Evaluation of TSH And T4 Levels in Idiopathic Sudden Sensorineural Hearing Loss Patients

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Abstract

**Objectives:** to research on TSH, T4 levels and audiologic values of unilateral ISSNHL patients.

**Methods:** The files of patients who treated with sudden sensorineural hearing loss diagnosis were retrospectively reviewed. Hematologic and biochemical parameters, vitamin B-12, free T4 (tetraiodothyronine), TSH (thyroid stimulating hormone), the initial and last audiologic report after treatment were recorded.

**Results:** The study group consist of 60 patients (32 patients responder, 28 patient nonresponder) and the control group had 30 healthy persons. There was no significant difference in TSH and free T4 between the responder group and the control group. Serum free T4 was significantly lower in the nonresponder group comparing to the control group. There was no significant difference in TSH and T4 levels between subgroups of sudden hearing loss.

**Conclusion:** Small variations in T4 levels can be a risk factor for ISSNHL even if the patient has euthyroidism and these changes can be either acute or chronic.

**Keywords:** Sudden; Hearing loss; Thyroid

**Introduction**

Sudden sensorineural hearing loss (SSNHL) is defined as hearing loss of at least 30 dB at 3 consecutive frequencies in less than 3 days. A number of agents are responsible for its etiology but they have only been determined in 10-15% of cases [1]. Viral infections, vascular reasons, intracochlear membrane rapture, perilymphatic fistula, metabolic diseases, inner ear autoimmune disease, acoustic tumors, psychogenic diseases are at the forefront of its etiology [2]. 95% of the cases are unilateral and approximately 90% of cases with unknown etiology are defined as idiopathic sudden sensorineural hearing loss [3] (ISSNHL).

Metabolic diseases are a risk factor for sudden hearing loss and one of these diseases is thyroid hormone disorders. Thyroid hormone levels are quite important for normal functioning of the cochlear and it has been reported that hearing functions have dynamic changes in hyper-thyroidism states [4]. Thyroid hormones are crucial for complete normal development of cochlear. Thyroid hormones are also necessary for normal development of middle ear and ossicular chain [5]. It is a well known fact that children born with congenital hypothyroidism can develop sensorineural hearing loss if thyroid hormone replacement is not done [6].

The etiology of ISSNHL has not been completely shed light on. It has been reported in a study that the rate of partial and total recovery of ISSNHL patients is 57% and the average recovery is 15 Db [7]. The etiology of one case in these patients was found to be history of thyroid gland disease and in the other cases cigarette was found to be responsible. Studies that research on the relationship between idiopathic sudden sensorineural hearing loss and thyroid hormones are limited in the literature. In this study we aimed to research on TSH, T4 levels and audiologic values of unilateral ISSNHL patients who were treated and either recovered or didn't recover by comparing them to a healthy control group.

**Materials and Method**

The files of patients who presented with sudden sensorineural hearing loss and were treated in Necmettin Erbakan University Meram Medical Faculty Ear Nose and Throat department between the years 2007 and 2016 were retrospectively reviewed. A total of 352 patient files were collected. Hematologic (leukocyte, hemoglobin, hematocrit, platelet values) and biochemical results, vitamin B-12, free T4 (tetraiodothyronine), TSH (thyroid stimulating hormone), temporal bone MRI, the initial and last audiologic report after treatment, history and physical examination were recorded from the patient files. Cigarette smokers, those with blood disorders, coronary artery disease, hypertension, lipid and cholesterol metabolic disorders, endocrine disorder, diabetes melitus, pediatrict patients, those with audiovestibular tumors, those with a history of drug use that is ototoxic and that affects thyroid function, those who have had a viral infection in the last 1 month, and those with abnormal hematologic
and biochemical blood parameters were excluded from the study. After this exclusion sudden hearing loss with unknown etiologic risk factor was termed ISSNHL.

A total of 32 patients who met the criteria for recovery of more than 10 dB (the group that responded to treatment) and a total of 28 patients with a recovery of less than 10 dB (the group that didn't respond to treatment) in the audiologic tests were included into the study therefore forming two groups. Healthy individuals without any documented disease in the hospital's data base were chosen to form the control group. TSH, free T4, initial and last audiologic values after treatment of the 60 patient group and 2 subgroups accepted to have ISSNHL were evaluated by comparing to healthy individuals. Calculation of the average audiologic values was done by dividing the total of 250 Hz, 500 Hz, 1000 Hz, 2000 Hz, 4000 Hz and 8000 Hz by six. Treatment of ISSNHL was done by administration of 1 mg/kg/day predniizonol (I.V.), piracetam (nootropil) ampul 8 mg/day (I.V.) (UCB Pharma, Turkey), and Dextran RMI (Rheomacrodex) 1000 ml/day (I.V. (Eczacıbaşı,Turkey). ADVIA Centaur™ Siemens Healthcare (NY, USA) equipment was used for measurement of TSH and T4 values. Audiometric measurements were done with Clinical Audiometer AC33 (Denmark) in a sound proof cabin. This study was approved by the Local Ethics Committee of Necmettin Erbakan University Meram Medical Faculty.

Statistical analysis

Data was analyzed using SPSS 23.0 (USA) program. Comparison between those that showed recovery, those that didn't show recovery and the control group was done by Krusal Wallis analysis of variance whereas post-hoc comparison was done by Mann Whitney U test with Bonferroni correction. Pearson correlation test was done to investigate whether or not there was any correlation between T4, TSH and the initial and last audiologic value in the groups. P<0.05 was accepted to be significant.

Results

There were 18 females and 14 males in the 32 patient group that responded to treatment (responders) and their average age was 42.75 (19-72) years. There were 16 females and 12 males in the 28 patient group that did not respond to treatment (non-responders) and their average age was 44.50 (19-68) years. The control group had 30 healthy persons, 16 females and 14 males with an average age of 47.21 (19-68) years. There was no significant difference in age and sex between the groups (p>0.05). There was no significant difference in TSH and free T4 between the responder group and the control group (p>0.05). Comparing the nonresponder group to the control group, there was no significant difference in TSH (p>0.05), whereas T4 was significantly lower in the nonresponder group (p<0.05). Comparing the subgroups of sudden hearing loss, there was no significant difference in TSH and T4 (p>0.05). Comparing the sudden hearing loss groups to the control group, the initial and last audiologic values were significantly higher in the sudden hearing loss groups (p<0.05). Comparing the sudden hearing loss groups, the last audiometric values were significantly higher in the nonresponder group (p<0.05). TSH and T4 values in all the patients that were diagnosed with ISSNHL without discriminating between the groups were compared to those in the control group and only T4 levels were found to be significantly lower in the sudden hearing loss group (p<0.05). TSH and free T4 values in the sudden hearing loss groups had no significant correlation with the initial and last audiometric values (average values ± standard deviation have been given in Tables 1 and 2).

| Table 1: Demographic features, Laboratory and audiometric results of the study groups ISSNHL: idiopatic sensorineural sudden hearing loss, TSH: thyroid stimulating hormone. T4: tetraiodothyronine, dB: desibel, Initial air-bone conduction: Initial values at the time of diagnosis, Last air-bone conduction: values after medical treatment. |
| --- |
| **Comparison of Group** | **Control** | **Responder** | **Nonresponder** | **Responders** | **Control** |
| **p value** | **Responder group (n=32)** | **Nonresponder group (n=28)** | **Responder group (n=28)** | **Nonresponder group (n=28)** |
| Age | 0.098 | 0.088 | 0.076 | 0.337 |
| Gender | 0.083 | 0.094 | 0.079 | 0.445 |
| TSH | 0.753 | 0.784 | 0.682 | 0.148 |
| Free T4 | 0.382 | 0.003 | 0.412 | 0.007 |
| Initial air conduction | 0.001 | 0.001 | 0.06 | 0.001 |
| Initial bone conduction | 0.001 | 0.001 | 0.06 | 0.001 |
| Last air conduction | 0.208 | 0.001 | 0.017 | 0.001 |
| Last bone conduction | 0.704 | 0.001 | 0.024 | 0.001 |

| Table 2: P values are showing comparison between the groups. |
Discussion

In this study free T4 levels in sudden hearing loss patients who did not respond to treatment was found to be lower than in the control group. Furthermore evaluation of ISSNHL patients without dividing into subgroups, free T4 levels was also found to be lower than in the control group. From this it can be hypothesized that even with small variations of T4 levels hearing function can be affected.

The relationship between hearing and thyroid function has been identified in various studies. It has been reported that hearing function can be impaired in congenital and acquired hypothyroidism, endemic cretinism, pendred syndrome, and in patients resistant to thyroid hormone [8–11].

The relationship between thyroid hormone and hearing function dates back to the prenatal period. Thyroid hormone is necessary for the development of hearing function [9]. In the prenatal period, organ of corti is quite sensitive to thyroid hormone at the maturation stage and lack of it can cause permanent hearing loss. Thyroid hormone increases the expression of genes that encode for thyroid hormone receptor [8]. Even though important proteins coded by these genes are present for the physiologic process and structural development of the inner ear [12], the molecular mechanism behind permanent hearing loss due to hypothyroidism is still unknown (Figure 1).

Through human and animal studies acute thyroid hormone deficiency states have been shown to result in changes in hearing function. Psaltakos et al. [14] used pure tone audiometry and transient evoked otoacoustic emission to record the hearing levels of 52 patients who had thyroidectomy operation before and 6–8 weeks after the operation. Levothyroxine was not administered to the patients and a follow up of thyroid stimulating hormone was done after the operation. Hearing levels increased in all the postoperative audiometric measurements and when otoacoustic measurements were compared before and after surgery, S/N ratio was found to be lower after surgery. Changes in the levels of thyroid hormone and its effects on hearing function in this study coincided with our thesis. However changes in free T4 levels of the groups in our study were neither determined to be acute nor chronic. This is because in our study even though thyroid hormone levels were within the normal reference range, hearing function can be affected by small variations in the level of free T4 and this should not be ignored. Contrary to this Mra and Wax [15] did not identify any changes on either audiogram or otoacoustic emission for a period of 6 weeks of acute decrease in thyroxin after total thyroidectomy operation. As for this study it was suggested that acute changes in thyroid hormone levels did not affect hearing function and therefore it was thought that chronic changes might be more effective.

Thyroid hormone level is an important prognostic factor as an etiology for sudden hearing loss. In a study done by Narozy et al. [16] high dose steroid treatment for 10 days, normal thyroid function tests, preserved labyrinth function and disease being diagnosed during spring season were taken to be positive prognostic factors. This study is supported by the fact that there is a positive correlation between free T4 levels and response to treatment in our study. In a case-control study done by Nakashima et al. [17] 109 ISSNHL patients were evaluated and it was reported that sensitivity to cold, hypertension and history of thyroid disease were risk factors for sudden hearing loss. Just like in the last study even though patients in our study had euthyroidism it should be kept in mind that history of thyroid gland disease can be a risk factor for hearing loss. This is supported by the fact that 6 of the patients in our study had a history of thyroid disease.

At a glance the limitations of our study is it being a retrospective study and the number of patients being relatively small. Patients were not questioned on the possible risk factors and data was only obtained from evaluation of the patient files. A meaningful free T4 cut off value that can be used to predict the risks and prognosis of sudden hearing loss was not calculated.

Conclusion

Thyroid hormone level is an important factor for normal hearing function. Small variations in T4 levels can be a risk factor for ISSNHL even if the patient has euthyroidism and these changes can be either acute or chronic. To clear these uncertainties controlled prospective studies with wider patient population is required.

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