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ORIGINAL RESEARCH

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Extended high frequency hearing loss in tinnitus-positive chronic suppurative otitis media patient

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Abstract

Objectives: To determine the association between extended high frequency hearing loss and tinnitus in normal cochlear function based on a conventional audiometry chronic suppurative otitis media (CSOM) patient.

Design and methods: A cross-sectional study was conducted on 220 ears diagnosed as having CSOM with an equal number of tinnitus and without tinnitus groups. Only those with normal cochlear function based on conventional pure tone audiometry (250 Hz to 8 kHz) were included. They were further tested for hearing at extended high frequencies of up to 16 kHz. The severity of tinnitus was tested using a tinnitus questionnaire.

Results: The prevalence of extended high frequency hearing loss in the normal cochlear function CSOM patients with tinnitus was 81.8% (95% CI 74.5%, 89.1%), whereas the prevalence in the tinnitus negative group was 30.0% (95% CI 21.3%, 38.7%). There was a significant association between extended high frequency hearing loss and tinnitus in CSOM patients ($P < .001$). The average thresholds were significantly higher in the tinnitus group at all extended high frequencies tested with an increasing trend of significance toward the higher frequencies. However, this study did not observe any association between the severity of tinnitus and extended high frequency hearing loss.

Conclusion: The development of tinnitus among normal cochlear function CSOM patients indicates that the damage has occurred at a higher frequency level. However, the severity of tinnitus does not predict the degree of higher frequency hearing loss. Therefore, the presence of tinnitus warrants more aggressive monitoring and treatment to prevent sensorineural hearing loss from developing into the speech frequencies.

Level of Evidence: 4.

KEYWORDS
hearing loss, high frequency, otitis media, suppurative, tinnitus

1 | INTRODUCTION

A patient is diagnosed as having chronic suppurative otitis media (CSOM) when he or she has an infection of the middle ear with tympanic membrane perforation for more than 12 weeks. Generally, CSOM results in conductive hearing loss from the perforation of the tympanic membrane and damage to the middle ear structures. However, cochlear dysfunction which manifests as sensorineural hearing loss (SNHL) may occur due to the passage of inflammatory agents well as topical ototoxic agents used in its treatment through the round window.[1] It has been reported that the damage to the cochlea may not be related to the duration of the disease.[2,3]

Tinnitus is defined as a perception of sound without an adequate acoustic stimulus.[4] It can cause devastating effects on the psychological wellbeing of the patient if left untreated. Many theories postulate the etiology of tinnitus, but most authors agree regarding cochlear dysfunction as one of the culprits.[5]

Bridging these two ideas, it is believed that cochlear dysfunction, as a sequalae of CSOM, which is manifested as sensorineural hearing loss, is what causes the development of tinnitus in CSOM patients. The early changes of SNHL, which include the deterioration of hearing at extended high frequencies, can be detected using extended pure tone audiometry. This early detection would mean that a more aggressive approach toward management needs to be undertaken by the attending physician so that hearing loss does not progress and so that CSOM patients do not suffer from tinnitus later in life.

Studies by Sandeep et al have shown that tinnitus is one of the otological symptoms in CSOM apart from earache and hearing loss.[6] They noted that approximately one in five CSOM patients will experience tinnitus with varying degrees of severity. Manche et al reported that almost 60.9% of CSOM cases are associated with tinnitus.[7] In this large epidemiological study involving a total of 3255 subjects, middle ear and inner ear diseases had a significant association with the development of tinnitus. Furthermore, almost all tinnitus subjects (96.9%) had hearing loss.

The outer hair cells are most vulnerable to toxic effects and ototoxic medications that are able to penetrate the round window, especially for those at the basal turn of the cochlear that is responsible for high pitch hearing.[8] The imbalance in intracochlear calcium that influences the cilia hair cells, as well as spontaneous otoacoustic emission and stereocilia decoupling of the outer hair cells are among the postulated theories of tinnitus generation according to Jastreboff.[9] All in all, the resulting cochlear dysfunction is what most of the authors accept as the cause the tinnitus.

This study aimed to determine the association between extended high frequency hearing loss and tinnitus in CSOM patients with normal cochlear function and to determine the association between extended high frequency hearing loss and the severity of tinnitus in CSOM.

2 | MATERIAL AND METHODS

A cross-sectional study was done at the otorhinolaryngology clinic in a tertiary hospital. This study was approved by our university's Human Research Ethics Committee with the code number USM/JEPeM/17100533. The study was performed according to the Helsinki Declaration, and written consent was obtained from each subject.

Based on the study by MacAndie and O'Reilly,[10] and using PS software, the calculated sample size was 220 ears diagnosed as CSOM but with normal cochlear function, with a ratio of one to one (110 ears from CSOM with tinnitus and 110 ears from CSOM without tinnitus as a control). They were selected by a systematic random sampling method and screened for inclusion and exclusion criteria. The inclusion criteria included patients aged between 13 and 50 years old with clinically diagnosed CSOM. Only ears with normal cochlear function according to conventional PTA (250 Hz to 8 kHz) were included in the study. Those patients who had other risks of developing sensorineural hearing loss, such as chronic exposure to noise, familial history of young-onset hearing loss, as well as patients with a known history of receiving systemic ototoxic drugs were excluded from the study.

Patients who consented were interviewed to complete a proforma which included the patient's demographic data, past medical history, past surgical history, and medication history. Then, an otoscopic ear examination was performed to confirm the diagnosis of CSOM. Following the diagnosis, the patient underwent a PTA that measures hearing thresholds from 125 Hz to 8 kHz. Only those with normal bone conduction were included and hence were referred to as having normal cochlear function.

The ears were grouped into tinnitus and without tinnitus groups based on self-reported tinnitus symptoms. Following their grouping, CSOM patients with tinnitus were given a Malay language validated Tinnitus Handicap Questionnaire (BEST) to assess the severity of their tinnitus.[5] The CSOM with no tinnitus group served as a control group. Extended high frequency pure tone audiometry was done using the MADSEN clinical audiometer (Madsen Astera 2, Denmark) with a Sennheiser HDA 300 closed circumoral earphone at the frequencies of 9, 10, 11.2, 12.5, 14, and 16 kHz. Extended high frequency hearing loss (EHFHL) was defined as the inability of the patient to hear the highest level of the stimulus given starting at the highest frequency tested downward (inability to hear at 16 kHz or 16 and 14 kHz or 16, 14, and 12.5 kHz or so on). For the purpose of measuring the average threshold at each frequency, those with the inability to hear the highest level of the stimulus were considered to have a hearing threshold of 10 dB HL higher than the respective stimulus. The highest-level stimuli were 100, 95, 90, 85, 75, and 50 dB for each frequency tested from 9 to 16 kHz, respectively.

The data was analyzed using IBM SPSS version 24.0 software. Categorical data was summarized with frequencies and percentages. The prevalence of extended high frequency hearing loss in CSOM patients with normal cochlear function was calculated at a 95% confidence interval. Fisher's exact test was used to determine the association of tinnitus and EHFHL in CSOM patients with normal cochlear function, as well as the association between the severity of tinnitus and EHFHL in the patient. The level of significance was set at the .05 level.

TABLE 1 Demographic profile of study population (n = 220)

	Tinnitusn = 110	No tinnitusn = 110	Totaln = 220
	n(%)	n (%)	n (%)
Gender			
Male	25 (22.7)	34 (30.9)	59 (26.8)
Female	85 (77.3)	76 (69.1)	161 (73.2)
Age	33.2 (12.62)[a]	27.3 (11.53)[a]	<.001[b]
Race			
Malay	108 (98.2)	108 (98.2)	216 (98.2)
Chinese	2 (1.8)	0 (0.0)	2 (0.9)
Indian	0 (0.0)	2 (1.8)	2 (0.9)
Site of CSOM			
Right	55 (50.0)	57 (51.8)	112 (50.9)
Left	55 (50.0)	53 (48.2)	108 (49.1)
Degree of hearing loss (dB HL)[c]			
Normal	0 (0.0)	4 (3.6)	4 (1.8)
Mild (20-40)	40 (36.4)	70 (63.6)	110 (50.0)
Moderate (41-70)	70 (63.6)	36 (32.7)	106 (48.2)

[a]Mean (SD).
[b]Significant difference of mean age between tinnitus and non-tinnitus group using independent *t*-test.
[c]Significant association between degree of hearing loss and tinnitus using Chi-square test; *P* = .001.

TABLE 2 Association of tinnitus and extended high frequency hearing loss (n = 220)

			Extended PTA		
Variables		n	No EHFHLn (%)	EHFHLn (%)	*P*-value
Tinnitus	Tinnitus	110	20 (18.2)	90 (81.8)	<.001[a]
	No tinnitus	110	77(70.0)	33 (30.0)	

[a]Fisher's exact test.

TABLE 3 Comparison of mean threshold of different frequencies between tinnitus and non-tinnitus group using independent *t*-test (n = 220)

	Tinnitus(n = 110)	Non-tinnitus(n = 110)			
Variables	Mean (SD)	Mean (SD)	Mean difference (95% CI)	*t* statistic (df)	*P* value
9 kHz	48.64 (18.51)	45.59 (17.98)	3.045	218	.548
10 kHz	49.55 (19.78)	42.50 (17.14)	7.045	213.701	.043
11.2 kHz	53.41 (22.03)	45.73 (19.81)	7.682	215.596	.016
12.5 kHz	57.68 (22.19)	47.77 (18.98)	9.909	212.889	.010
14 kHz	60.23 (23.03)	49.27 (17.32)	10.955	202.401	<.001
16 kHz	55.05 (11.74)	42.59 (15.46)	12.455	203.373	<.001

3 | RESULTS

Table 1 shows the demographic data of the study population. There were 220 ears included in the analysis with an almost equal number for the right and left sides. As is shown in Tables 2, 90 out of 110 CSOM ears with tinnitus had EHFHL, a prevalence of 81.8% (95% CI 74.5%, 89.1%). On the other hand, 33 out of 110 CSOM ears without tinnitus had EHFHL, a prevalence of 30% (95% CI 21.3%, 38.7%). There was a significant association between EHFHL and tinnitus in CSOM ears (*P* < .001).

Table 3 compares the average threshold between the tinnitus and non-tinnitus groups. The present study observed a significant occurrence of EHFHL in the tinnitus group. The *P*-value showed an increasing trend of significance with an increase in frequencies. Table 4 presented the association of the severity of tinnitus and EHFHL in 110 ears with CSOM and tinnitus. Ninety-five subjects were in the

TABLE 4 Association of severity of tinnitus and extended high frequency hearing loss (n = 110)

Variables		n	Extended PTA		*P* value
			No EHFHLn (%)	EHFHLn (%)	
Severity of tinnitus	Mild-moderate (0-50)	95	17 (17.9)	78 (82.1)	0.542[a]
	Mod-severe (51-100)	15	3 (20.0)	12 (80.0)	

[a]Fisher's exact test.

mild to moderate BEST score grouping, accounting for a prevalence of 86.4%. The rest of the 15 subjects (13.6%) experienced moderate to severe tinnitus based on the BEST score grouping. However, the present study did not observe any significant association between the severity of tinnitus and EHFHL ($P > .05$).

4 | DISCUSSION

In the advance of technology, the development of a mean to test extended high frequency hearing loss has opened a new window in the assessment in otology. The use of extended high frequency testing serves not just for the early detection of inner ear malfunction, but also as a preventative measure to take before irreversible damage is done.[11] Multiple recent studies have continued to support the earlier findings that extended high frequency hearing is the first to show changes in various inner ear pathologies.[11-13] Furthermore, especially in ototoxicity cases where monitoring with extended frequency PTA is much more established, the evidence of ototoxicity can be gathered much earlier than conventional PTA and can show any.[14,15]

CSOM causes conductive hearing loss (CHL) due to tympanic membrane perforation and ossicular chain changes.[16] In a proportion of cases, sensorineural element hearing loss from cochlear damage has also been found in conventional pure tone audiometry.[2,17] Smitha and Apurva found a 28% prevalence in sensorineural components from conventional PTA in CSOM patients.[16]

The perception of tinnitus is associated with cochlear damage, especially in the basal turn, as concluded by Fabijanska et al.[18] This is also supported by the discordant damage theory advanced by Jastreboff which posits that the unbalanced neural activity between damaged outer hair cells and intact inner hair cells is a possible cause of tinnitus.[18,19] However, abnormal neural activity from any level of the auditory pathway may cause subjective tinnitus.[20]

In the case of CSOM, the inflammatory mediators, pore-forming toxins[21] and ototoxicity elements from eardrops may diffuse through the round window[17] causing direct damage to the basal turn of the cochlear, which is responsible for high frequency hearing.[17] Cochlear tissues have also been found to be capable of initiating their own immunological response to inflammatory stimuli from the middle ear, forming various inner ear cytokines.[22] Cureoglu et al,[23] in their retrospective human temporal bone analysis, noted prominent loss of cochlear basal turn outer and inner hair cells, with a significant decrease of the basal turn area of the spiral ligament and stria vascularis in the temporal bones of CSOM compared with controls. Joglekar et al,[24] while confirming these findings, also found serofibrinous precipitates and inflammatory cells in the scala tympani of the basal turn and cochlear aqueduct.

Our study observed the statistically significant association of tinnitus with EHFHL, which is most significant at higher frequencies followed by lower ones. This finding can be correlated to the existing fact that the basal turn of cochlear that is responsible for high frequency hearing is closest to the middle ear, which can be directly affected in middle ear diseases like CSOM.

The present study did not show any significant correlation between the severity of tinnitus and the degree of EHFHL. In other words, the severity of tinnitus could not predict how much damage had occurred to extended high frequency hearing. Therefore, the mere presence of tinnitus warrants a more thorough treatment in terms of early surgical intervention, regular consultations and audiology tests, as well as more careful usage of topical antibiotics and more aggressive active infection controls.

It is difficult to confirm whether EHFHL is conductive or sensorineural in a case of conductive hearing loss. However, hearing loss at the extended high frequency in otitis media is attributed to sensorineural rather than conductive.[25] In addition, our study showed an increasing trend of significance difference of mean thresholds with an increase in frequencies which is in line with sensorineural hearing loss. The upper limit of age taken in our study was 50 years old in order to exclude those with presbycusis.

The development of tinnitus among normal cochlear function CSOM patients indicates that damage has occurred at a higher frequency level. However, the severity of tinnitus does not predict the degree of higher frequency sensorineural hearing loss. Therefore, the presence of tinnitus warrants more aggressive monitoring and treatment to prevent sensorineural hearing loss from developing into the speech frequencies.

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CONFLICT OF INTEREST

The authors declare no conflicts of interest.

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