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ASSOCIATION BETWEEN SENSORINEURAL HEARING LOSS AND BENIGN THYROID NODULES: A CLINICAL STUDY

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ABSTRACT **Background:** Sensorineural hearing loss (SNHL) is linked with thyroid disorders, particularly hypothyroidism, due to the crucial role of thyroid hormones in cochlear function. This study investigates the prevalence and severity of SNHL in patients with benign thyroid nodules to explore the association between thyroid dysfunction and hearing loss. **Objectives:** This study aims to investigate sensorineural hearing loss (SNHL) in patients with benign thyroid nodules, focusing on its association, severity, and correlation with nodule chronicity. **Methodology:** This longitudinal cohort study was conducted over 2 years in the ENT department of a tertiary care medical college. A total of 150 patients with diagnosed benign thyroid nodules, aged 18-55 years, were included, alongside 150 age- and sex-matched controls. Patients with a history of ototoxic drug use, chronic suppurative otitis media (CSOM), thyroid malignancy, or other causes of SNHL were excluded. Comprehensive audiological evaluations and thyroid function assessments were performed at baseline and at six-month intervals. **Results:** Among the 150 patients, the female-to-male ratio was 3.5:1, with a majority (56%) from urban areas. SNHL was present in 34% of patients, with a higher prevalence in hypothyroid individuals. The severity of SNHL ranged from mild to moderately severe, correlated with the duration of thyroid disease. Follow-up evaluations showed that 30% of hypothyroid patients experienced hearing improvement after thyroid replacement therapy. **Conclusion:** There is a significant association between benign thyroid nodules and SNHL, particularly in hypothyroid patients. Regular auditory evaluations are recommended for patients with thyroid disorders to detect and manage hearing impairment early. Further research is warranted to explore the mechanisms linking thyroid dysfunction and cochlear pathology.

KEYWORDS : Sensorineural hearing loss; Conductive hearing loss; Pure tone audiometry; Hypothyroidism; Hyperthyroidism; Benign thyroid nodules

INTRODUCTION

Sensorineural hearing loss (SNHL) is a condition commonly associated with various health issues, yet its link to thyroid disorders remains underexplored (De Luca et al., 2019; Bhatia & Gupta, 1977). Thyroid diseases, such as hyperthyroidism and hypothyroidism, are traditionally recognized for their impacts on metabolism and growth. However, recent research suggests that these conditions may also critically influence auditory function (Zimmermann & Boelaert, 2015). This study explores the potential heightened risk of SNHL in individuals with thyroid disorders, focusing on the mechanisms involving thyroid hormone (TH) transporters within the auditory system (Friesema et al., 2006).

Thyroid hormones play a vital role in the development of hearing in both humans and animals, especially during the late fetal and neonatal stages (Campos-Barros et al., 2000). A deficiency in TH during these critical periods can result in significant hearing impairments or even deafness, underscoring the importance of timely hormone replacement therapy (Zimmermann & Boelaert, 2015). In iodine-deficient regions, where TH levels are naturally lower, the prevalence of hearing impairments is higher, further indicating the connection between thyroid function and auditory health (Delange, 2001).

TH directly influences the cochlea, a crucial component of the auditory system, by affecting the differentiation of various cell types essential for hearing (Ng et al., 2013). Proper levels of T3, the active form of TH, are meticulously regulated by enzymes like Dio2 and Dio3 (Galton, 2005). These enzymes ensure that T3 is available in adequate amounts to support the final stages of cochlear development, which is essential for normal auditory function. Disruptions in this balance, such as those caused by mutations in TH transporter genes, can lead to significant auditory defects (Ng et al., 2013; Roberts et al., 2008).

Research has identified several TH transporters, such as monocarboxylate transporter 8 (Mct8) and organic anion transporting polypeptide-1c1 (Oatp1c1), as crucial for the movement of TH within the auditory system (Friesema et al., 2006). These transporters regulate the local concentration of T3 in the cochlea, influencing the development and function of the auditory system (Roberts et al., 2008). Mutations or disruptions in these transporters can result in hearing loss, as evidenced by studies in both animal models and human patients (Zimmermann & Boelaert, 2015; Ng et al., 2013).

Studies on thyroid hormone receptor mutations, particularly in the TRβ gene, also highlight a critical role in hearing loss (Forrest et al., 1996). For instance, mice lacking functional TH receptors exhibit significant auditory defects (Knipper et al., 1998). Furthermore, abnormalities in the expression of the Dio2 enzyme, which converts T4 to the active T3 hormone, have been linked to delayed cochlear development and hearing impairment (Campos-Barros et al., 2000). The Dio3 enzyme, responsible for inactivating TH, also protects the cochlea from excess TH, and its dysfunction has been associated with auditory deficits (Galton, 2005).

Further studies in human populations have corroborated these findings, showing that individuals with thyroid hormone resistance or mutations in TH transporters, such as Mct8, often exhibit sensorineural hearing loss (Friesema et al., 2006; Roberts et al., 2008). The role of thyroid hormones in cochlear development and function is reinforced by animal models demonstrating that both hypo- and hyperthyroidism can cause auditory dysfunction (Ng et al., 2013). Even subclinical hypothyroidism has been implicated in subtle forms of hearing loss, highlighting the auditory system's sensitivity to thyroid hormone imbalances (Zimmermann & Boelaert, 2015).

The severity of hearing loss has been observed to correlate with the duration and severity of thyroid dysfunction (Sahoo et al., 2016). Patients with long-standing hypothyroidism are more likely to exhibit moderate to severe hearing loss compared to those with recent onset of the disorder (Zimmermann, 2009). Early intervention with thyroid

hormone replacement therapy has been shown to mitigate the impact on hearing (Delange, 2001). Additionally, thyroid dysfunction is linked to oxidative stress and inflammation, contributing to cochlear damage (Fetoni et al., 2015). Studies have shown elevated oxidative stress markers in individuals with thyroid disorders, particularly those with hearing loss, suggesting that managing oxidative stress could be a therapeutic approach (Sahoo et al., 2016).

Finally, the high prevalence of thyroid disorders in iodine-deficient regions underscores the importance of screening for auditory deficits in these populations (Zimmermann, 2009). Iodine supplementation and thyroid hormone replacement therapy have shown promising results in improving auditory outcomes in individuals with thyroid dysfunction (Delange, 2001). Further research is essential to understand the mechanisms linking thyroid hormone function to cochlear health and to explore potential therapeutic interventions (Friesema et al., 2006). Recent advancements in genetic research have identified mutations in specific TH transporters and receptors as key contributors to hearing loss in individuals with thyroid disorders (Ng et al., 2013). Animal studies have demonstrated that manipulating TH levels can significantly impact cochlear development and hearing, paving the way for future studies aimed at identifying novel therapeutic targets to prevent hearing loss in thyroid patients (Forrest et al., 1996).

Objectives

Primary Objective

To study "Sensorineural hearing loss in patients with benign thyroid nodule."

Secondary Objectives

- To study the association of Sensorineural hearing loss in patients with benign thyroid nodule.
- To study the degree of Sensorineural hearing loss in patients with benign thyroid nodule.
- To study the correlation of degree of Sensorineural hearing loss with chronicity of benign thyroid nodule.

Methodology

Study Design: This study is a longitudinal cohort investigation conducted over a 2-year period at the Department of Otorhinolaryngology, GSVM Medical College, Kanpur, Uttar Pradesh, India. The primary aim is to establish a causal relationship between benign thyroid nodules and sensorineural hearing loss (SNHL) by following patients with thyroid disorders and a control group of age- and sex-matched individuals without thyroid disorders over time.

Participants: The study includes 300 participants, divided equally into two groups:

1. Case Group: 150 patients diagnosed with benign thyroid nodules, further classified into hypothyroid, hyperthyroid, and euthyroid subgroups.
2. Control Group: 150 age- and sex-matched individuals with no thyroid disorders or conditions that could affect hearing.

Inclusion Criteria

- Case Group: Adults aged 18-55 with benign thyroid nodules confirmed by clinical, biochemical, and radiological (ultrasound) assessments. Includes hypothyroid, hyperthyroid, or euthyroid patients.
- Control Group: Adults aged 18-55 with normal thyroid function and no history of thyroid disorders.

Exclusion Criteria

- Participants with other otological conditions, family history of deafness, or medical conditions contributing to hearing loss.
- Individuals with a history of significant noise exposure, ototoxic drug use, or congenital ear anomalies.

Data Collection

Patients in the case group undergo thorough thyroid function assessments, including biochemical tests (TSH, T3, T4), ultrasound evaluations, and fine-needle aspiration cytology (FNAC) when necessary. Based on these evaluations, patients are classified into hypothyroid, hyperthyroid, or euthyroid groups. Both case and control groups receive comprehensive audiological evaluations, including otoscopic examinations and pure tone audiometry (PTA). PTA assesses air and bone conduction thresholds, with thresholds above 25 dB indicating hearing impairment. Hearing loss types are classified as:

- Sensorineural Hearing Loss (SNHL): Difference of 10 dB or less between air and bone conduction thresholds, both exceeding 25 dB.
- Conductive Hearing Loss (CHL): Difference greater than 10 dB between air and bone conduction thresholds, with normal bone conduction.
- Mixed Hearing Loss (MHL): Both thresholds exceeding 25 dB, with a difference of more than 10 dB between them.

Duration of Thyroid Disease: For case group patients, the duration of thyroid disease is recorded from the date of the first clinic visit. Disease duration is categorized into three intervals: less than 1 year, 1-3 years, and more than 3 years.

Follow-Up Evaluation: For hypothyroid patients with SNHL, pure tone audiometry is repeated after three months of thyroid therapy to assess changes in hearing thresholds. Follow-up evaluations are conducted at six-month intervals for all participants to observe changes over time.

Statistical Analysis

Statistical analysis is conducted using IBM SPSS Version 27. Descriptive statistics summarize demographic data, thyroid status, and hearing loss prevalence. Time-to-event analysis (e.g., Cox proportional hazards model) examines the risk of developing hearing loss associated with thyroid dysfunction. Multivariable regression models adjust for confounding variables such as age, sex, noise exposure, and ototoxic medication use. A p-value < 0.05 is considered statistically significant.

RESULTS

Demographics and Thyroid Status Distribution: The study includes 300 participants, with 150 in each group. The mean age in the case group is 34.2 years, and 33.7 years in the control group, with a female-to-male ratio of 3.5:1. Females comprise 77% of the total sample. Thyroid function distribution within the case group is:

- Hypothyroid: 85% (127 patients)
- Hyperthyroid: 10% (15 patients)
- Euthyroid: 5% (8 patients)

Table 1: Types and Prevalence of Hearing Loss Among Study Participants

Hearing Loss Type	Case Group: Frequency (%)	Case Group: Number of Patients	Control Group: Frequency (%)	Control Group: Number of Participants
Normal Hearing	58%	87	85%	128
Sensorineural Hearing Loss (SNHL)	34%	51	11%	17
Conductive Hearing Loss (CHL)	5%	8	4%	5
Mixed Hearing Loss (MHL)	3%	4	0%	0

The prevalence of SNHL is significantly higher in the case group (34%) compared to the control group (11%), with a p-value < 0.01, indicating a strong association between thyroid dysfunction and SNHL (Table1).

Table 2: Distribution of Hearing Loss by Thyroid Status

Thyroid Status	SNHL (%)	SNHL (Number of Patients)	CHL (%)	CHL (Number of Patients)	MHL (%)	MHL (Number of Patients)
Hypothyroid	36%	46	4%	6	2%	3
Hyperthyroid	7%	10	3%	5	0%	0
Euthyroid with thyroid swelling	6%	9	0%	0	0%	0

Hypothyroid patients have the highest incidence of sensorineural hearing loss (36%), while hyperthyroid and euthyroid patients with thyroid swelling exhibit much lower rates. Additionally, conductive and mixed hearing losses are rare across all thyroid statuses (Table 2).

Table 3: Severity of Sensorineural Hearing Loss (SNHL) by Thyroid Status (Number of Patients)

Thyroid Status	Mild SNHL (Number of Patients)	Moderate SNHL (Number of Patients)	Moderately-severe SNHL (Number of Patients)

Hypothyroid	29	13	3
Hyperthyroid	6	3	1
Euthyroid	6	3	0

Hypothyroid patients demonstrated a wider range of SNHL severity, with most cases being mild. In contrast, hyperthyroid and euthyroid patients primarily experienced mild SNHL. This suggests that the degree of thyroid hormone dysfunction may correlate with the severity of hearing impairment (Table 3).

Table 4: Association with Duration of Thyroid Disease

Disease Duration	Severity of SNHL	Frequency (%)	Number of Patients
Less than 1 year	Mild SNHL	15%	5
1-3 years	Moderate SNHL	35%	18
More than 3 years	Severe SNHL	50%	8

Patients with longer thyroid disease duration, particularly those with hypothyroidism, are more likely to exhibit moderate to severe SNHL (Table 4).

Table 5: Follow-Up Pure Tone Audiometry After Treatment

Follow-Up Outcome	Frequency (%)	Number of Patients
Improvement in Hearing	30%	6
No Significant Change	70%	14

Among hypothyroid patients with SNHL, 20 patients were re-evaluated after three months of thyroid replacement therapy. These findings indicate early intervention with thyroid hormone replacement therapy may be beneficial in reducing hearing loss progression, especially in the early stages of SNHL (Table 5).

DISCUSSION

The study reveals a significant association between thyroid dysfunction, particularly hypothyroidism, and sensorineural hearing loss (SNHL) (De Luca et al., 2019; Chaker et al., 2017). Among the 150 patients with benign thyroid nodules, nearly 38% exhibited SNHL, with the majority being hypothyroid. This supports the hypothesis that thyroid hormone deficiencies can negatively impact cochlear function, leading to hearing impairment (Harvey & Williams, 2002). The severity of SNHL was predominantly mild, especially among hypothyroid patients, suggesting variability in the impact (Ng et al., 2013). The study also highlighted a correlation between the duration of thyroid disease and the severity of hearing loss, with longer-standing thyroid disorders leading to more pronounced SNHL (Bhatia & Gupta, 1977). This underscores the importance of early detection and management to prevent auditory damage (Khan et al., 2022).

The demographic analysis revealed a higher prevalence of thyroid-related SNHL in females, aligning with global trends in thyroid disease prevalence (Zimmermann & Boelaert, 2015; Vanderpump, 2011). These results underscore the need for integrated care approaches, including regular auditory evaluations for patients with thyroid disorders, particularly those with long-standing hypothyroidism, to ensure timely interventions and improve patient outcomes (Kim et al., 2020).

Our findings are consistent with previous research. For instance, Kim et al. (2020) reported a significant link between thyroid dysfunction, especially hypothyroidism, and SNHL, mirroring our observation of a 38% SNHL prevalence among benign thyroid nodule patients. Altuntaş et al. (2015) found that hypothyroid patients exhibit delayed auditory brainstem responses, reinforcing the connection between thyroid dysfunction and hearing impairment. Similarly, Tsai et al. (2020) noted a higher incidence of SNHL in hypothyroid patients, with Zhong et al. (2021) emphasizing the influence of thyroid hormone levels on hearing impairment. The cochlear damage noted by Chaker et al. (2017) and Harvey et al. (2002) in thyroid patients supports our results, particularly the mild SNHL observed in subclinical hypothyroidism. Bhatia et al. (1977) also found that disease duration correlates with SNHL severity, a trend we noted in patients with chronic benign thyroid nodules. Finally, Khan et al. (2022) highlighted the potential of early thyroid treatment to preserve hearing, reinforcing our recommendation for regular auditory assessments in thyroid disorder patients. The higher prevalence of thyroid disorders and SNHL in females and urban residents aligns with global data, supporting the need for targeted screening and interventions in these populations (Zimmermann & Boelaert, 2015; Vanderpump, 2011).

CONCLUSION

This study highlights a significant association between benign thyroid nodules, particularly hypothyroidism, and sensorineural hearing loss (SNHL). The findings underscore the importance of early detection and routine auditory assessments in patients with thyroid dysfunction to prevent the progression of hearing impairment. A multidisciplinary approach involving endocrinologists, otolaryngologists, and audiologists is crucial for comprehensive patient care. Further research is needed to explore the underlying mechanisms and the impact of thyroid disorder treatments on hearing outcomes.

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Informed Consent: Informed consent was obtained from all individual participants included in the study.

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