ORIGINAL ARTICLE

Analysis of clinical audiological characteristics of noise-induced silent hearing loss

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Received: August 20, 2023 **Accepted:** November 3, 2023 **Online Published:** November 6, 2023
DOI: 10.5430/dcc.v10n1p22 **URL:** https://doi.org/10.5430/dcc.v10n1p22

ABSTRACT

Objective: To explore the characteristics of audiology examination of Noise-Induced Hidden Hearing Loss (NIHHL), and then explore its valuable diagnostic methods.

Methods: A total of 80 young men aged between 19 and 35 were selected. They were classified into the experimental group with noise exposure history and the control group without noise exposure history, with 40 men in each group. We carried out extended high-frequency audiometry test for good ears; conducted speech in noise measurement and distortion product emission for good ears; DPOAE test, compare and analyze the results of each test.

Results: The extended high frequency hearing threshold test results showed that the hearing threshold of the experimental group was larger than that of the control group at the frequency above 8 kHz, with significant statistical difference ($p < .01$). The results of speech audiometry under noise showed that the signal-to-noise ratio loss in the experimental group was greater than that in the control group, with a significant statistical difference ($p < .01$). The results of otoacoustic emission showed that the signal-to-noise ratio of the experimental group was lower than that of the control group at the frequency of 6 kHz and above, with a statistically significant difference ($p < .01$). There is a correlation between pure tone audiometry and DPOAE test results at high frequencies (6 kHz: $p < .0001$, r = -.478; 8 kHz: $p < .0001$, r = -.491); There was a correlation between speech audiometry and DPOAE test results under noise ($p = .031$, r = -.299).

Conclusions: Compared with the control group, the extended high frequency pure tone hearing threshold increased or could not be elicited in the experimental group, speech recognition ability decreased significantly under noise, DPOAE was normally elicited but signal-to-noise ratio decreased, and there was a certain correlation between them. The above three audiological examination methods have a certain reference value for early recognition of hidden hearing loss.

Key Words: Noise-induced recessive hearing loss, Extended high frequency threshold test, Speech audiometry under noise, Otoacoustic emission

1. INTRODUCTION

Hearing health problems caused by noise have been paid more and more attention. According to the report of the World Health Organization, 1.1 billion young people around the world are at risk of Hearing Loss caused by excessive noise injury. Therefore, Hidden Hearing Loss (HHL) has been widely concerned. Studies have shown that noise exposure, aging and the use of ototoxic drugs are the inducing factors of HHL. The concept of HHL and its clinical features were first systematically reported by Liberman in 2015.[1] HHL is a kind of above threshold hearing disorder with normal hearing threshold detection results and decreased speech

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recognition rate only in noisy environment. Its clinical features are reduced language comprehension and spatial localization in noisy environments. In recent years, in view of the pathogenesis, clinical manifestations and characteristics of audiology examination of Noise-Induced Hidden Hearing Loss (NIHHL), many scholars have carried out many experimental studies, but at present, there is still no one audiology examination can completely diagnose NIHHL. A comprehensive assessment should be made by combining multiple examinations.

The research methods to be selected in this study include the following three: extended high-frequency audiometry; speech in noise; and distortion product emission (DPOAE). To explore the audiological characteristics of NIHHL and the correlation between various examination methods, in order to further understand the clinical characteristics of NIHHL, and hope to provide theoretical reference for the subsequent research on NiHHL-related topics.

2. MATERIALS AND METHODS

2.1 Test object

The test subjects included 80 male with and without a history of noise exposure, aged 19-35 years. Inclusion criteria: (1) No history of hearing loss caused by genetic ear disease or drug-related deafness, no history of head trauma or other ear diseases; (2) no history of shooting activities in daily life; (3) No history of exposure to chemical reagents or heavy metals; and (4) Avoid exposure to any high-intensity sound environment for at least 12 hours before starting the test. The experimental subjects were divided into experimental group and control group according to whether there was a long history of noise exposure. The experimental group consisted of 40 subjects with 40 ears and an average age was 27.69 $\pm$ 6.34 years, the average exposure time was 3.7 $\pm$ 3.3 years, the average exposure intensity was 85 $\pm$ 6.7 dB(A), and the exposure frequency was 5-7 days/week, 6-8 hours a day. The control group consisted of 40 subjects with 40 ears and an average age of 23.31 $\pm$ 4.32 years.

2.2 Methods

2.2.1 *Ear examination*

Check the auricle, rule out inflammation, trauma and deformity, and check the external auditory canal and eardrum by ear endoscopy. Take out cerumen if there is plug in the external auditory canal. After the auricle, external auditory canal and tympanic membrane were normal, pure tone audiometry and acoustic impedance test were performed, and the average threshold of pure tone audiometry (0.25, 0.5, 1, 2, 3, 4, 6, 8 kHz) was less than 20 dB HL. The tympanic curve of acoustic impedance was type A, and acoustic reflection existed.

2.2.2 *Extend high frequency pure tone hearing threshold test*

CONERA audiometer made in Denmark is used in the acoustic insulation shielding room with background noise < 25 dB(A). The operation method conforms to the national standard GB/T16403-1996 pure tone audiometry. Extended high frequency test 9 k-20 kHz 8 frequencies (9 k, 10 k, 11.2 k, 12.5 k, 14 k, 16 k, 18 k, 20 kHz). To reach the maximum output of the instrument still no reaction only calculates the number of no reaction, and the rate of eliciting the reaction ear with the threshold detection rate.

2.2.3 *Speech audiometry under noise*

The CONERA audiometer made in Denmark was used to play the Chinese version of the BKB-SIN speech test material under noise at the intensity of 70 dB HL through the computer in a sound-proof room meeting the national standards. The six sentences in the audiometry list were played randomly with the headset as the transmission mode, and the subjects were asked to retell what they heard, and the number of correct keywords were recorded. The signal-to-noise ratio loss is calculated according to the formula to reflect the subjects' speech recognition ability under noise. The greater the signal-to-noise ratio loss, the worse the speech recognition ability under noise.[2]

2.2.4 *Distortion product otoacoustic emission*

Danish Erlisten me Capella otoacoustic emission tester was adopted, and the test project was DPOAE. Parameter Settings: F2:F1 = 1.22, L1 = 65 dB SPL, L2 = 55 dB SPL, and the test frequency (F2) was 0.5, 1, 1.5, 2, 3, 4, 5, 6, 7, 8 kHz. The test was carried out in an acoustic insulation room with background noise < 25 dB(A), and the DP amplitude and signal to noise ratio at 2F1-F2 were recorded. The passing condition of the test is that the signal-to-noise ratio is greater than 6dB within 1 min, and the DP amplitude is greater than -10 dB. The pass rate and signal-to-noise ratio are analyzed.

2.2.5 *Statistical methods*

SPSS22.0 was used for data statistics, and the difference between the two groups was statistically analyzed by independent sample t test. Correlations among the tests were analyzed using Pearson correlation coefficient. $p < .05$ indicated statistical significance.

3. RESULTS

3.1 Pure tone audiometry

Compared with the experimental group and the control group, the extended high frequency audiometry in the experimental group was higher than that in the control group, with statisti-

cal difference ($p < .05$). The detection rate decreased with the increase of stimulus frequency. See Table 1.

3.2 Speech audiometry under noise

The results showed that the signal-to-noise ratio loss of the experimental group (5.12 ± 3.18 dB) was greater than that of the control group (2.79 ± 2.12 dB), and the difference was statistically significant ($p < .05$).

3.3 DPOAE

All frequency DPOAE in the experimental group and the control group were extracted. At frequencies below 6 kHz, there was no statistically significant difference in the signal-to-noise ratio between the two groups. At frequencies above 6 kHz, the signal-to-noise ratio of the experimental group was lower than that of the control group, and there was a statistically significant difference ($p < .05$), as shown in Table 2.

3.4 Correlation results

According to Pearson correlation coefficient calculation, pure tone audiometry and DPOAE test results are correlated at the frequencies of 6 kHz and 8 kHz ($p < .001$), and the correlation coefficients are shown in Table 3. The results of speech audiometry under noise were correlated with the mean signal-to-noise ratio of each frequency in DPOAE test ($p = .031$, $r = -.299$).

Table 1. The average threshold of 9-20 kHz (dB HL, x±s) and its detection rate (%) in the two groups

Group	9 kHz	10 kHz	11.2 kHz	12.5 kHz	14 kHz	16 kHz	18 kHz	20 kHz
Control	0.79±8.38 (100)	1.95±5.09 (100)	1.22±7.01 (100)	-2.13±5.79 (100)	2.52±8.89 (100)	13.11±15.33 (100)	-4.22±12.78 (95.46)	-14.37±6.13 (64.85)
Test	8.39±11.1** (100)	18.91±13.87** (100)	24.22±17.31** (100)	27.09±19.85** (100)	36.8±19.38** (100)	47.14±9.18** (65.34)	20.05±7.88** (22.56)	0.02±9.22** (14.74)
p	.001	.000	.000	.000	.000	.000	.000	.000

Note. **Compared with the control group, there was a very significant difference ($p < .01$).

Table 2. Average DP signal-to-noise ratio (dB, x±s) between 0.5-8kHz in the two groups

Group	0.5 kHz	1 kHz	1.5 kHz	2 kHz	3 kHz	4 kHz	5 kHz	6 kHz	7 kHz	8 kHz
Control	10.18±3.25	18.76±5.48	20.46±6.47	21.74±7.26	20.54±8.11	22.59±7.08	27.56±6.28	29.95±4.57	29.87±5.76	24.46±5.12
Test	9.87±4.49	17.72±3.49	21.64±6.01	17.87±9.86	18.41±4.32	21.67±4.89	25.78±5.23	23.18±6.27**	24.92±5.48**	19.78±6.43**
p	.758	.410	.641	.118	.275	.315	.160	.002	.005	.004

Note. **Compared with the control group, there was a very significant difference ($p < .01$).

Table 3. Correlation of pure tone audiometry and DPOAE test results at each frequency

Frequency (kHz)	0.5	1	2	3	4	6	8
Correlation coefficient	-0.297*	-0.024	-0.093	-0.218	-0.211	-0.478**	-0.491**
p	.033	.868	.516	.120	.133	.000	.000

Note. * Compared with the control group, there was a significant difference; Compared with the control group, there was a very significant difference ($p < .01$)

4. DISCUSSION

From physiological theory, the change of auditory sensitivity after noise stimulation is determined by the functional state of sensory hair cells in cochlea, and outer hair cells (OHCs) are more sensitive to sound stimulation than inner hair cells (IHCs). Therefore, it is concluded that the noise-induced auditory system function impairment is mainly related to the injury or loss of OHCs. It has been found in recent years that synaptic damage between IHCs and primary spiral ganglion neurons (SGNs) occurs earlier than that of OHCs after noise exposure, leading directly to the degenerative death of SGNs.[1,3,4] The mechanism is as follows: The ribbon synapse (RS) attached to the cochlear presynaptic membrane is located between the cochlear IHCs and SGNs. It is surrounded by many synaptic vesicles. There are many Ca^{2+} channels in the presynaptic membrane. When stimulated by noise, Ca^{2+} channels open in large numbers, RS releases excess excitatory amino acid glutamate via synaptic vesicles which acts on glutame-specific receptors in the postsynaptic membrane to promote Ca^{2+} entry into the postsynaptic membrane sensory hair cells, causing Ca^{2+} overload in SGNs, resulting in cell edema, oxidative stress reaction, and eventually SGNs death.[5] Moreover, many studies have shown that even if partial repair of synapses is possible, functional defects still exist, which affect the information processing ability of human auditory system.[4,6,7] Current studies have shown that damage and repair of RS may be the basis for the formation of NIHHL.[8]

The conventional pure tone hearing threshold test ranges from 125 Hz to 8,000 Hz, which is limited to reflect the damage caused by the early noise stimulation of cochlea. The conventional pure tone hearing threshold test results of the experimental group and the control group in this study are both within the normal range. The extended high-frequency audiometry can measure the hearing threshold of human ear of 10 kHz-20 kHz, which can reflect the early lesions of cochlea caused by noise. On the one hand, Von Bekesy's traveling wave theory has clarified that high-frequency acoustic waves are mainly transmitted in the cochlear floor gyr, and previous studies have confirmed that OHCs damage occurs earliest and heaviest in the cochlear floor gyr after noise stimulation. When hair cells are damaged, OHCs injury in the basal gyrus of cochlea is the most significant first, and the corresponding high-frequency hearing loss is the most obvious. However, this part of hearing loss cannot be detected by constant frequency pure tone audiometry. Some studies[9] have shown that 10 k-20 kHz extended high-frequency hearing detection can reflect the potential hearing impairment of normal hearing ears at frequencies below 8 kHz and can expand the localization detection of the function of cochlear basal gyrus. On the other hand, the nerve fibers responsible for transmitting high-frequency sound waves are in the periphery (surface) of the auditory nerve, while the fibers related to transmitting low-frequency sound waves are in the center of the auditory nerve, and the periphery is damaged before the center.[10] Therefore, the high-frequency region of cochlea is the most sensitive to noise stimulation, and the early acoustic damage of the inner ear first occurs in the frequency range of 10 kHz-20 kHz, and the abnormal occurrence of frequency above 10 kHz is earlier than other frequencies.[11] According to this conclusion, domestic scholars have studied the noise exposure history. In addition, extended high frequency hearing detection was performed on those with normal constant frequency pure tone hearing threshold detection, which proves that extended high frequency hearing detection has certain clinical significance for the early diagnosis of noise-induced hearing loss.[12–14] The extended high frequency test results of this study are consistent with those of previous studies.

Spiral ganglion type I cells are bipolar neurons whose peripheral processes form synapses with IHCs. Based on spontaneous rate (SR), spiral ganglion type I cells are divided into low SR neurons and high SR neurons.[15] High SR neurons accounted for about 3/4, and their threshold was low. They were responsible for simple signal processing and acoustic signal processing in quiet environment. The number of low SR neurons is small, accounting for only 1/4, and its threshold value is relatively high, which is crucial for processing acoustic signals in noisy environment.[16] Long-term noise exposure selectively damages low SR neurons.[7] Therefore, the damage of low SR neurons is not easy to be detected by threshold test, but it will lead to abnormal results of speech audiometry under noise. In this study, the results of speech audiometry under noise in the experimental group showed that the signal-to-noise ratio loss was significantly higher than that in the normal group.

In this study, DPOAE was used to assess cochlear function. Otoacoustic emission is a kind of audio energy originating from the cochlea and released into the external auditory canal through the reverse conduction of the ossicular chain and tympanic membrane. This energy is generated by the active mechanical activities of OHCs. Otoacoustic emission has stable frequency characteristics and test stability, which can keenly reflect the function of cochlear OHCs, so it has been widely used in clinic. Studies have shown that DPOAE can be abnormal even when there is no change in conventional pure tone audiometry, suggesting that DPOAE can detect early mild lesions of cochlear OHCs. Other studies have shown that although DPOAE can be elicited in patients with recessive hearing loss, the amplitude and signal-to-noise ratio of DP are abnormal. Therefore, for early injury of inner ear, the sensitivity of DPOAE is better than that of conventional pure tone audiometry, and the application of DPOAE can screen out susceptible population sensitive to noise.[17,18] In this study, the detection rate of DPOAE in both the experimental group and the control group was 100%, and the signal-to-noise ratio of 6 kHz and above frequency in the experimental group was significantly lower than that in the control group, indicating that DPOAE has a certain value in the diagnosis of hidden hearing loss.

The study shows that the results of extended high frequency audiometry and speech audiometry under noise are correlated with the test results of DPOAE. The reason is that extended high frequency audiometry, speech audiometry under noise and DPOAE are sensitive to the detection of early cochlear injury, namely, extended high frequency audiometry can reflect the nerve conduction function in the high frequency region of the bottom gyrus of cochlea, speech audiometry under noise can show the higher auditory center function, and DPOAE can reflect the function of the outer hair cells of cochlea. Therefore, the combined application of the three in clinic is of certain value for early detection of NIHHL.

ACKNOWLEDGEMENTS

We greatly appreciate the valuable contributions of every team member who took the time to participate in this study. Thank you to the leaders and teachers of the research department for their help.

AUTHORS CONTRIBUTIONS

Prof. Chunmin Li was responsible for study design and revising. Xiaoxu Du and Dandan Zhang were responsible for data collection. Prof. Tuya Bai drafted the manuscript and Xiuli Xu revised it. All authors read and approved the final manuscript.

FUNDING

This work was supported by the Science and Technology Bureau of Baotou City (project number jkws202057).

CONFLICTS OF INTEREST DISCLOSURE

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

INFORMED CONSENT

Obtained.

ETHICS APPROVAL

The Publication Ethics Committee of the Sciedu Press. The journal's policies adhere to the Core Practices established by the Committee on Publication Ethics (COPE).

PROVENANCE AND PEER REVIEW

Not commissioned; externally double-blind peer reviewed.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

DATA SHARING STATEMENT

No additional data are available.

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