Original Research Article

Assessment of audiologic evaluation in patients with acquired hypothyroidism

P. V. Balaji[1*], M. Thirumaran[2], V. Sharath babu[3]

[1]Professor, [2]Associate Professor, [3]Assistant Professor
Department of Physiology, Vinayaka Mission Medical College, Karikal, India
[*]Corresponding author email: **drbalaji.vmcc@gmail.com**

International Archives of Integrated Medicine, Vol. 3, Issue 7, July, 2016.

Available online at http://iaimjournal.com/
ISSN: 2394-0026 (P) **ISSN: 2394-0034 (O)**
Received on: 27-06-2016 **Accepted on:** 06-07-2016
Source of support: Nil **Conflict of interest:** None declared.
How to cite this article: P. V. Balaji, M. Thirumaran, V. Sharath babu. Assessment of audiologic evaluation in patients with acquired hypothyroidism. IAIM, 2016; 3(7): 222-227.

Abstract

Introduction: Hypothyroidism is a common endocrine disorder resulting from deficiency of thyroid hormone or, more rarely, from their impaired activity at tissue level. In its clinically overt form, hypothyroidism is a relatively common condition, with an approximate prevalence of 2% in adult women and 0.2% in adult men. Deficiency of the hormone has a wide range of effects, because all metabolically active cells require thyroid hormone.

Aim of the study: To determine the incidence of deafness in patients suffering from goiterous hypothyroidism exclusively and to evaluate the role of L-thyroxine therapy in improving the hearing in this group of patients.

Materials and methods: Two groups were included: a hypothyroidism group (HG, n-30), and a control group (CG, n-30). Parameters studied: gender, time of hypothyroidism, co-morbidities, cochleovestibular symptoms, biochemistry and hormonal exams (TSH, T4), tonal audiometry, TOAEs and BERA.

Results: All participants were women, 70% of the HG had Hashimoto thyroiditis, and 60% of the HG had had the diagnostic of the hypothyroidism for at least five years. Depression and hypertension were frequent in HG. All HG patients had altered TSH values and 50% had diminished T4 values. Sensorineural hearing loss was detected in 22 ears from the HG and in seven from the CG. BERA was normal in the CG and altered in 10 ears from the HG, showing L-V increase. TOAEs were absent in 12 ears from the HG and in four from the CG.

Conclusions: HG patients had more cochleovestibular symptoms, higher audiometric thresholds, and increase in L-V in the BERA and absence or reduction in TOAEs amplitudes. Such alterations were not associated with THS and free T4 levels.

Key words

Hypothyroidism, Thyroid gland, Deficiency, Sensorineural hearing loss.

Introduction

Hypothyroidism is a common endocrine disorder resulting from deficiency of thyroid hormone or, more rarely, from their impaired activity at tissue level. Pre-valence is 1.9% in women and it increases with age. It may be a primary process in which the thyroid gland produces insufficient amounts of thyroid hormone (e.g. autoimmune thyroiditis, previous radio-iodine or surgical treatment of hyperthyroidism), but can also be secondary, that is, lack of thyroid hormone secretion due to inadequate secretion of either thyrotropin {that is, thyroid-stimulating hormone (TSH)} from the pituitary gland or thyrotropin-releasing hormone (TRH) from the hypo-thalamus (secondary or tertiary hypothyroidism). Hearing loss was first reported in acquired hypothyroid- ism in 1907 [1]. Over a period of time, a distinct associa- tion between hypothyroidism and auditory system dysfunction has been reported in medical text though there are studies which have failed to elucidate a definitive relationship between hypothyroidism and deafness [2]. The medical literature now mentions vertigo, hearing loss, tinnitus and pruritic external auditory canal as important vestibular and audiological symptoms of hypothyroidism .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 is still uncertain. This is attributed to the marked paucity of literature on the cited subject. The incidence of hearing loss varies from 25% to 50% with a higher incidence in congenital hypothyroidism. Moreover, the results of audiological evaluation f patients with hypothyroidism under treatment with L-Thy-roxine (LT) are conflicting. Approximately 30% of genetic hearing losses are part of a syndrome, where there is the presence of distinctive associated clinical conditions [3]. Pendred syndrome is the commonest form of syndromic deafness. It typically involves enlargement of the vestibular aqueduct as well as thyroid problems. The hearing loss is usually profound and bilateral, but is not always present at birth. Onset can occur during infancy and early childhood, and the hearing loss can rapidly progress. Waardenburg syndrome can cause bilateral or unilateral hearing loss of variable severity [4]. The syndrome typically involves pigmentation abnormalities and gastrointestinal symptoms. Only 20-50% of individuals with Waardenburg syndrome will have hearing loss.

Usher syndrome involves deafness and retinitis pigmentosa, a progressive degeneration of the retina that leads, eventually, to blindness. In Usher syndrome Type 1 there is a profound congenital hearing loss and a relatively early onset of retinitis pigmentosa, often during the first decade of life [5]. Type 1 affects around 40% of all individuals with Usher syndrome. Usher syndrome Type 2, affecting about 57%, involves a less severe hearing loss and a later onset of retinitis pigmentosa. Type 3, affecting around 3%, has a variable severity of hearing loss which can be progressive. CHARGE syndrome involves cochlear and semicircular canal anomalies. Other characteristics can include congenital heart defects, kidney problems, cleft lip or palate, and growth retardation. The hearing loss can be sensorineural, conductive, or mixed. CHARGE syndrome is rare, estimated to affect about 1 in 10,000 live births [6]. There are many other syndromes that can involve permanent hearing loss, including Branchio-oto-renal (BOR) syndrome, Jervell, Lang-Neilsen syndrome (JLNS), Down syndrome, and Apart syndrome. Auditory acuity reduction has been associated with thyroid gland dysfunction and has been described by numerous authors [7]. In 1974, Ritter stressed that hearing loss can be the most common otorhinolaryngological manifestation of congenital and acquired hypothyroidism, and auditory symptoms may happen alone or in

association with vertigo and tinnitus. The real incidence of hearing loss in patients with hypothyroidism is still uncertain, and it may affect 25% of the patients with acquired hypothyroidism and 35-50% of the patients with congenital hypothyroidism [8]. The pathophysiological mechanisms of hearing loss in hypothyroidism are not totally unveiled. It is known that in this hormonal disorder there is a reduction in cell energy production, compromising the microcirculation and, consequently, oxygenation and the metabolism of the involved organs. Inner ear structures are also affected, such as the stria vascularis and the Organ of Corti [9]. Thyroid hormones control protein synthesis, the production of myelin and enzymes and the level of lipids in the central nervous system. Moreover, T4 can act as a neurotransmitter. Thus, it is believed that under hypothyroidism, hearing impairment can originate in the cochlea, in the central auditory pathways and/or in the retrocochlear region.

Materials and methods

The patients whose diagnoses were confirmed were categorised using following factors: age, sex, previous history, comorbid conditions present, imaging details, preoperative calcium levels after getting the proper informed consent. We set up two study groups: a group with hypothyroidism (GH), made up of 30 patients with acquired hypothyroidism, confirmed by clinical and laboratory exams, and the control group (GC), made up of 30 volunteers without thyroid gland disease, belonging to the corresponding age range, submitted to the same evaluation sequence used in the study group. The following exclusion criteria were used: be older than 60 years, having been submitted to prior ear surgery, having an altered otoscopic exam, work in a noisy environment or having an audiometric exam with a result matching that of noise-induced hearing loss, having a conductive hearing loss or type B or C tympanometric curve, report the use of ototoxic medication, report prior history of hereditary hearing loss, report hearing loss since childhood or having a genetic syndrome. The audiometric results were classified as to type and degree. As far as type is concerned, the losses were classified in: conductive (airway thresholds above 25 dB and the normal bone conduction threshold, with an air-bone gap); mixed (air and bone conduction thresholds above 25 dB, with air-bone gap) and sensorineural (air and bone conduction thresholds above 25 dB, without air-bone gap). The patients with conductive or mixed hearing loss were taken off the study. Concerning the grade, the hearing losses were classified into: mild (thresholds between 26 and 40 dB); moderate (thresholds between 41 and 70 dB); severe (thresholds between 71 and 90 dB) and profound (thresholds above 91 dB). Our statistical methods to study the associations between the variables and comparison of proportions between the groups were the chi-square test, the Fisher's exact test and the Goodman test. In comparing the attributes with normal distribution, we used the t Student test, considering 5% as level of significance.

Results

Age distribution of cases was as per **Table – 1**. Hearing loss with correlation with mean T4 level was as per **Table – 2**. Response to L-thyroxine treatment (frequency specific/each ear) frequency was as per **Table – 3**.

Discussion

Goitre is defined as an enlargement of thyroid gland. Worldwide about 90% of cases are due to iodine deficiency. In countries that use iodized salt, Hashimoto's thyroiditis is the most common cause. Further it is important to note that over a period of time, patients of goitre due to lack of iodine can develop hypothyroidism. Hence, the evaluation of hearing in goitrous hypothyroidism assumes clinical importance. A subgroup of children with permanent hearing loss has auditory neuropathy or auditory dyssynchrony, commonly known as Auditory Neuropathy Spectrum Disorder, (ANSD). Typically in ANSD there are normally functioning cochlear outer hair cells but abnormal auditory brainstem

responses [10]. It may be present in 10 to 15% of all children with hearing loss and in 15 to 20% of children with severe or profound hearing loss. The degree of hearing loss can range from mild to profound, but often involves difficulties with speech discrimination [11]. Hearing aids are helpful for many children with ANSD, but are ineffective for some who find speech sounds much distorted. Depending on the development of the auditory nerve, cochlear implants may work well. ANSD can be genetic in cause, and can be part of syndromes including Charcot-Marie-Tooth syndrome and Friedreich's ataxia; it is also associated with perinatal causes including asphyxia and severe jaundice.

Table – 1: Age distribution of cases.

Age of the patients (Years)	Group A	Group B	Total
10-20	5	6	11
20-30	10	9	19
30-40	12	10	22
40- 50	8	12	20
50-60	4	11	15
60-70	6	7	13

(*No of Pt's: Number of patients; *Group-A: Goitrous hypothyroid patients with hearing loss; *Group-B: Goitrous hypothyroid patients with normal hearing.)

Table – 2: Hearing loss (with correlation to mean T4 levels).

Hearing Loss	Number of Cases (n = 100)	Mean T4 (micrograms/dl)
Normal	61	2.7
Mild (25 - 40 db)	11	2.6
Moderate (41 - 55 db)	10	2.4
Moderately severe (56 - 70 db)	14	2.3
Severe (71 - 90 db)	04	1.8
Profound (>90 db)	ni	N/A

Table – 3: Response to L-thyroxine treatment (frequency specific/each ear) frequency.

Improvement in hearing						
	-10	**-5**	**0**	**10**	**15**	**Total**
250	2	3	14	12	9	40
500	1	5	6	9	8	29
1000	1	6	6	7	9	29
2000	8	4	6	2	8	28

In this study we recorded an overall statistically significant hearing improvement after thyroxine treatment But the author's would like to highlight that a significant 10 db improvement was observed in only 5 cases i.e. only about 13% cases had an objective audiological improvement though many cases claimed a subjective improvement in hearing. And one case also had deterioration in hearing. Further it was observed that the thyroxine treatment influences the hearing maximally at 1000 Hz, the frequency at which 31 ears had a significant gain of 10 db or more. There are diverse views regarding improvement in hearing in hypothyroid patients

with thyroxine treatment [12]. Although it was not the endeavour of this study to analyze the vestibular changes in goitrous hypothyroid- ism, the authors would like to highlight that almost 39% cases had a history of vertigo, out of which 26 patients had an subjective improvement after levothyroxine therapy at the end of 6 months. In this context it would be important to note that the medical text mentions that al- most 66% of patients of hypothyroidism suffer from vertigo Tinnitus too was present in 17 cases. Nine of these cases recorded a subjective improvement in form of decrease in the duration and intensity of the tinnitus. Thus a joint involvement of cochlear and vestibular sys- tem is also seen in goitrous hypothyroidism as has been reported for other metabolic disorders [13]. The authors would also like to highlight that no case of Meniere's disease *i.e.* the classical triad of vertigo, deafness with tinnitus was recorded in this case series, though the literature reports an intrinsic relationship between hypothyroidism and Meniere's disease (a recent study quotes a prevalence of 32%)

Interpretations of these results must take into consideration the limitations of our analysis. As data from a single tertiary health care centre was used, it reflects the experience of our geographical area and may not be generalized. Information from observational studies can be subject to potential biases (e.g. selection bias) and confounding. Critics may contend that 6 months follow-up period is short. Furthermore, the results were not ascertained blindly. Last but not the least; we were unable to discern the reason for this decreased sensorineural hearing loss in goitrous hypothyroidism. This could be due to the demographic profile of our patients. But then the authors would like to emphasise that the very cause of hearing loss in hypothyroidism is debatable and controversial [14]. Nevertheless, the strength of this study lies in its prospective character and independent statistical validation, which allowed for accurate assessment of data with-out depending upon recalled information in accordance with evidence based medicine. The authors would like to highlight that this study represents the largest series of patients on hypothyroidism. The true value of this study in context of existing literature lies in the audiological evaluation of patients belonging to the subgroup of goitrous hypothyroid, hitherto unreported in medical literature [15].

Conclusion

HG patients had more cochleovestibular symptoms, higher audiometric thresholds, and increase in L-V in the BERA and absence or reduction in TOAEs amplitudes. Such alterations were not associated with THS and free T4 levels.

References

1. Australian Hearing. Demographic details of persons under the age of 21 years with a hearing impairment who are fitted with a hearing aid, 2011.
2. Alford RL, Arnos KS, Fox M, Lin JW, Palmer CG, Scott DA, et al. American College of Medical Genetics and Genomics guideline for the clinical evaluation and etiologic diagnosis of hearing loss. Genetics in Medicine, 2014; 16(4): 347-55.
3. Nance WE. The genetics of deafness. Mental Retardation and Developmental Disabilities Research Reviews, 2003; 9(2): 109-19.
4. Grosse SD, Ross DS, Dollard SC. Congenital cytomegalovirus (CMV) infection as a cause of permanent bilateral hearing loss: A quantitative assessment. Journal of Clinical Virology, 2008; 41(2): 57-62.
5. Bhagya V, Brid SV, Doddamani M. Incidence of hearing loss in infants at risk. International Journal of Biological and Medical Research, 2011; 2(4): 1102-05.
6. Morzaria S, Westerberg BD, Kozak FK. Systematic review of the etiology of bilateral sensorineural hearing loss in children. International Journal of Pediatric Otorhinolaryngology, 2004; 68(9): 1193-98.

7. Furutate S, Iwasaki S, Nishio S, Moteki H, Usami S. Clinical profile of hearing loss in children with congenital cytomegalovirus (CMV) infection: CMV DNA diagnosis using preserved umbilical cord. Acta Oto-laryngologica, 2011; 131(9): 976-82.
8. Dowley AC, Whitehouse WP, Mason SM, Cope YM, Grant J, Gibbin KP. Auditory neuropathy: unexpectedly common in a screened newborn population. Developmental Medicine and Child Neurology, 2009; 51(8): 642-6.
9. V. T. Anand, S. B. S. Mann, R. J. Dash, et al. Auditory Investigation in Hypothyroidism. Acta Oto-Laryngologica (Stockholm), 1989; 108(1-2): 83-87.
10. J. A. DeVos. Deafness in Hypothyroidism. Journal of Laryngology & Otology, 1963; 77: 390-414.
11. Anand VT, Mann SBS, Dash RJ, Mehra YN. Auditory investigations in hypothyroidism. Acta Otolaryngol. (Stockh), 1989; 108: 83-7.
12. Figueiredo LCMS, Lima MAMT, Vaisman M. Alterações na audiometria de tronco encefálico em mulheres adultas com hipotireoidismo subclínico. Rev Bras Otorrinolaringol., 2003; 69: 542-47.
13. Khechinaschvili S, Metreveli D, Svanidze N, Knothe J, Kevanishvili Z. The hearing system under thyroid hipofunction. Georgia Med News, 2007; 144: 30-3.
14. Azevedo M, Sales B, Maia L, Leal E. Interpretação dos Testes de função tireoideana. In: Vilar L, editor. Endocrinologia Clínica. 3 ª. Edição. Rio de Janeiro: Guanabara Koogan; 2006, p. 219-30.
15 Papi G, Uberti ED, Betterle C, Carani C, Pearce PT, Braverman LE, Roti E. Subclinical hypothyroidism. Curr Opin Endocrinol Diabetes Obes., 2007; 14: 197-208.