Investigating the Possible Audiological Effects of Hypothyroidism

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Abstract

Objective: We investigated audiological function in patients diagnosed with primary hypothyroidism secondary to Hashimoto’s thyroiditis.

Methods: Forty-one patients diagnosed with primary hypothyroidism secondary to Hashimoto’s thyroiditis and 20 control subjects participated in this study. Otoscopic examination, immittance testing, pure tone audiometry, and otoacoustic emission testing were completed on all participants. In addition, all participants were screened for the presence of tinnitus.

Results: All participants’ demonstrated normal otoscopic examination and 0.226-kHz tympanometry test findings. Pure tone audiometric (PTA) testing revealed mild sensorineural hearing loss (SNHL) in 16 patients diagnosed with primary hypothyroidism (39%) compared to only one participant in the control group. Eight patients with primary hypothyroidism reported bothersome tinnitus (19.5%), and transient evoked otoacoustic emissions (TEOAE) were absent in 8 left ears (19.5%) and 4 right ears (9.75%).

Conclusion: Hypothyroidism secondary to Hashimoto’s thyroiditis may be a cause of mild SNHL, particularly at high frequencies, in addition to bothersome tinnitus. Absent otoacoustic emissions suggest cochlear outer hair cells as the possible lesion site in patients with hypothyroidism.

Keywords: Hypothyroidism; Hashimoto’s thyroiditis; Sensorineural hearing loss; Tinnitus; Otoacoustic emission; Outer hair cells

Introduction

Thyroid hormones are critical regulators of metabolic rate and this effect contributes to many developmental processes, such as central nervous system maturation and body growth [1]. Thyroid disorders secondary to iodine deficiency are not uncommon because around one-third of the world’s population lives in areas of iodine deficiency [2]. Moreover, autoimmune thyroid diseases are among the most frequent endocrine diseases. The estimated total prevalence of hypothyroidism is about 1% in the general population, and even higher in females [3]. Thyroid hormones are essential for the physiological development of the cochlea [1]; therefore, thyroid gland dysfunction affects the integrity of the auditory system [4,5].

An association between hypothyroidism and hearing loss has been suggested by multiple studies [1,6,7-9]. Arduc and colleagues evaluated 30 patients with Hashimoto’s thyroiditis and reported mild sensorineural hearing loss (SNHL) at frequencies of 250, 500, and 6000 Hz [8]. Santos et al. [9] tested 30 female patients with hypothyroidism and found SNHL in 22 ears and absent transient evoked otoacoustic emissions (TEOAEs) in 12 ears, along with increased latencies of waves I, III, and V in the auditory brain stem response (ABR) test. Anand et al. [6] found hearing loss in 16 of 20 patients (80%) and tinnitus in 3 patients (15%) with hypothyroidism. Kinmer and colleagues [10] reported an association between thyroid hormone deficiency and eventual hearing loss. Khechinaschvili et al. [11] tested 50 patients diagnosed with hypothyroidism and found SNHL in 74% and absent TEOAE in 52%, indicating that hypothyroidism leads to peripheral and central hearing disturbances.

Kumar et al. [1] found a significant difference in hearing thresholds between controls and patients with hypothyroidism. Indeed, 23 of 30 participants diagnosed with hypothyroidism had elevated hearing thresholds and 15 had absent TEOAE, suggesting a specific effect of hypothyroidism on the cochlea. Malik et al. [7] found hearing loss in 25 of 45 hypothyroid patients (55.56%) and tinnitus in 7 patients (15.56%).

Tinnitus accompanies most auditory disorders and is also relatively common in the general population [12,13]. Nevertheless, the most common cause of tinnitus is hearing loss [12]. This phenomenon may also be found in other non-ear related diseases such as hypertension, atherosclerosis, diabetes, and thyroid dysfunction [13]. The precise pathophysiology of tinnitus is still unclear [12].

Patients with autoimmune thyroid diseases have elevated tinnitus risk [7,14,15]. Kim and colleagues [16] suggested that tinnitus is more likely to occur in females with a history of thyroid disease. Nevertheless, there have been few studies on the pathophysiology of tinnitus in acquired hypothyroidism.

Studies examining the effect of hypothyroidism on inner ear morphology have reported that the first inner ear structure to show morphologic changes is the tectorial membrane [17]. A constant alteration in normal structure was found in addition to thickening of...
the basilar membrane. Other studies have reported enlargement of the intercellular spaces in striae vasculosa concomitant with degeneration of marginal and intermediate cells, as well as inner and outer hair cell degeneration, tectorial membrane irregularities, and debris in the cochlear duct [18]. Studies examining the effect of hypothyroidism on cochlear function have suggested that abnormal thyroid hormone levels lead to defects in neurological and morphological development of the organ of Corti [19], suggesting that thyroid hormones exert direct effect on the cochlea.

Despite remaining controversies surrounding the effects of hypothyroidism on the auditory system, numerous studies have reported hearing loss as a result of primary hypothyroidism. The aim of this study is to investigate the effect of primary hypothyroidism secondary to Hashimoto's thyroiditis on hearing threshold, outer hair cell function, and the presence of tinnitus. This study differs from previous research in that a full audiological test battery was conducted and the presence of tinnitus was investigated in this specific population.

Materials and Methods

Participants

Forty-one adults diagnosed with primary hypothyroidism secondary to Hashimoto's thyroiditis and a control group of 20 otologically normal adults participated in this study. The patient sample included 37 females and 4 males, while all participants in the control group were female. Patient age ranged between 16−50 years (mean ± SD, 34.2 ± 9.5 years). The age range of control group participants was also 16−50 years and distribution was well matched to the patient group (30.52 ± 9.6 years).

All participants were screened prior to the main test battery by otoscopic examination and measurement of middle ear pressure to ensure no recent history of middle ear disease. Subjects older than 50 years were excluded to eliminate the risk of participation. The study was approved by the Institutional Review Board (IRB) at Jordan University Hospital.

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All participants were screened prior to the main test battery by otoscopic examination and measurement of middle ear pressure to ensure no recent history of middle ear disease. The acoustic immittance of the middle ear was measured using an AA222 Interacoustic diagnostic tympanometer.

The patient group had confirmed autoimmune thyroid disease as evidence by positive serum thyroid peroxidase antibodies and/or positive thyroid ultrasonic findings for Hashimoto’s thyroiditis associated with primary or subclinical hypothyroidism (baseline thyroid stimulating hormone [TSH]>5.0 U/ml). The control group had normal TSH levels and was negative for thyroid peroxidase antibodies.

Subjects older than 50 years were excluded to eliminate the risk of age-related hearing loss (presbyacusis) as this tends to occur with increasing frequency after the fifth decade [20]. The other exclusion criteria were as follows: prior ear surgery, a history of noise exposure, history of hereditary hearing loss, a genetic syndrome, and central nervous system pathology.

All patients provided informed written consent prior to participation. The study was approved by the Institutional Review Board (IRB) at Jordan University Hospital.

Evaluations

Hearing thresholds were tested from 250-8000 Hz using an AC40 clinical audiometer through TDH supra-aural earphones. Bone conduction was also tested over 500-4000 Hz. British Society of Audiology (BSA) standard clinical procedures were implemented [21].

The signal presentation was 1-3 s to comply with BSA recommended procedures. The pause between presentations was random to prevent participants from anticipating the next signal.

Cochlear outer hair cell function was tested by pass/fail TEOAE using a MADSSEN Capella device. This test covered both higher and lower frequency emissions. A total of 8 valid peaks in alternating directions were required for normal outer hair cell function.

Patients were asked about the presence of tinnitus, and whether it affected their quality of life. Presence was defined as continuous tinnitus for more than one minute daily in either ear for more than 6 months.

Statistical analysis

SPSS software version 23 was used for all statistical analysis. The Shapiro Wilk test was used to test the normality of the distributions. The degree of hearing loss (%), the presence of tinnitus, and absence of otoacoustic emission results were determined separately for each ear and compared between ears and groups by independent sample T test or Pearson’s Chi-Square test as indicated.

Results

Hearing loss was not necessarily bilateral, so the results presented are ear-and frequency-specific. Hearing thresholds was considered elevated at ≥ 20 dB Hl following BSA clinical procedures (Table 1) [21].

<table>
<thead>
<tr>
<th>Hashimoto’s group</th>
<th>R250</th>
<th>L250</th>
<th>R500</th>
<th>L500</th>
<th>R1kHz</th>
<th>L1kHz</th>
<th>R2kHz</th>
<th>L2kHz</th>
<th>R4kHz</th>
<th>L4kHz</th>
<th>R8kHz</th>
<th>L8kHz</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>10.5</td>
<td>11.5</td>
<td>8.41</td>
<td>10.4</td>
<td>6.46</td>
<td>7.8</td>
<td>8.17</td>
<td>8.54</td>
<td>7.44</td>
<td>10</td>
<td>12.6</td>
<td>11.6</td>
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<tr>
<td>SD</td>
<td>7.48</td>
<td>13.6</td>
<td>7.45</td>
<td>16.1</td>
<td>6.34</td>
<td>17.71</td>
<td>8.85</td>
<td>15.8</td>
<td>9.29</td>
<td>17.3</td>
<td>16.2</td>
<td>15.9</td>
</tr>
<tr>
<td>Control group</td>
<td>R250</td>
<td>L250</td>
<td>R500</td>
<td>L500</td>
<td>R1kHz</td>
<td>L1kHz</td>
<td>R2kHz</td>
<td>L2kHz</td>
<td>R4kHz</td>
<td>L4kHz</td>
<td>R8kHz</td>
<td>L8kHz</td>
</tr>
<tr>
<td>Mean</td>
<td>5.5</td>
<td>4.4</td>
<td>4.3</td>
<td>2.8</td>
<td>4.3</td>
<td>3.8</td>
<td>5</td>
<td>3.3</td>
<td>4.5</td>
<td>4.5</td>
<td>6.5</td>
<td>6.3</td>
</tr>
<tr>
<td>SD</td>
<td>3.95</td>
<td>3.94</td>
<td>2.93</td>
<td>2.55</td>
<td>3.72</td>
<td>2.75</td>
<td>3.97</td>
<td>2.93</td>
<td>4.26</td>
<td>3.94</td>
<td>3.66</td>
<td>3.93</td>
</tr>
</tbody>
</table>

Table 1: Hearing threshold at each frequency; L: left; R: right; SD: standard deviation Hearing thresholds.
In the patient group, PTA testing at the lowest frequency range revealed mild SNHL in 8 right ears (19.5%) and 9 left ears (22%) of 250 Hz. At 500 Hz, 8 right ears (19.5%) and 4 left ears (9.75%) demonstrated SNHL. For the mid frequency range, SNHL was present in 3 right ears (7%) and one left ear at 1000 Hz. At 2000 Hz, 6 patients had SNHL in the right ear (14.6%) and two in the left ear (4.8%).

For the higher frequency range, 5 right ears (12%) and 5 left ears (12%) presented with mild SNHL at 4 kHz, four had mild SNHL and one had a profound hearing loss. At 8 kHz, 9 patients had SNHL in the right ear (22%) and 8 in the left ear (19.5%). Only one participant in the control group presented with mild SNHL at any frequency. Hearing threshold differed significantly between groups (p<0.05 by independent sample T-test) expect at 4 kHz in the right ear (p=0.1) (Figure 1).

Overall, PTA testing revealed mild SNHL in 16 participants with primary hypothyroidism (39%). To assess differences between the hearing thresholds in patients and controls, patients were divided into two groups with and without hearing loss and the mean thresholds and frequencies compared to the control group (Figure 2).

Otacoustic emission

In patients, TEOAE were absent in 8 left ears (19.5%) and 4 right ears (9.57%), and two patients showed bilateral TEOAE absence. Only one subject in the control group had absent TEOAE, which was in the right ear. Three patients with absent TEOAE in the left ear and 4 with absent TEOAE in the right ear exhibited hearing loss. Moreover, the two patients with absent bilateral TEOAE had bilateral hearing loss. There was no significant difference in TEOAE absence rate between right and left ears in patients (p=0.165 by Pearson's Chi-Square test). There was a significant difference between TEOAE absence rate between patient and control left ears (p=0.01) but not between patient and control right ears (p=0.57).

Summary of findings

<table>
<thead>
<tr>
<th>Findings</th>
<th>Number (n)</th>
<th>Proportion</th>
<th>Correlation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hearing loss (SNHL)</td>
<td>16</td>
<td>39%</td>
<td>6 participants with bilateral tinnitus had hearing loss; the remaining 2 had no hearing loss.</td>
</tr>
<tr>
<td>Tinnitus</td>
<td>8</td>
<td>19.5%</td>
<td></td>
</tr>
<tr>
<td>Absent TEOAE in left ear</td>
<td>8</td>
<td>19.50%</td>
<td>3 had hearing loss and the remaining 5 had no hearing loss.</td>
</tr>
</tbody>
</table>
Absent TEOAE in right ear 4 9.75% All 4 had hearing loss.

Table 2: Summary of findings.

This study found mild SNHL, tinnitus, and reduced outer hair cell function in participants with primary hypothyroidism. The exact figures and correlation are summarized in Table 2.

Discussion

Thyroid autoimmune diseases including acquired hypothyroidism have been reported to affect neurological processes in the auditory system as well as inner ear morphology [17-19,21]. In this study, detailed audiological assessment revealed significantly higher incidences of mild to moderate sensorineural hearing loss (SNHL) and tinnitus in Hashimoto’s thyroiditis (39% and 19.5%, respectively) compared to healthy sex- and age-matched controls. Further, TEOAE absence was higher in patients than controls.

Absence of TEOAE was found in all patients with SNHL, which may indicate cochlear abnormalities induced by congenital hypothyroidism in the rat. This study suggests that hypothyroidism secondary to Hashimoto’s thyroiditis causes mild SNHL, particularly at high frequencies, in addition to bothersome tinnitus. Absent otoacoustic emissions suggest that cochlear outer hair cells are damaged by hypothyroidism.

Conclusion

This study suggests that hypothyroidism secondary to Hashimoto’s thyroiditis causes mild SNHL, particularly at high frequencies, in addition to bothersome tinnitus. Absent otoacoustic emissions suggest that cochlear outer hair cells are damaged by hypothyroidism.

References