

The effect of L-thyroxine hormone therapy on hearing loss in hypothyroid patients

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Objective

The aim of this work was to study whether or not the hearing loss encountered in some hypothyroid patients can be improved with L-thyroxine hormone therapy, as other symptoms of hypothyroidism.

Study design

The study design was a prospective cohort one.

Patients and methods

This study included 30 patients of either sex who were proved to be in a hypothyroid state with an additional symptom of hearing impairment. The study was carried out in Alexandria Main University Hospital and was conducted in a prospective manner. All selected patients were proved to be in a hypothyroid state in the Endocrinology Department. Moreover, their symptom of hearing impairment was confirmed by thorough audiological examination supplemented by pure-tone audiometry plus tympanometry that was carried out in the ENT Department and the associate Audiology Unit. L-thyroxine treatment for hypothyroidism was initiated in all cases for 6 months to maintain a state of euthyroidism. At the end of 6 months, a repeat audiogram and tympanometry were performed in all patients in order to evaluate the efficacy of the said treatment protocol on the hearing in these patients. Cases proved to have hearing impairment irrelevant to the hypothyroid condition were excluded. Informed consent was compulsory for recruitment in this study.

Results

Post-treatment assessment revealed that variable improvement was achieved in 48% of ears, whereas 52% did not respond to the given treatment, with the impairment remaining nearly the same as it was before. Of the improved ears, in 15% of ears hearing levels were restored to normal hearing levels. Thereby, adherence to replacement therapy may reverse the hearing deficits in a few number of hypothyroid patients.

Conclusion

There is a definitive improvement in hearing with the use of L-thyroxine treatment of hypothyroid patients with hearing impairment.

Keywords:

hearing loss, hypothyroidism, L-thyroxine treatment

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Introduction

Auditory acuity reduction has been associated with thyroid gland dysfunction and has been described by numerous authors. It is generally accepted that the auditory system is particularly sensitive to thyroid system disorders when compared with other systems, especially during development. Hearing loss is significantly related to low free T4 and can be the most common otorhinolaryngological manifestation of congenital and acquired hypothyroidism. Deafness may present alone or in association with vertigo and tinnitus [1].

Hearing loss was first reported in acquired hypothyroidism in 1907 [2]. Over a period of time, a distinct association between hypothyroidism and auditory system dysfunction has been reported in

medical text [2], although there are studies that have failed to elucidate a definitive relationship between hypothyroidism and deafness [3–5]. Hypothyroidism is associated with all types of deafness: sensorineural, mixed, and conductive; however, the real incidence and pathophysiology of this hearing loss in these patients are still uncertain.

The incidence of hearing loss varies from 25 to 50% with a higher incidence in congenital hypothyroidism [6,7]. Moreover, the results of audiological evaluation of patients with hypothyroidism under treatment

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with L-thyroxine are conflicting. There are studies that have highlighted the importance of this modality of treatment in improving hearing in hypothyroid patients [8–12], but the literature is also replete with studies that have found no correlation between the two [3–5,13,14].

Considering this, in order to broaden the studies in this line of research, a prospective study was initiated in diagnosed hypothyroid patients with hearing impairment with the aim to study whether or not the hearing loss encountered in some hypothyroid patients can be improved with L-thyroxine hormone therapy, as other symptoms of hypothyroidism.

Patients and methods

This study included 30 patients of either sex who were proved to be in a hypothyroid state with an additional symptom of hearing impairment. The study was carried out in Alexandria Main University Hospital and was conducted in a prospective manner.

Inclusion criteria

- (1) Having hypothyroidism for a sufficient period of time that had resulted into some clinical features of hypothyroidism and confirmed by the laboratory tests, with concomitant complaint of hearing impairment state, documented by thorough otological examination supplemented with pure-tone audiometry (PTA) and tympanometry confirmation.
- (2) Acceptance of the adopted daily dose of l-thyroxin tablet, the adjustment of which was based on regular monitoring of blood thyroxine level every 2 months.
- (3) Adherence to the assigned dose of L-thyroxine to achieve the condition of euthyroid state, for a period of at least 6 months (the adopted period in the present study).

Exclusion criteria

- (1) Hypothyroid patients with hearing impairment due to other causes irrelevant to the hypothyroid condition.
- (2) Recent cases of hypothyroidism – for example, of few weeks after total thyroidectomy, or thyroid ablation therapy; even the relevant patients mentioned a concomitant hearing impairment. In such cases the hypothyroid state lacks the enough time to affect organ function and can be difficult to be blamed in the causation of such hearing defect.

- (3) Patients not restricted to the study protocol (e.g. the regular monitoring during the study period, nonadherence to the given therapy, and lost contact).

Methods

The entire selected group (30 patients) was collected from the Endocrinology Department – Thyroid Clinic. Medical examination supplemented by the proper history taking and laboratory investigations was carried out, including estimation of serum thyroid-stimulating hormone (TSH), total and free T₄, and total and free T₃ levels. Moreover, routine evaluation of sugar level, lipid profile, uric acid, and creatinine was carried out.

In the Otolaryngology Department, each patient was subjected to clinical examination including the following: the external neck, for any thyroid swelling (goiter); oropharyngeal and laryngeal examination, for any edematous soft tissues; nasal examination for mucosal edema; and otological examination, to verify the ear condition, which should be free of any other pathology except the concurrent hearing impairment. Slight drum retraction or thickening was acceptable.

In the Audiology Unit confirmation of the hearing impairment was verified with PTA and tympanometry.

The PTA determines the mean threshold levels in air conduction and bone conduction along the frequency range of 250–8000 Hz. A pure-tone average refers to the average of hearing threshold levels at a set of specified frequencies: typically 500, 1000, 2000, and 4000 Hz. This value gives a snapshot of an individual's hearing level in each ear.

PTA determined the type of hearing loss whether sensorineural hearing loss (SNHL), mixed deafness or conductive deafness. Thereafter, the hearing threshold level for each patient was calculated as the mean thresholds of 500, 1000, 2000, and 4000 Hz frequencies in air-conduction curve.

According to Clark (1981), the degree of hearing loss was classified as follows:

- (1) Minimal (15–25 dB).
- (2) Mild (26–40 dB).
- (3) Moderate (41–55 dB).
- (4) Moderately severe (56–70 dB).
- (5) Severe (71–90 dB).
- (6) Profound (>90 dB).

Tympanometry was performed using single-component, single-frequency tympanometry with a probe tone of 226 Hz. Each time we recorded the type of middle ear compliance and the stapedius reflex.

Audiological assessment was carried out twice: the first (preliminary) when the assigned patient was enrolled in the study, and the second (final) when the patient reached the end of 6 months during which he/she maintained in euthyroid state, as monitored every 2 months to ensure stability. The period which has been exhausted in adjusting the given l-thyroid dose to achieve a steady and continuous euthyroid state was not included. Only those patients who maintained a euthyroid state for 6 months were considered eligible for the second (final) assessment.

Both PTA and tympanometry studies were performed each time in the same sound-proof booth by the same examiner and using the same tools to minimize the possible interpersonal evaluation errors. While recording the PTA or the tympanometry, the patient might require retesting more than once for confirmation and achieving the proper assessment (test-retest reliability).

Results

Of 30 patients in our study, 24 (80%) were female and six (20%) were male. The age group of patients ranged from 26 to 58 years, with a mean age of 42 years (Table 1).

Table 1 Sex and age distribution in the studied group (n=30)

	N (%)
Sex	
Male	6 (20)
Female	24 (80)
Age (years)	
20–30	6 (20)
30–40	10 (33.3)
40–50	10 (33.3)
50–60	4 (13.4)
Minimum–maximum	26.0–58.0
Mean±SD	41.93±9.13
Median	41.8

Table 2 The reported serum levels of thyroid-stimulating hormone and free T4 among the selected patients (n=30) at the start of the study

	Min.–max.	Mean±SD	Median
TSH (normal=0.4–4 mU/l)	10.20–35.9	22.01±6.4	23.10
Free T4 (normal=0.9–1.9 ng/dl)	0.25–0.80	0.43±0.11	0.43

Max., maximum; min., minimum; TSH, thyroid-stimulating hormone.

The acquired hypothyroid state reported among our patients was of the manifest type and can be attributed to variable causes –namely, Hashimoto's disease in 16 (53.3%) female patients, post-thyroiditis in four (13.3%) female patients, postpartum in one (3.3%) female, total or partial thyroidectomy in six (20%) patients (three female and three male), and idiopathic in further three (10%) male patients.

The reported serum levels of TSH and free T4 for the total of 30 patients who constituted the sample of the present study and diagnosed accordingly as having the primary (manifested) type of acquired hypothyroidism varied from 10.2 to 35.9 mU/l for TSH, with a mean of 22.1 mU/l, and from 0.25 to 0.80 ng/dl for free T4, with a mean of 0.43 ng/dl (Table 2).

In the present study we encountered 28 ears of 60 (46.6%) affected by SNHL: 22 (36.6 %) ears by mixed deafness, four (6.6 %) ears by CD, and six (10 %) ears possessing normal hearing thresholds. All selected patients were complaining of hearing impairment in both ears, but on audiological assessment 24 (80%) patients were actually suffering from bilateral hearing impairment, whereas six (20%) patients were suffering from unilateral hearing impairment (Table 3).

As regards the reported degree of hearing impairment, among the 54 affected ears, there were nine (16.6%) ears with minimal hearing loss (15–25 dB), 26 (48%) ears with mild hearing loss, and 19 (35.4%) ears with moderate hearing loss. Among those ears with SNHL ($n=28$), six were of minimal degree, 13 were of mild degree, and nine were of moderate degree. In ears with MD ($n=22$), two were of minimal hearing loss, 12 were of mild hearing loss, and eight were of moderate hearing loss. Among ears with CD ($n=4$), one was of minimal hearing loss, one with mild hearing loss, and two with moderate hearing loss. In the present work we did not encounter severe forms of hearing impairment among our hypothyroid patients (Table 4).

When correlating the severity of hearing loss among patients and the relevant levels of TSH and free T4, a statistically significant value was detected – the higher the hypothyroid state, the greater the severity of hearing impairment (Tables 5 and 6).

Final (second) assessment results

In the second assessment, we found 13/60 ears reporting within-normal thresholds; five were already normal since the first assessment, and eight ears achieved normal level by the end of the study. Moreover, there were 9/60 ears with less hearing acuity as compared with their hearing

acuity at the start of the study. The threshold level remained stationary, or moved within 5 dB up or down, in 20/54 ears throughout the treatment period; thus, no change was reported. Thus, a total of 29/54 (53.7%) ears did not change or become worse by the end of the study period, despite the given treatment. However, 17/54 ears gained 5–10 dB, whereas 9/54 ears gained 10–16 dB. This gain allowed 8/54 (15%) of previously impaired ears to restore hearing levels to normal hearing levels by the end of the study –that is, it is expected that the concomitant hearing loss could be reversed in 15% of ears with proper treatment (Table 7).

Those ears that attained better hearing might change from higher category of hearing loss into lesser

Table 3 Type of hearing in studied ears

Type of hearing as reported in first assessments	Number of ears in first assessment (n=60 ears)
Normal	6
SNHL	28
MD	22
CD	4

SNHL, sensorineural hearing loss.

Table 4 Correlation between the type of hearing loss and its degree among the affected ears (n=54)

Degree of hearing loss	Ears with SNHL (n=28)	Ears with MD (n=22)	Ears with CD (n=4)	N (%)
Minimal HL (15–25 dB)	6	2	1	9 (16.6)
Mild HL (26–40 dB)	13	12	1	26 (48)
Moderate HL (41–55 dB)	9	8	2	19 (35.4)

HL, hearing loss; SNHL, sensorineural hearing loss.

Table 5 Correlation between the severity of hearing loss and the levels of the relevant thyroid-stimulating hormone among 54 ears with hearing impairment

Detected levels of TSH and its average (normal=0.4–4 mU/l)	Severity of hearing loss			r_s	P
	Minimal HL (15–25 dB)	Mild HL (26–40 dB)	Moderate HL (41–55 dB)		
Min.–max.	10.2–15.5	13.5–25.4	23.2–35.3	0.675	<0.001*
Mean±SD	12.9±2.2	21.9±3.44	27.3±4.62		
Median	12.5	23.0	25.10		

HL, hearing loss; max., maximum; min., minimum; r_s , Spearman's coefficient; TSH, thyroid-stimulating hormone. *Statistically significant at $P \leq 0.05$.

Table 6 Correlation between the severity of hearing loss and the levels of the relevant T4 among 54 ears with hearing impairment

Detected levels of T4 and its average (normal=0.9–1.9 ng/dl)	Severity of hearing loss			r_s	P
	Minimal HL (15–25 dB)	Mild HL (26–40 dB)	Moderate HL (41–55 dB)		
Min.–max.	0.37–0.80	0.32–0.60	0.24–0.40	0.517	0.003*
Mean±SD	0.53±0.2	0.42±0.1	0.36±0.1		
Median	0.50	0.40	0.35		

HL, hearing loss; max., maximum; min., minimum; r_s , Spearman's coefficient. *Statistically significant at $P \leq 0.05$.

Table 7 Pure-tone audiometry findings of 60 ears at the final assessment after achieving an euthyroid state for 6 months

Type of hearing as reported in first and second assessments	Number of ears in first assessment (n=60 ears)	Number of ears in second assessment (n=60 ears)			
		Hearing deterioration (n=9 ears)	No change (n=25 ears) ^a	5–10 dB gain (n=17 ears)	10–16 dB gain (n=9 ears)
Normal	6	1	5	–	–
SNHL	28	3	11	9	5
MD	22	5	9	4	4
CD	4	–	–	4	–

SNHL, sensorineural hearing loss. ^aThis number included five ears having within normal threshold since first assessment.

category, or stayed within the same category if the gaining was only few dBs. Thus, the final assessment of the affected ears ($n=54$) reported hearing levels within normal in eight ears, of minimal losses in 17 ears, of mild losses in 20 ears, and of moderate losses in nine ears. In general, an improvement in the hearing status was recorded in 26/54 (48%) ears after 6 months of therapy with proper L-thyroxine dosage. This was statistically significant ($P \leq 0.05$) (Table 8).

As regards the aim of the study, we can postulate that maintaining a euthyroid status for at least 6 months could bring an improvement in the hearing disability in about 48% of the affected ears. However, the gained hearing ability is not so marked and ranges only from 5 to 16 dB. Moreover, reversibility of the acquired hearing impairment, with restoring nearly a normal hearing level, in such cases, could be expected in 15% (8/54) of affected ears.

Tympanometry findings in hypothyroid patients usually demonstrate type A curve. If the hearing loss is of MD or CD type the tympanometric finding may change into type C or B. Tympanometric findings of the present study demonstrated that the majority of the impaired ears had type A curve (72.3%) and 22.7% had type C curve, whereas none had type B curve. Thus, the CD element that was encountered among some of our tested ears was proposed to originate from Eustachian tube dysfunction.

Improvement in PTA results usually is accompanied by similar improvement in middle ear compliance. This was observed when 7/15 (46.6%) ears with previous type C curve changed into type A curve with the given substitutional therapy (Fig. 1).

Table 8 The progress of the average hearing threshold level after achieving an euthyroid state for 6 months' period

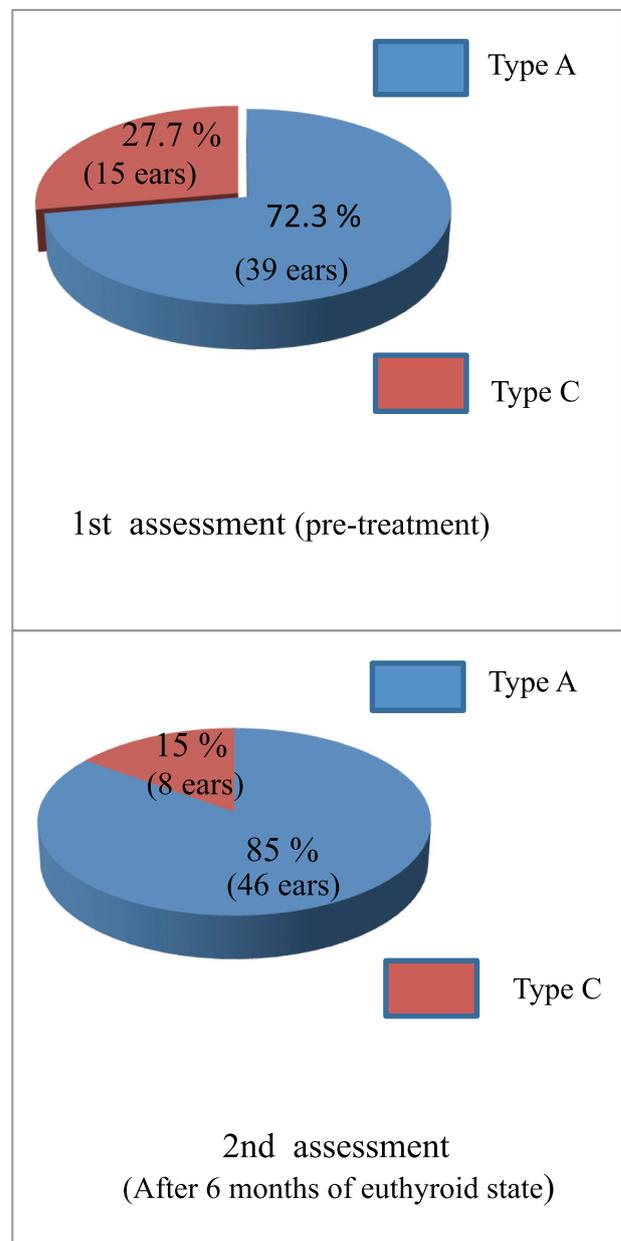
Severity of hearing	Type of hearing loss [N (%)]		
	SNHL (n=28)	Mixed (n=22)	CHL (n=4)
Before treatment			
Normal	0 (0.0)	0 (0.0)	0 (0.0)
Minimal	6 (21.4)	2 (9.1)	1 (25.0)
Mild	13 (46.4)	12 (54.5)	1 (25.0)
Moderate	9 (32.1)	8 (36.4)	2 (50.0)
After treatment			
Normal	4 (14.3)	2 (9.1)	2 (50.0)
Minimal	8 (28.6)	8 (36.4)	1 (25.0)
Mild	11 (39.3)	8 (36.4)	1 (25.0)
Moderate	5 (17.9)	4 (18.2)	0 (0.0)
<i>P</i>	0.001*	0.003*	0.048*

CHL, conductive hearing loss; SNHL, sensorineural hearing loss. *Statistically significant at $P \leq 0.05$.

Discussion

Insufficient synthesis of thyroid hormones for various reasons causes hypothyroidism. The most common cause of acquired hypothyroidism is Hashimoto's thyroiditis. The disease usually affects women much more frequently compared with men, and the individuals are for the most part of middle age. In the present study, female patients constituted 80% of the sample, and Hashimoto's disease was the cause of hypothyroid state in 53.3%, and thyroidectomy was accused for 20%, and the reported mean age was 42 years. Our findings are in

Figure 1



Pie chart illustrating the percentage of type A and type C tympanograms encountered in the first and second assessments among the tested ears ($n=60$)

great agreement with those of Arduç *et al.* [15], who found that 74% of patients had hypothyroidism secondary to Hashimoto's thyroiditis and 26% had hypothyroidism due to previous partial thyroidectomy, and with those of Malik *et al.* [16], who found that age varied between 21 and 40 years in their study group comprising 45 hypothyroid patients, with female patients constituting 77.77%.

The most common symptom of hypothyroidism with regard to ear, nose, and throat is hearing insufficiency accompanied with cochlea-vestibular symptoms such as tinnitus and dizziness. Hence, the evaluation of hearing in hypothyroidism assumes clinical importance and has been studied by some earlier studies [16,17].

The exact incidence of hearing loss in acquired hypothyroidism is not universally agreed upon. Some studies encountered an incidence of 25–80% among affected patients and declared that the replacement therapy, which often requires lifelong treatment, sometimes can solve a person's hearing loss [18]. Some other studies reported an incidence of 25% among patients with acquired hypothyroidism and 35–50% among those with congenital hypothyroidism [19,20]. Other studies on hypothyroid patients reported as follows: Malik *et al.* [16] reported an incidence of 71.11% among their 45 patients and the degree of hearing loss increased with the duration of the disease; Thornton and Jarvis [19] reported an incidence of 36% (demonstrated an average threshold >25 dB) among their 21 hypothyroid patients; Anand *et al.* [21] reported bilateral subjective hearing loss that affected 45% of their 25 patients, and 20% of them had nearly symmetrical hearing loss in both ears. They added that, in 55.55% of patient, the hearing loss was of insidious onset, whereas in 33.33% it was of a progressive course, and tinnitus was also complained by 15.55% of patients and dizziness by 6.66% of patients.

However, despite these findings other researchers have failed to document definite evidence to support the association between hypothyroidism and deafness, and also the site of auditory lesion remains speculative [16].

In the present study we did not determine the incidence of hearing loss among hypothyroid patients as the inclusion criteria imposes that every selected patient should have a concomitant hearing impairment beforehand. The main concern of the current work was to explore whether there is any role of the given

replacement therapy in improving such hearing deficit. Accordingly, we have no comment about the exact incidence of hearing impairment among hypothyroid patients in our center. This actually requires another wider study assigned to test hearing among all hypothyroid patients presented to the thyroid clinic.

The encountered hearing impairment presented bilaterally in 80% of patients and was nearly symmetrical in both ears in about 50% of them. However, the majority of cases with asymmetrical types of hearing impairment demonstrated average hearing losses in the two ears of approximate levels. We did not encounter patients who reported progressive deafness, but 23% of them reported variable forms of tinnitus and 13% suffered from dizziness attacks as well.

The obtained results in the current study are in accordance with those encountered before [15,16]. They emphasized that hearing impairment, in about 50% of ears of hypothyroid patients, is usually of insidious gradual onset, progresses slowly, and affects both ears in nearly an equal manner. However, in the other 50% of ears the hearing impairment may be distributed asymmetrically between the two ears and exhibited variation in the type and the severity of deafness.

Once it was accepted that there is a relationship between hypothyroidism and hearing loss, the location of the affected part in ear has been subject to many new studies, which showed that one or multiple parts such as endocochlear, retrocochlear, or central hearing ways might be affected. Moreover, the occurrence of edema of the Eustachian tube and middle ear mucosa and the possible ossicular chain dysfunction can predispose for CD in hypothyroid patients [16,17,19]. Dokianakis *et al.* [22] detected moderate SNHL in 8/23 and MD in 4/23 of their studied hypothyroid patients. Malik *et al.* [3] detected that 25% of patients had SNHL, 28.10% had MD, and 46.90% had CD. They advocated that subjective hearing loss in hypothyroid patients may be either due to actual conductive or SNHL.

Our obtained results in this concern is greatly in agreement with those recorded before. In the present study, SNHL constituted the main type of hearing impairment among the tested ears (51.8 %), followed by MD that was detected in 40.7%, whereas CD affected only 7.5% of ears. As reported in the current study, most of the patients (87%) were snorers and

complained of nasal stuffiness, and in 66% of them edema and puffiness of the nasal and pharyngeal mucosa was found. These findings might reflect a similar condition also present and affecting the Eustachian tube and middle ear mucosae. Moreover, in 18/54 of ears the drum membrane was found slightly retracted and thickened. In 15/54 ears the middle ear compliance was diminished, as detected with type C tympanogram. These reported findings were presumably accused for the conductive element of hearing impairment that was combined with SNHL in 22/54 ears that presented with MD or a pure CD in further 4/54 ears. Previous studies declared that conductive hearing loss in hypothyroidism is secondary to Eustachian tube and middle ear mucosal edema and accumulations of glycosaminoglycans [19].

Most trials in this concern have described mild–moderate deafness to occur in patients with acquired hypothyroidism. Such deafness often occurred insidiously and nearly symmetrically in both ears. In a previous study by Anand *et al.* [21] an incidence of 20% symmetrical or nearly symmetrical bilateral hearing impairment was reported among hypothyroid patients. In a study by Santos *et al.* [19], which was conducted on 30 hypothyroid patients, bilateral moderate SNHL was reported in 11 (36.6%) patients. Moreover, in the study by Karakuş *et al.* [23], mild hearing impairment was present in the majority of ears (73.3%) and moderate impairment in only 26.6% of ears, whereas severe or profound hearing impairment was not found in any case.

In the current work most of the hypothyroid patients with hearing impairment demonstrated mild–moderate hearing loss (83.4% of ears – mild in 48% ears and moderate in 35.4% ears), whereas minimal losses were observed in 16.6% ears. None of the selected patients suffered severe deafness and even the occurred hearing deficits were not of much annoyance to them. Thus, our results in this concern are in great accordance with those previously obtained. The degree of hearing loss was reported by some studies to increase with the duration of the disease. Moreover, there was a significant correlation between the hypothyroid state and the concomitant hearing impairment; the more the hypothyroidism, the greater the hearing impairment. This was reported before [15,16,19] as also, to some extent, in our study. Despite this, debate still remains as regards the underlying pathophysiology and even hypothyroid-associated deafness as a specific entity [16,23].

There are diverse views on improvement in hearing in hypothyroid patients with thyroxine treatment. Studies by Van't Hoff and Sturat [9], Rubinstein *et al.* [10], and Anand *et al.* [22] have reported variable improvements in hearing following thyroxine therapy. Hearing loss in hypothyroidism was found remediable and even reversible after proper replacement therapy. In a study by Di Lorenzo *et al.* [24] a significant improvement in the hearing condition was reported in hypothyroid patients, after 6–12 months of L-thyroxine replacement therapy. Similarly, Karakuş *et al.* [23] found that the hearing loss of the patients improved significantly after 4 months of substitutional therapy. They reported that SNHL improved completely in 43.3% of ears, which returned to normal and the cochlear changes had returned to normal following substitution therapy. Malik *et al.* [16] reported post-treatment improvement in hearing in 12.5% ears with SNHL, 11.11% ears with MD, and in 30% of ears with CD. Even the encountered tinnitus and dizziness improved in 57.14% and 44.4 patients, respectively. However, studies by Post [13], DeVos [14], Parving *et al.* [3] and yet another study by Parving *et al.* [4] have not reported any significant improvement in hearing after treatment with thyroxine. Thus, there are still a number of authors expecting variable improvement in the hearing defect (at times in a dramatic manner) on correction of the hypothyroid state, and yet others refute that any objective hearing improvement in this context actually occurs [16].

In the present study the assigned patients should adhere to a period of at least 6 months of continuous replacement therapy to achieve a condition of euthyroid state. Adopting this period in the present study was according to that advocated by some previous studies as being the average time required for the body systems in hypothyroid patients to restore normal function. Assessment of the assigned patients by the end of this period (final assessment) revealed that about 48% of ears achieved variable degrees of improvement. Reversibility of the hearing impairment and restoration of normal hearing was reported in 15% of the affected ears. Improved hearing levels are usually associated with correction of middle ear compliance. However, no improvement, or even minimal deterioration, was recorded in 52% of ears despite the given treatment.

Because the location, degree of reversibility, and underlying mechanism of the hearing deficits, when associated with hypothyroidism, have yet not fully defined. Moreover, because there is a possibility of

other etiologies for the hearing impairment status in such patients, there is uncertainty whether the PTA data reflects the actual role of L-thyroxine. Continuation of the pretreatment hearing impairment state, or its deterioration, despite achieving euthyroidism, could be referred to the causes mentioned before. This was encountered in 52% of ears in the present study when tested using PTA in the final assessment. However, as also was mentioned previously [16], it was found that the overall sense of well-being achieved results in a subjective improvement in hearing regardless of whether this was actually real [16].

Moreover, most of the studies of brainstem electric response do not show significant reversal following L-thyroxine therapy. Thus, it is widely accepted that improvement in hearing following L-thyroxine therapy is attributed to improved general condition of the patient resulting in improved cooperation in psychoacoustic testing, the so-called functional improvement in central deafness [24].

Conclusion

The present study was carried out on thirty patients with confirmed hypothyroidism and hearing impairment. Complete clinical examination and laboratory investigations were carried out as regards the audiological system. It was found that hypothyroidism affects the ear at multiple sites producing various types of hearing impairment – namely, conductive, sensorineural, and mixed. The patients were then given L-thyroxine and follow-up was carried out after 6 months of being in euthyroid state, which revealed a statistically significant improvement in hearing threshold in 48% of ears, in which sensory impairment was more common to be improved. The middle ear compliance and pressure, on impedance audiometry, also improved.

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Conflicts of interest

There are no conflicts of interest.

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