Received: 3 August 2021 | Revised: 9 November 2021 | Accepted: 31 January 2022

DOI: 10.1002/lio2.755

ORIGINAL RESEARCH

Laryngoscope Investigative Otolaryngology

Autoimmune thyroiditis in patients with sudden sensorineural hearing loss

Xiao-Mei Sun MD | Shi-Min Zhuang MD | Zhi-Wen Xiao MD | Jia-Qi Luo MD | Zhen Long MD | Lin-Chan Lan BA | Hui-Qing Zhang BA | Guan-Ping Zhang MD

Department of Otolaryngology Head and Neck Surgery, The Sixth Affiliated Hospital of Sun Yat-sen University, Guangzhou, China

Correspondence
Guan-Ping Zhang, Department of Otolaryngology Head and Neck Surgery, The Sixth Affiliated Hospital of Sun Yat-sen University, Guangzhou, 510655, China.
Email: zhguanp@mail.sysu.edu.cn

Abstract

Objective: This study aims to evaluate the association between autoimmune thyroiditis and Sudden Sensorineural Hearing Loss (SSNHL).

Methods: Hundred and five patients with SSNHL were enrolled. Audiometric tests, serum thyroid autoantibodies (TPOAb, TgAb) were studied. Based on the thyroid autoantibody results, patients were divided into two groups: thyroid autoantibody-positive and negative. The relationship between thyroid autoimmunity and audiological characteristics was analyzed.

Results: Twenty-six patients (24.8%) of the SSNHL had thyroid autoantibody elevated. The pure tone average (PTA) of patients with and without thyroid autoantibody is 60 ± 38.51 and 54.99 ± 33.87 dBHL, respectively. The PTA was significantly improved in both groups after treatment ($p < 0.001$), but the hearing gains were similar in both groups ($p = 0.205$). Hearing loss of 2000–8000 Hz was worse than 125–1000 Hz among thyroid autoantibody-positive patients ($p < 0.05$), but the hearing improvement of both groups have no significant difference. The hearing improvement of 125–1000 Hz is significantly better than 2000–8000 Hz among patients with thyroid autoantibody negative ($p < 0.05$).

Conclusions: We speculate that a potential association between thyroid autoimmunity and SSNHL. Thyroid autoimmunity may be a pathogenesis factor of SSNHL and associated with more severe hearing loss of high-frequency hearing.

KEYWORDS

autoimmune thyroiditis, inner ear disease, sudden sensorineural hearing loss, thyroid autoantibody, thyroid function

1 | BACKGROUND

Sudden sensorineural hearing loss (SSNHL) is a common and alarming complaint that often results in an urgent visit to ENT.[1] SSNHL affects 5 to 60.9 per 100,000 people annually, and the incidence is increasing.[2–5] The common symptoms are hearing loss, sometimes

Xiao-Mei Sun and Shi-Min Zhuang have contributed equally to this study and also considered as co-first authors.

accompanied by tinnitus, ear fullness, dizziness, vomiting, and/or nausea. The diagnostic audiometric criterion of SSNHL is a decrease in the hearing of 30 decibels affecting at least three consecutive frequencies.[5] The cause of SSNHL is still not clear, it is generally believed that it is related to viral infection, vascular accident, autoimmunity, and so on.[6–9]

Thyroid is the largest endocrine organ in the human body, and it secretes thyroid hormone, which is necessary to maintain the normal metabolism of the human body and can promote protein synthesis and regulate the metabolism of sugar, fat, water, salt, and vitamins. In addition, thyroid hormones play an important role in the development and maturation of spiral ganglion cells, hair cells, and hearing, formation of nerve myelin, oxygenation, and metabolism of Corti and stria vascularis.[10–16] Hyperthyroidism and hypothyroidism both can affect hearing, such as an increase change in wave V latency in the auditory brainstem response (ABR) and higher audiometric thresholds and TOAEs abnormality.[17–19]

Hashimoto's thyroiditis, with an incidence of 0.3–1.5 cases per 1000 people, is characterized by increased thyroid volume, lymphocyte infiltration of parenchyma, and antibodies specific to thyroid antigens.[20] TgAb (antithyroglobulin antibody) and TPOAb (antithyroperoxidase) are the common indicators for the diagnosis of autoimmune thyroiditis. Thyroid peroxidase can stimulate the body to produce TPOAb, which destroys thyroid cells through the activation of complement and antibody-dependent cell-mediated cytotoxicity and meanwhile causes the activation and infiltration of Th1 cells, resulting in immune damage.[21–23]

Several studies reported that Hashimoto's thyroiditis is associated with BPPV, Meniere's disease.[24,25] But few studies have been conducted on the relationship between sudden sensorineural hearing loss and autoimmune thyroiditis.

This retrospective study aims to investigate the prevalence of autoimmune thyroiditis in patients with SSNHL, and to evaluate the possible association between thyroid autoimmunity and sudden sensorineural hearing loss.

2 | MATERIALS AND METHODS

2.1 | Participants

Our study retrospectively analyzed patients diagnosed as SSNHL in the department of otorhinolaryngology of the sixth Affiliated Hospital of Sun Yat-Sen University between 2018 and 2020, with the approval of the Ethics Committee of the Sixth Affiliated Hospital of Sun Yat-Sen University (No: 2021ZSLYEC-176).

We excluded patients with preexisting thyroid disease and autoimmune diseases, previous audiological or otology diseases, ototoxicity deafness, trauma, ear surgery, perforated tympanic membrane, hereditary hearing loss, and central nervous system pathology. Patients with complete laboratory tests of thyroid function (including TSH, free T4, and free T3), thyroid autoantibody (ATPO and TgAb), and pure tone audiometry results were included. Pure tone audiometry results at onset and 4 weeks after treatment were adopted. If multiple pure tone audiometry was performed, the final result will be used to calculate the hearing improvement.

2.2 | Pure tone audiometry

Pure tone audiometry was performed by an Astera Clinical Audiometer (model1066, GN OTOMETRICS, Denmark) with an earphone (TDH 39) and bone-conduction vibrator (B71) in sound-proof booths standard. Pure tone air thresholds were tested at 250, 500, 1000, 2000, 4000, 6000, and 8000 Hz, and bone conduction thresholds were measured at 500, 1000, 2000, and 4000 Hz in each ear. The hearing condition was assessed by the pure tone average (PTA) at 500, 1000, 2000, and 4000 Hz. The hearing threshold of the frequency with no response to the maximum output was calculated according to the air conduction output threshold of the machine's maximum output. The average hearing threshold of the patient with no response at full frequency was 120 dB HL. The maximum output of 125, 250, 500, 1 K, 2 K, 4 K, and 8 K Hz is 80, 100, 115, 120, 120, 120, and 105 dB HL. According to the hearing frequency, the audiogram is divided into the low-frequency region (125, 250, 500, and 1 K Hz) and middle & high frequency region (2 K, 4 K, and 8K Hz), the hearing threshold is calculated separately. Siegel's criteria were used to evaluate the hearing improvement (complete recovery: final PTA better than 25 dB; partial recovery: ≥15 dB gain and final PTA of 25–45 dB; slight recovery: ≥15 dB gain and final PTA < 45 dB; no improvement: patients who showed <15 dB of gain).[26]

2.3 | Laboratory assays

Serum samples were obtained within 24 h after admission and sent to the Laboratory Center of the Sixth Affiliated Hospital of Sun Yat-Sen University for determination of thyroid function (FT3, FT4, and TSH) and thyroid autoantibodies (TPOAb, TgAb), by the Abbott Architect device and chemiluminescence microparticle immunoassay method. The operation of the instrument was strictly under the reagent instructions. Serum TPOAb (reference range 0–5.61 IU/ml, TGAb (reference range 0–4.11 IU/ml), TSH reference range 0.35–4.94 uIU/ml, FT3 (reference range 2.63–5.7 pmol/L), FT4 (reference range 9.01–19.05 pmol/L). Positive was defined as the TPOAb, TGAb levels out of reference range. When the TPOAb level was higher than 1000 IU/ml, it was calculated as 1000 IU/ml.

2.4 | Statistical analysis

The SPSS version 20.0 software package (IBM Corporation, Armonk, NY) was used for statistical analysis. Continuous variable data are expressed as the mean ± SD. Data with normal distribution were compared by t-test, while the Wilcoxon rank test was performed among abnormal distribution data. Categorical data are expressed as

percentages (%). The relationship between parameters was assessed by the χ^2 test. A p value of <0.05 was considered indicative of statistical significance.

3 | RESULTS

Hundred and forty-one cases of sudden deafness were identified, including 105 cases with complete data at the beginning of treatment. There were 48 males and 57 females, with a mean age of 41.86 ± 16.98 years old. Ninety patients had a hearing evaluation at 2 weeks after treatment in our department. The positive incidence of TPOAb, TGAb was 18.1% (19/105) and 21.9% (23/105), respectively. And the indicator of immune thyroiditis positive incidence (at least one abnormal of TPOAb and TGAb) was 24.8% (26/105). According to the abnormity of thyroid autoantibody status, patients were divided into two groups for further study and analysis: thyroid autoantibody-positive and thyroid autoantibody negative. Clinical characteristics are shown in Table 1.

PTA before and after treatment was compared between thyroid autoantibody normal and abnormal. The hearing level was significantly improved in both groups after treatment ($p < 0.001$), but the degree of hearing threshold improvement was similar in both groups ($p = 0.205$) (Table 2).

Taking 1000 Hz as the boundary, the pure tone audiometry threshold of patients was defined as low-frequency hearing (125–1000 Hz) and medium-high frequency hearing (2000–8000 Hz), and further analysis was carried out. The hearing loss of 125–1000 and 2000–8000 Hz is 54.33 ± 29.03 and 63.08 ± 39.52 dB HL, respectively, when patient's thyroid autoantibody is positive. Meanwhile, the hearing loss of 125–1000 Hz and 2000–8000 Hz in patients without thyroid autoantibody is 52.49 ± 26.61 and 55.02 ± 36.39 dB HL, respectively.

Hearing loss of 2000–8000 Hz was worse than 125–1000 Hz of thyroid autoantibody-positive patients; ($p < 0.05$), but the hearing improvement of both groups has no significant difference. Hearing loss of 125–1000 Hz was similar to 2000–8000 Hz in patients without thyroid autoantibody abnormal. ($p > 0.05$). But the 125–1000 Hz

TABLE 1 Clinical features and thyroid profile of patients

Variables	Thyroid autoantibody		p
	Negative	Positive	
N ($n = 105$)	79 (75.2%)	26 (24.8%)	
Age (year old)	42.04 ± 17.69	41.31 ± 14.89	0.837
Sex			
Female	35 (61.4%)	22 (38.6%)	<0.001[a]
Male	44 (91.7%)	4 (8.3%)	
Thyroid features			
TPOAb (IU/ml)	0.67 ± 0.90	171.62 ± 275.71	<0.001[b]
TGAb (IU/ml)	1.09 ± 0.58	118.99 ± 191.40	<0.001[b]
TSH (uIU/ml)	1.05 ± 0.73	1.50 ± 1.93	0.563[b]
FT4 (pmol/L)	12.55 ± 1.83	12.47 ± 1.47	0.834[c]
FT3 (pmol/L)	4.00 ± 0.72	4.0 ± 0.72	0.999[c]
Pure tone audiometry (PTA) (dB HL)	54.99 ± 33.87	60 ± 38.51	0.529[c]
Recovery ($n = 90$)			
Complete recovery ($n = 36$)	28	8	
Partial recovery ($n = 5$)	4	1	
Slight improvement ($n = 8$)	7	6	
No improvement ($n = 36$)	27	9	

[a]χ^2 test. Normal levels: ATPO < 5.61 IU/ml, TGAb < 4.11 IU/ml, TSH 0.35–4.94 uIU/ml, (FT3) 2.63–5.7 pmol/L, FT4 9.01–19.05 pmol/L.
[b]Wilcoxon rank test.
[c]*t* test.

TABLE 2 Comparison of pure tone average between thyroid autoantibody positive and negative patients

Thyroid autoantibody	Before treatment(dB HL)	After treatment(dB HL)	PTA improvement(dB HL)	p
Negative ($n = 66$)	55.84 ± 35.04	44.72 ± 33.85	11.13 + 20.62	<0.001
Positive ($n = 24$)	63.49 ± 38.03	46.01 ± 29.29	17.40 + 20.58	<0.001
p			0.205	

hearing improvement is significantly better than the 2000–8000 Hz hearing threshold ($p < 0.05$). (Figures 1 and 2).

According to the Siegel strategy, the hearing outcomes of all patients after treatment were divided into four groups, complete recovery, partial recovery, slight improvement, no improvement. The characteristics of thyroid function and thyroid autoantibody of each group were shown in Figures 3 and 4. Patients presenting with complete recovery and partial recovery were combined into one group for further analysis. The thyroid function and thyroid autoantibody were compared pair by pair among complete and partial recovery, slight improvement, and no improvement patients. The results showed that the TGAb level of the complete and partial recovery group was significantly higher than that of the slight improvement group ($p < 0.05$), while there was no statistical significance in the other groups ($p > 0.05$).

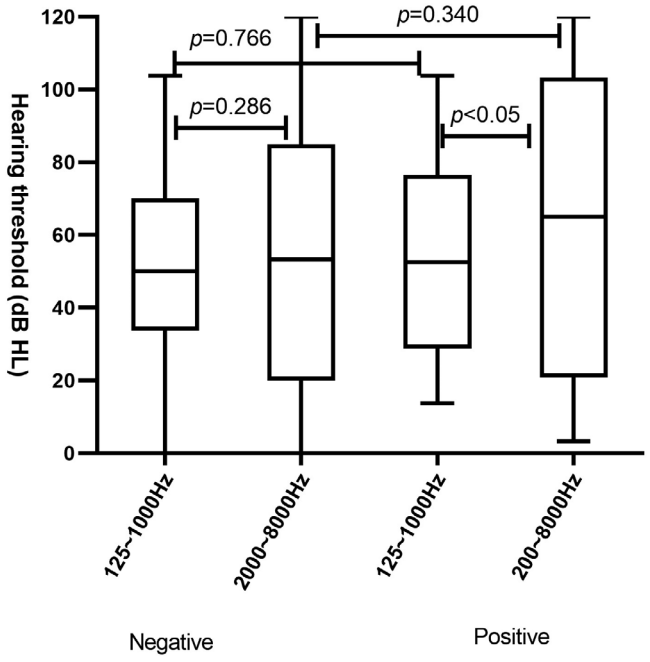

FIGURE 1 Hearing threshold of 125–1000 Hz and 2000–8000 Hz in patients

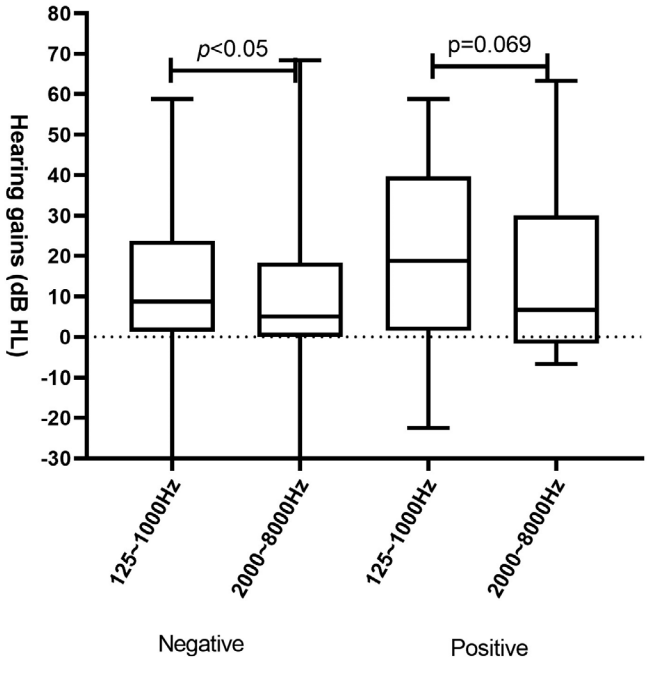

FIGURE 2 The hearing gains of 125–1000 Hz and 2000–8000 Hz after treatment

4 | DISCUSSION

The etiology and pathophysiological mechanism of sudden deafness have not been fully elucidated. Both local and systemic factors may cause sudden deafness. The common causes include vascular disease, virus infection, autoimmune disease, infectious disease, etc. However, the relationship between thyroid autoantibody and sudden deafness has not been determined.

Elevated TPO-Ab can be detected in 5%–20% of euthyroid normal subjects, 74% of Graves' disease patients, and 99.3% of Hashimoto's disease patients. In our study, the thyroid autoantibody abnormality was s 24.8% (TPO-Ab18.1% and TG Ab21.9%, respectively). A retrospective study in Taiwan showed that 5.7% of 3331SSNHL cases had preexisting thyroid diseases including thyroid goiter, thyroiditis, hypothyroidism, and hyperthyroidism, whereas 4.0% of 13,324 controls had thyroid diseases.[27] Our results showed that the detection rate of thyroid autoantibodies in patients with sudden deafness was slightly higher than that in normal subjects reported in the literature. There was some potential association between thyroid autoantibodies and SSNHL.

More and more studies have focused on the population of people with normal thyroid function and immune thyroiditis.[28] In patients with normal thyroid function, the increased TPO-Ab titer usually indicates that patients may be accompanied by hypothyroidism, or they may be in the early stage of thyroid disease. Hypothyroidism has a predictive effect on coronary heart disease. Elevated TPO-Ab levels are associated with increased carotid and intimal media thickness, and coronary artery vessels are impaired in patients with subclinical hypothyroidism.

TPOAb positivity is a major risk factor for oxidative stress in HT patients with normal thyroid function. TPOAb level was an independent influencing factor for insulin resistance (HOMA-IR) and C reactive protein (CRP), independently of thyroid function in nonobese individuals. And mild deviation of thyroid function within the normal range, chronic inflammation, and insulin resistance may be the links between autoimmune thyroiditis (AIT) and atherosclerosis.[29]

Vascular disease and blood supply of the inner ear are closely associated with sudden deafness. The labyrinthine artery is a small branch of the anterior inferior cerebellar artery, which is the terminal vessel for nourishing cochlea without collateral circulation. Hair cells consume large amounts of oxygen and are very sensitive to hypoxia, which can easily lead to hair cell damage.

We compared the PTA between patients with thyroid autoantibody-positive and negative. Patients both gained significant

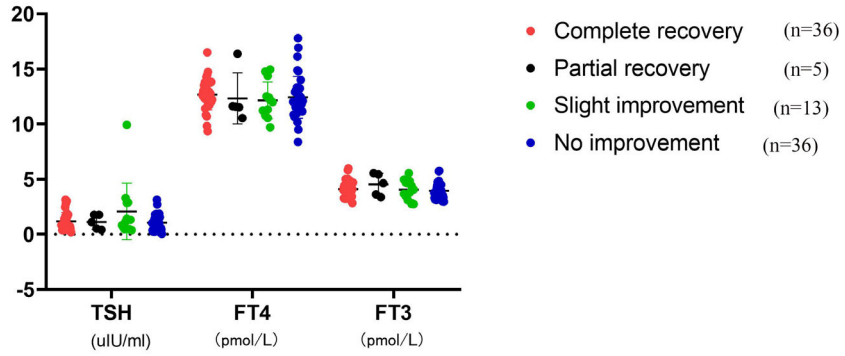

FIGURE 3 Thyroid function in patients with different hearing outcomes

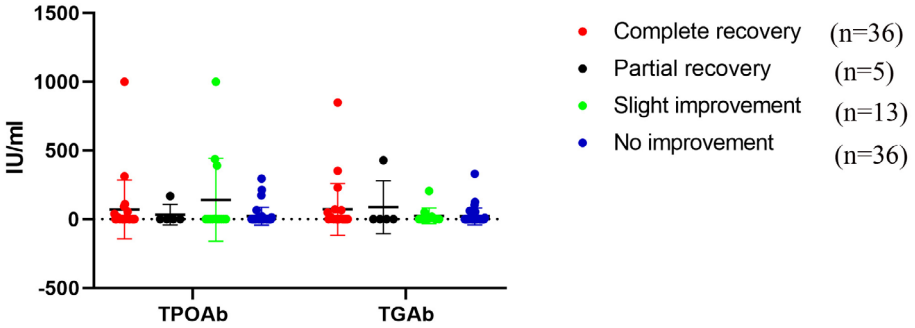

FIGURE 4 Thyroid autoantibody in patients with different hearing outcomes

hearing improvement, and the improvement did not differ in the frequency of 500, 1000, 2000, and 4000 Hz. But when we divide the pure tone audiometry results of patients into low-frequency hearing (125–1000 Hz) and medium-high frequency hearing (2000–8000 Hz) according to frequency. We found that the hearing loss of 2000–8000 Hz was significantly worse than 125–1000 Hz in thyroid autoantibody-positive patients ($p < 0.05$). It suggests that although the final hearing improvement is similar, the hearing loss degree of high frequency and low frequency is not proportional in sudden deafness combined with AIT, and the decrease of high-frequency hearing loss is more serious. However, patients without thyroid autoimmune have asymmetric hearing loss. Interestingly, in our results, only this group of patients showed significantly higher improvement in low-frequency hearing than in high-frequency hearing threshold. This is not consistent with the previous view that immune-related hearing loss is more effective.[8,30,31]

Serum TPOAb levels are used to predict the incidence of AIT in healthy individuals since elevated serum TPOAb levels are usually the first sign of hypothyroidism due to Hashimoto's thyroiditis. Thyroid status is essential for normal organogenesis of the cerebellum, including growth and arborization of the Purkinje neurons, as well as for myelination and synaptogenesis.[32] Thyroid autoantibodies not only act on thyroid tissue but also recognize cerebrovascular system antigens, cortical neurons, cerebellar and cortical astrocytes. The hearing loss associated with Hashimoto's thyroiditis may be a manifestation of Hashimoto's thyroiditis encephalopathy, and the hearing loss has a unique manifestation.

Hashimoto's thyroiditis also can cause brain damage.[33–35] TPOAb can be found in cerebrospinal fluid.[36,37] Previous studies showed that TPOAb in cerebrospinal fluid can be derived from intrathecal synthesis or antibodies in the blood crossing the blood–brain barrier.[36,38–40] Autoimmunity plays an important role in the injury. Autoantibodies can bind to various thyroid antigens (thyroperoxidase, thyroglobulin, and TSH-receptor) and several extra-thyroid antigens (alpha-enolase and other enzymes, gangliosides and MOG-protein, motoneuronal antigens), all of them expressed in the brain.[34] Thyroid antibodies also can result in immune complex deposition and cerebral vasculitis, including the labyrinthine artery.[41] Thyroid autoantibodies induce cytotoxic effects against a common target of thyroid cells and endothelial cells. To our knowledge, there is no previous research on the TPOAb and inner ear. We speculate that thyroid antibodies also can promote the occurrence of SSNHL through direct immune injury and immune-related vasculitis. Thyroid autoantibodies may affect the inner ear, auditory nerve, and even the auditory cortex through the above pathological damage, resulting in sudden sensory hearing loss and gradual hearing loss.

Although according to the current efficacy evaluation rules, there is no significant difference between our two groups of patients after steroid-based treatment. But the audiological features differed in another aspect. It is suggested that patients with thyroid autoimmune states may have other pathogenesis factors and clinical audiology characteristics. It is necessary to further study the relationship between thyroid autoantibodies and inner ear antigens, auditory nerve antigens, auditory cortex antigens.

5 | CONCLUSION

Based on our data we speculate that a potential association between thyroid autoimmunity and sudden sensorineural hearing loss. SSNHL

patients with thyroid autoimmunity positive have unique clinical audiological characteristics. Thyroid autoimmunity may be a pathogenesis factor of SSNHL and is associated with more severe high-frequency hearing loss.

Clinicians not only audiologists but also internists should pay attention to the possibility of the cochlea and auditory nerve injury associated with thyroid disorders. Due to the relatively few clinical studies on thyroid antibodies and SSNHL, immunotherapy lacks relevant evidence-based knowledge, and large-scale clinical studies are still needed in the future.

CONFLICT OF INTEREST

All the authors listed have approved the manuscript that is enclosed. The authors declare no conflict of interest.

AUTHOR CONTRIBUTIONS

Xiao-Mei Sun was a major contributor in writing the manuscript. Shi-Min Zhuang contributed to write parts of the manuscript. Zhi-Wen Xiao collected the data of the patients. Jia-Qi Luo checked the figure and images. Zhen Long assisted in the operation. Lin-Chan Lan performed the clinical examination. Hui-Qing Zhang prepared the materials. Guan-Ping Zhang designed the study. All authors read and approved the final manuscript.

ETHICS STATEMENT

Our retrospective study was approved by the Ethics Committee of the Sixth Affiliated Hospital of Sun Yat-Sen University (No: 2021ZSLYEC-176).

ORCID

Xiao-Mei Sun https://orcid.org/0000-0002-6383-2678

REFERENCES

1. Marx M, Younes E, Chandrasekhar SS, et al. International consensus (ICON) on treatment of sudden sensorineural hearing loss. *Eur Ann Otorhinolaryngol Head Neck Dis*. 2018;135:S23-s28.
2. Sato H, Kuwashima S, Nishio SY, et al. Epidemiological survey of acute low-tone sensorineural hearing loss. *Acta Otolaryngol*. 2017;137:S34-s37.
3. Nakashima T, Sato H, Gyo K, et al. Idiopathic sudden sensorineural hearing loss in Japan. *Acta Otolaryngol*. 2014;134:1158-1163.
4. Stachler RJ, Chandrasekhar SS, Archer SM, et al. Clinical practice guideline: sudden hearing loss. *Otolaryngol Head Neck Surg*. 2012;146:S1-S35.
5. Chandrasekhar SS, Tsai Do BS, Schwartz SR, et al. Clinical practice guideline: sudden hearing loss (update). *Otolaryngol Head Neck Surg*. 2019;161:S1-s45.
6. Saunders WH, Lippy WH. Sudden deafness and Bell's palsy: a common cause. *Ann Otol Rhinol Laryngol*. 1959;68:830-837.
7. Schuknecht HF, Kimura RS, Naufal PM. The pathology of sudden deafness. *Acta Otolaryngol*. 1973;76:75-97.
8. Li G, You D, Ma J, Li W, Li H, Sun S. The role of autoimmunity in the pathogenesis of sudden sensorineural hearing loss. *Neural Plast*. 2018;2018:7691473.
9. Jenkins HA, Pollak AM, Fisch U. Polyarteritis nodosa as a cause of sudden deafness. A human temporal bone study. *Am J Otolaryngol*. 1981;2:99-107.
10. Thornton AR, Jarvis SJ. Auditory brainstem response findings in hypothyroid and hyperthyroid disease. *Clin Neurophysiol*. 2008;119:786-790.
11. Di Lorenzo L, Foggia L, Panza N, et al. Auditory brainstem responses in thyroid diseases before and after therapy. *Horm Res*. 1995;43:200-205.
12. Rüsch A, Erway LC, Oliver D, Vennström B, Forrest D. Thyroid hormone receptor beta-dependent expression of a potassium conductance in inner hair cells at the onset of hearing. *Proc Natl Acad Sci U S A*. 1998;95:15758-15762.
13. Knipper M, Bandtlow C, Gestwa L, et al. Thyroid hormone affects Schwann cell and oligodendrocyte gene expression at the glial transition zone of the VIIIth nerve prior to cochlea function. *Development*. 1998;125:3709-3718.
14. Lautermann J, ten Cate WJ. Postnatal expression of the alpha-thyroid hormone receptor in the rat cochlea. *Hear Res*. 1997;107:23-28.
15. Campos-Barros A, Amma LL, Faris JS, Shailam R, Kelley MW, Forrest D. Type 2 iodothyronine deiodinase expression in the cochlea before the onset of hearing. *Proc Natl Acad Sci U S A*. 2000;97:1287-1292.
16. Knipper M, Gestwa L, ten Cate WJ, et al. Distinct thyroid hormone-dependent expression of TrKB and p75NGFR in nonneuronal cells during the critical TH-dependent period of the cochlea. *J Neurobiol*. 1999;38:338-356.
17. Santos KT, Dias NH, Mazeto GM, Carvalho LR, Lapate RL, Martins RH. Audiologic evaluation in patients with acquired hypothyroidism. *Braz J Otorhinolaryngol*. 2010;76:478-484.
18. Uziel A, Marot M, Rabie A. Corrective effects of thyroxine on cochlear abnormalities induced by congenital hypothyroidism in the rat. II Electrophysiological study. *Brain Res*. 1985;351:123-127.
19. Berker D, Karabulut H, Isik S, et al. Evaluation of hearing loss in patients with Graves' disease. *Endocrine*. 2012;41:116-121.
20. Ralli M, Angeletti D, Fiore M, et al. Hashimoto's thyroiditis: an update on pathogenic mechanisms, diagnostic protocols, therapeutic strategies, and potential malignant transformation. *Autoimmun Rev*. 2020;19:102649.
21. McLachlan SM, Rapoport B. Why measure thyroglobulin autoantibodies rather than thyroid peroxidase autoantibodies? *Thyroid*. 2004;14:510-520.
22. McLachlan SM, Rapoport B. Autoimmune response to the thyroid in humans: thyroid peroxidase—the common autoantigenic denominator. *Int Rev Immunol*. 2000;19:587-618.
23. Yoshida H, Amino N, Yagawa K, et al. Association of serum antithyroid antibodies with lymphocytic infiltration of the thyroid gland: studies of seventy autopsied cases. *J Clin Endocrinol Metab*. 1978;46:859-862.
24. Nacci A, Dallan I, Monzani F, et al. Elevated antithyroid peroxidase and antinuclear autoantibody titers in Ménière's disease patients: more than a chance association? *Audiol Neurootol*. 2010;15:1-6.
25. Chiarella G, Russo D, Monzani F, et al. Hashimoto thyroiditis and vestibular dysfunction. *Endocr Pract*. 2017;23:863-868.
26. Siegel LG. The treatment of idiopathic sudden sensorineural hearing loss. *Otolaryngol Clin North Am*. 1975;8:467-473.
27. Tsai YT, Chang IJ, Hsu CM, et al. Association between sudden sensorineural hearing loss and preexisting thyroid diseases: a nationwide case-control study in Taiwan. *Int J Environ Res Public Health*. 2020;17:834.
28. Garin MC, Arnold AM, Lee JS, Tracy RP, Cappola AR. Subclinical hypothyroidism, weight change, and body composition in the elderly: the cardiovascular health study. *J Clin Endocrinol Metab*. 2014;99:1220-1226.
29. Liu J, Duan Y, Fu J, Wang G. Association between thyroid hormones, thyroid antibodies, and cardiometabolic factors in non-obese individuals with normal thyroid function. *Front Endocrinol (Lausanne)*. 2018;9:130.

30. Yazdani N, Kakavand Hamidi A, Ghazavi H, et al. Association between macrophage migration inhibitory factor gene variation and response to glucocorticoid treatment in sudden sensorineural hearing loss. *Audiol Neurootol*. 2015;20:376-382.
31. García-Berrocal JR, Ramírez-Camacho R, Millán I, et al. Sudden presentation of immune-mediated inner ear disease: characterization and acceptance of a cochleovestibular dysfunction. *J Laryngol Otol*. 2003; 117:775-779.
32. Manto M, Hampe CS. Endocrine disorders and the cerebellum: from neurodevelopmental injury to late-onset ataxia. *Handb Clin Neurol*. 2018;155:353-368.
33. Ferracci F, Bertiato G, Moretto G. Hashimoto's encephalopathy: epidemiologic data and pathogenetic considerations. *J Neurol Sci*. 2004; 217:165-168.
34. Churilov LP, Sobolevskaia PA, Stroev YI. Thyroid gland and brain: enigma of Hashimoto's encephalopathy. *Best Pract Res Clin Endocrinol Metab*. 2019;33:101364.
35. Yoneda M. Hashimoto's encephalopathy and autoantibodies. *Brain Nerve*. 2018;70:305-314.
36. Ferracci F, Moretto G, Candeago RM, et al. Antithyroid antibodies in the CSF: their role in the pathogenesis of Hashimoto's encephalopathy. *Neurology*. 2003;60:712-714.
37. Chang JS, Chang TC. Hashimoto's encephalopathy: report of three cases. *J Formos Med Assoc*. 2014;113:862-866.
38. Dersch R, Tebartz van Elst L, Hochstuhl B, et al. Anti-thyroid peroxidase and anti-thyroglobulin autoantibodies in the cerebrospinal fluid of patients with unipolar depression. *J Clin Med*. 2020;9(8):2391.
39. Azuma T, Uemichi T, Funauchi M, Doi S, Matsubara T. Myelopathy associated with Hashimoto's disease. *J Neurol Neurosurg Psychiatry*. 2000;68:681-682.
40. Endres D, Dersch R, Hochstuhl B, et al. Intrathecal thyroid autoantibody synthesis in a subgroup of patients with schizophreniform syndromes. *J Neuropsychiatry Clin Neurosci*. 2017;29:365-374.
41. Shibata N, Yamamoto Y, Sunami N, Suga M, Yamashita Y. Isolated angiitis of the CNS associated with Hashimoto's disease. *Rinsho Shinkeigaku*. 1992;32:191-198.

How to cite this article: Sun X-M, Zhuang S-M, Xiao Z-W, et al. Autoimmune thyroiditis in patients with sudden sensorineural hearing loss. *Laryngoscope Investigative Otolaryngology*. 2022;7(2):571-577. doi:10.1002/lio2.755