article as: Shin S-H, Byun SW, Lee ZY, Park Y, Lee HY. Clinical features of non-lateralized tinnitus. *J Int Adv Otol.* 2023;19(6):497-502.

BACKGROUND: We aimed to analyze and confirm the clinical features of patients with non-lateralized tinnitus and to identify clues that can be used in their management.

METHODS: Data from 469 patients who visited a university hospital complaining of tinnitus between March 2020 and December 2021 were reviewed. The patients' medical histories, Tinnitus Handicap Inventory, Beck Depression Inventory, and numerical rating scale scores on tinnitus awareness, annoyance, loudness, and effect on life, audiological profiles, and quantitative electroencephalography findings were documented.

RESULTS: Forty-nine (10.4%) patients had non-lateralized tinnitus. They were older and had a shorter duration of symptoms (13.91 ± 34.16 months) than patients with bilateral tinnitus (duration: 39.15 ± 80.82 months) ($P < .05$). The accompanying symptoms, Tinnitus Handicap Inventory scores, and numerical rating scale scores were not significantly different between the 2 groups ($P > .05$). Patients with non-lateralized tinnitus had worse hearing at 12 kHz on the left side than those with unilateral tinnitus. Hearing asymmetry was least common in non-lateralized tinnitus ($n = 11/49$, 10.4%), followed by bilateral tinnitus ($n = 54/198$, 42.2%) and unilateral tinnitus ($n = 97/222$, 47.3%) ($P < .001$). Regarding quantitative electroencephalography, there were significant differences in the absolute power of the theta, alpha, beta, gamma, and total frequency bands based on tinnitus lateralization ($P < .001$).

CONCLUSION: Non-lateralized tinnitus can be perceived in elderly patients with symmetric and extended high-frequency hearing loss before habituation is achieved at an early stage of tinnitus. However, there was no difference in the questionnaire scores and accompanying symptoms; therefore, it may not be worth managing non-lateralized tinnitus separately from tinnitus in the ear.

KEYWORDS: Tinnitus, psychophysiology, hearing, high-frequency, surveys and questionnaires

INTRODUCTION

Tinnitus is the conscious awareness of a tonal or composite noise without an identifiable cause.¹ It is not a disease but a symptom that can be accompanied by a heterogeneous disorder. Understanding the pathophysiology of tinnitus is essential, and it can be explained by both top-down mechanisms, such as frontostriatal gating, and bottom-up mechanisms, like increased gain and thalamocortical dysrhythmia resulting from auditory deafferentation.^{2,3}

Therefore, a comprehensive evaluation is necessary to determine the underlying cause of tinnitus in each patient. This evaluation should include differentiating between pulsatile and non-pulsatile tinnitus, identifying the laterality of tinnitus, and investigating accompanying symptoms such as hearing loss, dizziness, autophony, cognitive impairment, neurologic deficits, previous history of loud noise exposure, current or past ototoxic medication, and accompanying somatosensory disorders.⁴ By identifying these factors, we can determine whether the tinnitus is primary or secondary and identify the underlying causes of secondary tinnitus. This is vital for developing effective treatment plans and improving the patient's quality of life.

Tinnitus can be classified based on its laterality into unilateral and bilateral tinnitus. In most cases, tinnitus is lateralized to the ear and tends to remain consistent in patients with chronic tinnitus.^{5,6} However, our previous study revealed differences in the mechanisms underlying unilateral and bilateral tinnitus.⁷ In patients with normal hearing, bilateral tinnitus may be associated with hyperactivity of the cochlear nucleus, while unilateral tinnitus may involve the higher cortical non-auditory area.⁷ It is important to note that many tinnitus patients, both with unilateral and bilateral tinnitus, may also have hearing loss. However, tinnitus can occur

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Received: August 12, 2022 • Accepted: June 26, 2023 • Publication Date: November 30, 2023

Available online at www.advancedotology.org



in patients with normal hearing as well, and further investigation is needed to fully understand the complex mechanisms that contribute to tinnitus in different patient populations. The laterality of tinnitus may also provide valuable clinical clues for ruling out secondary tinnitus. In unilateral cases, audiological tests may be necessary to determine the presence of asymmetric hearing loss, in addition to more thorough history-taking on the timing of hearing loss and the possibility of somatosensory tinnitus, based on a recent Delphi study and related papers.⁸⁻¹⁰ The experts involved in that study agreed that somatosensory tinnitus may be present if the patient experiences simultaneous neck or jaw pain, or if symptoms are exacerbated by such movements. Additionally, a history of head or neck trauma preceding the onset of tinnitus, varying psychoacoustic characteristics, increased tinnitus during certain postures, and discrepancies in audiogram results and unilateral tinnitus may also suggest the presence of somatosensory tinnitus. In contrast, bilateral tinnitus may be associated with age-related hearing loss, noise exposure, acoustic trauma, or otosclerosis.¹¹

Tinnitus can manifest in different ways, including as a tone or noise ringing unilaterally or bilaterally, or as a sound without an identifiable source or ringing from inside the head. However, there have been few studies addressing the characteristics and significance of non-lateralizing tinnitus. Some subjects initially described as having “non-lateralized” tinnitus may actually exhibit “bilateral” tinnitus upon detailed history-taking, which could explain why some subjects may reveal bilateral tinnitus in the tinnitus-matching test. However, due to the ambiguous nature of non-lateralized tinnitus, there remains limited understanding and a scarcity of research in this area. In this study, our aim is to address this gap in knowledge by analyzing the clinical features of patients with non-lateralized tinnitus and identifying key factors that can assist in their management. Through a comprehensive evaluation and investigation of the characteristics of non-lateralized tinnitus, we strive to contribute to the understanding and treatment of this specific subtype.

MATERIAL AND METHODS

Ethical Approval

The institutional review board of Ewha Mokdong Hospital University Hospital approved this retrospective cohort study and waived the need for written informed consent due to the retrospective nature of the study (IRB number: 2022-04-021-002).

Patients

The inclusion criteria were as follows: (i) Patients who visited a tinnitus clinic at a university hospital between March 2020 and December 2021 and (ii) patients with chronic tinnitus lasting 3 months or more. Documented variables included age, sex, accompanying symptoms

(aural fullness, headache, dizziness, attention problem, temporomandibular/neck pain, sleep disturbance, and history of exposure to loud noise), accompanying diseases such as diabetes mellitus and/or hypertension, and questionnaire results, including those from the Tinnitus Handicap Inventory (THI), Beck Depression Inventory (BDI), and numerical rating scale (NRS) (0 = no symptoms, 10 = maximal imaginable symptoms) for the assessment of tinnitus loudness, awareness, annoyance, and impact of tinnitus on life. Patients for whom THI was not documented were excluded from this study. For assessing tinnitus laterality, we utilized the following question: “Tell me where you hear your tinnitus. Does it come from one ear, both ears, or does it seem to be more spread out in your head?”

Audiological Tests and Hearing Asymmetry

The mean pure-tone hearing threshold was calculated as the arithmetic mean of hearing at 0.5, 1, 2, and 4 Hz. Hearing asymmetry was defined as an average interaural threshold difference of ≥ 15 dB at 2 adjoining frequencies.¹² Audiological tests performed included pure-tone audiometry, speech audiometry, auditory brainstem response, electrocochleography, and tinitogram consisting of pitch (Hz), loudness (dB SL), and residual inhibition (RI).

Resting-State Quantitative Electroencephalogram

For resting-state quantitative electroencephalogram (qEEG) analysis, scalp quantitative EEG with 19 electrodes was recorded using the MINDD scan (Ybrain, Seongnam-si, Gyeonggi-do, Republic of Korea) with a sampling rate of 500 Hz between 1 and 50 Hz. The international 10-20 electrode system was used in the placement of the electrodes. Before preprocessing, all data were imported into the EEGLAB toolbox for MATLAB (MATLAB R2021b, The MathWorks, Inc., Natick, Mass, USA). For preprocessing, we computed the average references for re-referencing, imported channel locations, applied the 1 Hz high-pass filter, applied clean_rawdata plugins, rejected bad channels, removed apparent artifacts, and conducted an independent component analysis using runica () to remove artifacts. Finally, the first 5 minutes of each artifact-free EEG were recorded. The power spectral density (PSD) was analyzed to calculate the absolute power at each frequency band.

Statistical Analysis

A descriptive analysis was conducted to evaluate patient characteristics. Student's *t*-test and/or one-way analysis of variance (ANOVA) was performed to compare the numerical data between non-lateralized and lateralized tinnitus. A post-hoc Tukey test was performed after ANOVA. Binary logistic backward conditional regression was used to identify the causal relationships between significant factors. For PSD, the absolute powers at each frequency band were compared using Student's *t*-test. All analyses were performed using Statistical Package for the Social Sciences for Windows version 27.0 (IBM SPSS Corp., Armonk, NY, USA). Statistical significance was set at $P < .05$.

RESULTS

Overall Patient Characteristics

Data from 469 patients were included in this study. The patients consisted of 246 men (52.5%) and 223 women (47.5%) with a mean age of 52.88 ± 15.03 years (range: 13-86) and a mean tinnitus duration of 29.40 ± 66.91 months. Regarding tinnitus laterality, right-sided, left-sided, bilateral, and non-lateralized tinnitus were noted in 91 (19.4%),

MAIN POINTS

- Forty-nine (10.4%) patients had non-lateralized tinnitus.
- Patients with non-lateralized tinnitus were older and had a shorter duration of symptoms.
- Hearing asymmetry was least common in non-lateralized tinnitus.
- It may not be worth managing non-lateralized tinnitus separately from tinnitus in the ear.

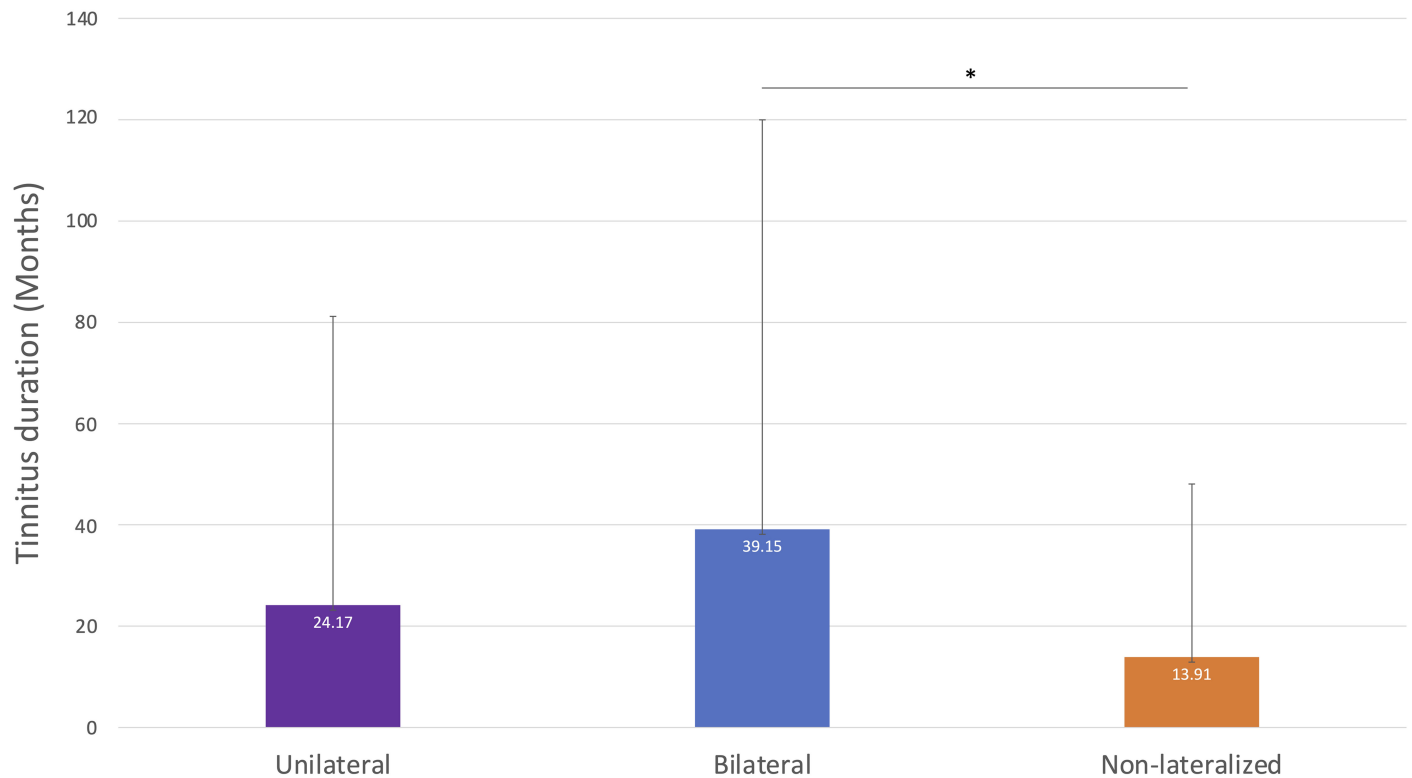


Figure 1. Comparative analysis of tinnitus duration in the unilateral, bilateral, and non-lateralized Tinnitus groups ($P < .05$).

131 (27.9%), 198 (42.2%), and 49 (10.4%) patients, respectively. The mean pure-tone thresholds of the right and left sides were 18.97 ± 16.47 and 19.40 ± 15.27 dB, respectively. Hearing asymmetry was present in 34.5% ($n = 162$) of the patients. The mean pitch and loudness were 3.44 ± 3.17 Hz and 6.89 ± 9.23 dB SL, respectively, on the right side, and 3.35 ± 3.14 Hz and 6.87 ± 11.25 dB SL, respectively, on the left side. Residual inhibition was measured in 43.5% ($n = 204$) of cases on the right side and 51.4% ($n = 241$) of cases on the left side. Complete and partial RI were observed on the right side in 43.6% ($n = 89$) and 2.9% ($n = 6$) of patients, respectively, and on the left side in 43.2% ($n = 104$) and 3.7% ($n = 9$) of patients, respectively. For the questionnaires, the initial THI and BDI scores were 45.60 ± 26.16 and 11.05 ± 9.24 , respectively. The initial NRS scores for awareness, annoyance, tinnitus loudness, and the effect on tinnitus were 7.36 ± 3.04 , 6.68 ± 2.77 , 6.35 ± 2.44 , and 5.19 ± 2.77 , respectively. Regarding accompanying symptoms, sleep disturbances were the most frequent (26.2%, $n = 123$), followed by aural fullness (23.9%, $n = 112$), headache (23.2%, $n = 109$), dizziness (22.2%, $n = 104$), attention problems (7.7%, $n = 36$), and temporomandibular/neck pain (7.7%, $n = 36$). Approximately 6.4% of patients ($n = 30$) had a history of exposure to loud noise. Diabetes mellitus and hypertension were noted in 8.1% ($n = 38$) and 19.2% ($n = 90$) of the patients, respectively.

Patient with Non-Lateralized Tinnitus

A total of 10.4% ($n = 49$) of tinnitus cases were not lateralized and were described as heard in the head or elsewhere. Patients with non-lateralized tinnitus were older (56.88 ± 13.31 vs. 52.41 ± 15.16 years; $P = .049$) and had a shorter duration of tinnitus onset (13.91 ± 34.16 vs. 31.24 ± 69.59 months; $P = .006$) than those with lateralized tinnitus. A post-hoc analysis revealed that the symptom duration of

non-lateralized tinnitus was shorter than that of bilateral tinnitus (39.15 ± 80.82 months) ($P = .005$) (Figure 1). None of the accompanying symptoms and the questionnaires significantly differed according to tinnitus lateralization ($P > .05$).

Regarding the audiological profiles, we compared the pure-tone thresholds at each frequency among all groups (Figure 2). The difference in hearing at 12 kHz on the left side was significant ($P = .034$). A post-hoc analysis showed that patients with non-lateralized tinnitus (84.17 ± 16.74 dB) had worse hearing at 12 kHz than those with unilateral tinnitus (66.60 ± 31.53 dB). However, no other significant group differences were observed in other audiological tests irrespective of lateralization ($P > .05$). Concerning the relationship between tinnitus laterality and hearing asymmetry, hearing asymmetry was least frequent in patients with non-lateralized tinnitus ($n = 11/49$, 10.4%), followed by bilateral tinnitus ($n = 54/198$, 42.2%) and unilateral tinnitus ($n = 97/222$, 47.3%) ($P < .001$, 2-sided exact test).

Comparison of Resting-State Quantitative Electroencephalography

The one-way ANOVA analysis revealed significant differences in theta-PSD, alpha-PSD, beta-PSD, gamma-PSD, and total PSD based on tinnitus laterality ($P < .001$) (Figure 3). Subsequent post-hoc tests specifically highlighted a significant distinction in alpha-PSD levels between unilateral, bilateral, and non-lateralized tinnitus cases (unilateral vs. bilateral tinnitus: $P < .014$, unilateral vs. non-lateralized tinnitus: $P = .002$, bilateral vs. non-lateralized tinnitus: $P = 0.002$). Additionally, alpha-PSD, beta-PSD, gamma-PSD, and total PSD exhibited significant differences between bilateral tinnitus and non-lateralized tinnitus, with the latter demonstrating increased PSD in all cases ($P < .001$).

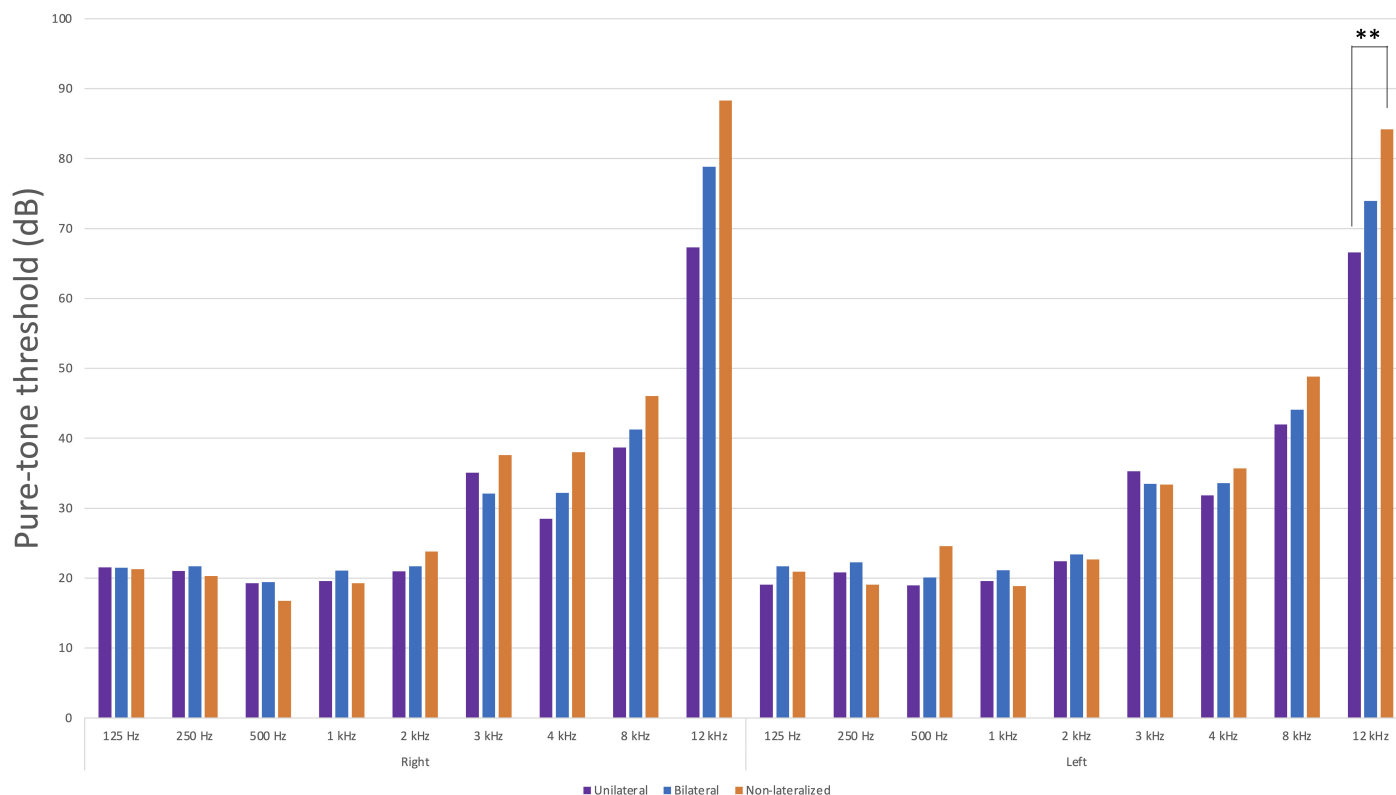


Figure 2. Distribution of pure-tone threshold in the unilateral, bilateral, and non-lateralized Tinnitus groups (** $P < .01$).

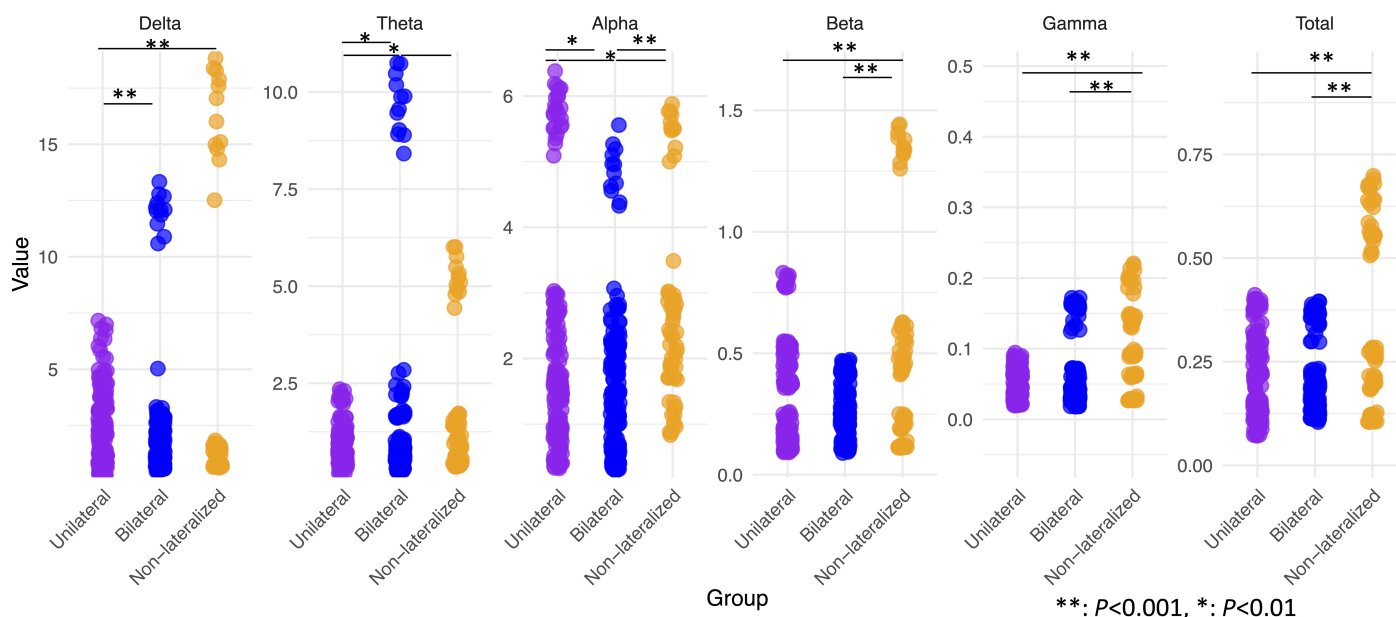


Figure 3. Power spectral density analysis of the different frequency bands (** $P < .01$).

DISCUSSION

In this study, approximately 10% of tinnitus cases were not lateralized. Non-lateralized tinnitus was mainly observed in the elderly and in patients with a short duration of tinnitus. Of the audiological profiles, the hearing threshold at 12 kHz differed alone non-lateralized tinnitus from bilateral tinnitus. Hearing asymmetry was the least frequent in patients without lateralized tinnitus. The accompanying symptoms and the questionnaires significantly did not differ according to tinnitus lateralization and the increased brain activities were

observed in non-lateralized tinnitus compared to unilateral or bilateral tinnitus. These results imply that non-lateralized tinnitus may accompany symmetric and extended high-frequency hearing loss in elderly patients before habituation is achieved at an early stage of tinnitus.

It has been established that auditory deafferentation may lead to tinnitus perception, and the relationship between unilateral tinnitus and ipsilateral asymmetric hearing loss is well known.¹³ Consistent

with a previous study, hearing asymmetry was most prominent in unilateral tinnitus.¹⁴ In addition, hearing asymmetry was least common in non-lateralized tinnitus. In a previous study of chronic tinnitus, 17% of patients with normal hearing complained of non-lateralized tinnitus, which was twice as frequent in the normal hearing group than in the unilateral or bilateral hearing loss groups.¹⁴ This implies that patients with non-lateralized tinnitus are likely to have normal hearing in conventional audiograms and that extended high-frequency hearing loss may exist.

A previous functional magnetic resonance imaging study observed low activation of the inferior colliculus (IC) contralateral to the tinnitus perception area in lateralized tinnitus.¹⁵ In contrast, IC activation was not observed in patients with non-lateralized tinnitus. The study suggested that this phenomenon might be caused by weak IC activation in non-lateralized tinnitus, similar to normal controls, compared to the more robust activation in lateralization. Interestingly, this finding was similar to ours, in that the absolute PSD was weaker in non-lateralized tinnitus. Furthermore, their study assumed that other brain areas might be involved in the perception of non-lateralized tinnitus instead of IC stimulation. Various brain networks, including the default mode network (DMN), salient network, and distress network, are involved in the development and maintenance of tinnitus. Tinnitus can be perceived without distress when the circuit breaker signal is linked to DMN, and the DMN regards tinnitus as a norm.¹⁶ In this study, the questionnaire scores did not significantly differ between lateralized and non-lateralized tinnitus. In all, non-lateralized tinnitus seems to cause minor changes in brain activity compared to lateralized tinnitus.

We also found that tinnitus lateralization did not differ according to accompanying symptoms. According to previous studies, headaches can trigger tinnitus; in particular, unilateral headaches can induce tinnitus by sensitizing the trigeminal system.¹⁷ It has also been reported that the directions of headaches and tinnitus are similar. Since we only documented the presence or absence of headache based on a retrospective medical chart review, it was impossible to confirm the difference in headache type and localization according to tinnitus laterality. In addition to headache, the distribution of accompanying temporomandibular disorders and neck pain did not differ according to tinnitus lateralization, suggesting that the relevance to the somatosensory system may not be high. Accompanying aural fullness and dizziness, as well as electrocochleography findings, did not show any significant differences according to the lateralization of tinnitus. In addition, better hearing thresholds at low frequencies in non-lateralized tinnitus imply that non-lateralized tinnitus may not be associated with endolymphatic hydrops.¹⁸ Since there was no significant difference in accompanying symptoms other than the significant difference in some audiological tests and qEEG according to lateralization, thorough history taking should be performed, and additional workup should be considered to evaluate patients with non-lateralized tinnitus.

The human auditory system inherently displays hemispheric asymmetry.¹⁹ Generally, the pure-tone thresholds at 3-6 kHz in the left ear are inferior to those on the right side, although it remains unclear whether these observations are caused by noise exposure or aging.²⁰ Likewise, the prevalence of tinnitus tends to be higher on the left side.²¹ There are suggestions that this might be caused by

the difference in the distribution of the medial olivocochlear efferent system, which modulates outer hair cell contraction and may have a protective role against an auditory stimulus that is predominant on the right side.²¹ In a study that investigated changes in the central auditory pathway using positive emission tomography, when 20 patients with bilateral tinnitus were compared with a normal control group, the fluorodeoxyglucose uptake of the left primary auditory cortex was higher than that of the right, regardless of the presence of tinnitus. An increase in fluorodeoxyglucose uptake in the secondary auditory cortex was observed on the right side in patients with tinnitus.¹⁹ In a study on patients with normal or mild hearing loss, when complaining of tinnitus on the left side, compared with healthy controls and right-sided tinnitus, decreased white matter volume of the left superior frontal gyrus and abnormal alterations in white matter integrity suggested that tinnitus lateralization influences the remodeling of white matter.^{14,21} In this study, as already stated, approximately 10% of tinnitus cases were not lateralized, and these patients also had different characteristics from those with bilateral tinnitus. Considering that auditory symptoms are inherently prone to lateralization, additional neurological and anatomical factors are essential to reveal which factors are mainly involved in recognizing non-lateralized tinnitus.

This study has the following limitations. First, the absence of long-term follow-up results may have weakened the strength of this study. A longitudinal study based on this study could address this limitation. Second, due to the retrospective nature of this study, the analysis of the accompanying symptoms was limited. Third, functional connectivity analysis could have been applied because there is a possibility that DMN or the posterior cingulate cortex may process non-lateralized tinnitus as a norm. However, we could not perform a more complex analysis because of a lack of technical support.

In conclusion, non-lateralized tinnitus can be observed in elderly patients with symmetric and extended high-frequency hearing loss before habituation is achieved at an early stage of tinnitus. However, there was no difference in the questionnaire scores and accompanying symptoms; therefore, it may not be worth managing non-lateralized tinnitus separately from tinnitus in the ear.

Ethics Committee Approval: This study was approved by the Ethics Committee of Ewha Mokdong Hospital (Approval No: 2022-04-021-002).

Informed Consent: N/A.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – H.Y.L.; Design – Y.P., Z.Y.L.; Supervision – S.H.S., S.W.B.; Resources – H.Y.L., Y.P.; Materials – Z.Y.L., H.Y.L.; Data Collection and/or Processing – Y.P., Z.Y.L.; Analysis and/or Interpretation – H.Y.L., Y.P.; Literature Search – H.Y.L., S.H.S., S.W.B.; Writing – S.H.S., Y.P.; Critical Review – S.H.S., S.W.B.

Declaration of Interests: The authors have no conflict of interest to declare.

Funding: This research was supported by a grant from the Korea Health Technology R&D Project through the Korea Health Industry Development Institute (KHIDI), funded by the Ministry of Health & Welfare, Republic of Korea (Grant Number: HI21C1574040021).

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