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ORIGINAL ARTICLE

Audiological Evaluation in Patients with Vitamin D Deficiency

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ABSTRACT

BACKGROUND: Vitamin D status has been linked to proper hearing and vestibular functions. Vitamin D deficiency was correlated with ear, nose, and throat patients. Also, several studies suggested the possible relationship between vitamin D deficiency and hearing loss primarily of sensorineural type.

METHODS The current study was performed on Seventy-five subjects, the cases were allocated into two groups; control group (of 25 healthy subjects with Serum level of 25(OH) D (>20 ng/mL)), and study group consisted of 50 adults which were divided equally into two subgroups, subgroup I; adults with serum level of 25-hydroxy vitamin D <12 ng/ml, and subgroup II; adults with serum level of 25-hydroxy vitamin D=12-20 ng/mL. All cases were subjected to basic audiological evaluation.

RESULTS: There were no statistically remarkable variations between study and control groups in pure tone threshold at different frequencies, speech reception thresholds, and word discrimination scores.

CONCLUSIONS The present study demonstrated that hearing sensitivity was not affected by vitamin D status.

INTRODUCTION

Vitamin D is a group of steroids that has a remarkable role in homeostasis and increased calcium, phosphate absorption and a variety of biological activities, the most efficient compounds are vitamin D3 and vitamin D2. Vitamin D also has a significant role in other organs beside its classic target organs including kidney, bone, and intestine [1]. Previous studies assumed that vitamin D has a crucial role in proper hearing and vestibular function [2]. Its deficiency was found to be common in ear nose and throat cases [3]. Abnormal levels of vitamin D were correlated with otitis media with effusion in children [4]. Vitamin D supplementation was promising in cases of idiopathic tinnitus [5]. **Kwon et al., (2016)** reported that vitamin D receptor (VDR) gene mutation was correlated with ear disorders including impaired balance and deafness. VDR knockdown in the zebrafish model caused morphological ear deformity including abnormal otoliths, and smaller otic vesicles. Also, the embryos had decreased ear sensory hair cells giving evidence that VDR has a crucial role in ear proper development [2]. **Ikeda et al., (1989)** reported that deficiency of vitamin D is one of the causative agents of sensorineural hearing loss (SNHL) [6]. Studies on mice with a mutation in receptor vitamin D gene have shown that it is associated with progressive sensorineural hearing loss (SNHL) [7]. **Mehta et al., (2020)** revealed that deficiency of vitamin D was correlated with SNHL in children [8].

In this work we hypothesized that there is a probable higher rate of SNHL in cases with deficiency of vitamin D.

METHODS

The present study was executed on seventy-five subjects; the patients were selected among those undergoing serum level assessments of 25-hydroxy vitamin D. The cases were allocated into two groups: study and control groups.

Control group: The group consisted of 25 healthy subjects of both genders ranging in age from 20 to 50 years with Serum level of 25-hydroxy vitamin D >20 ng/mL.

Study group: It consisted of fifty adults (25 in each subgroup) of both genders and age matched with the control group. The group was allocated into two subgroups.

Subgroup I: adults with Serum level of 25-hydroxy vitamin D less than 12 ng/ml.

Subgroup II: adults with Serum level of 25-hydroxy vitamin D is equal to 12-20 ng/ml.

All subjects fulfilling the following criteria were enrolled in the study; subjects of both genders, with age from 20 to 50 years, normal middle ear functions, and with no history of neurological disorder or head trauma. Investigations were performed in Audio-Vestibular Medicine Unit, ORL Department, Zagazig University, Egypt. Written informed consent was obtained from all participants, the study was approved by the Research Ethical ommittee of Faculty of Medicine, Zagazig University, and was done according to the code of ethics of the World Medical Association (Declaration of Helsinki) for studies involving humans.

Equipment: COBAS 6000's module e601 by electro-chemiluminescence immunoassay using Roche Cobas vitamin D total assay reagent.

- Sound treated room
- Two channel diagnostic audiometer, Madsen, model Orbiter 922.
- Immittancemeter, Madsen model (Zodiak 902).

Methods:

All included cases were subjected to complete history taking (personal, medical, ontological, and detailed dizziness history), ontological examination to exclude external ear disease, and basic audiological evaluation.

Basic audiological evaluation:

A. Conventional pure tone audiometry (PTA) including air conduction for octave frequencies from 250 Hz to 8000Hz and bone conduction for octave frequencies (500 Hz to 4000Hz). Hearing sensitivity was measured by sending pure tone signals to each ear by earphones and changing the intensity of signal pending the hearing threshold was collected for each frequency. Pure tone average (PTA) of hearing thresholds at 500 – 4000Hz was calculated. The degrees of HL express the severity of HL that varies from mild to profound degrees [9].

B. Speech audiometry including speech reception threshold testing (SRT) and word discrimination testing (WD %).

C. Immittancemetry including both tympanometry and acoustic Reflex thresholds which were elicited contralaterally using pure tones of 500, 1000, 2000 and 4000 Hz to ensure normal middle ear functions.

Statistical analysis

The Statistical Package for the Social Sciences (SPSS) was used to collect and analyze the data (SPSS version 20.0). For statistically significant results, the P value was equal or less than 0.05, and for high significant results, it was equal or less than 0.001.

RESULTS

There was no statistically significant variation as regards age and sex among all participants.

The study groups (group I, II) had statistically significant lower 25-hydroxyvitamin D levels than the controls (table 1).

In the present study, all the patients in the control and study groups fulfilled the audiological criteria for normal hearing; they had bilateral normal hearing sensitivity, except two subjects had mild sensorineural hearing loss (SNHL) at 8 KHz. Word discrimination scores of all subjects were excellent and normal middle ear function. Consequently, there was no remarkable variation among groups respecting pure tone thresholds (table 2), Speech Reception Thresholds (SRT) (table 3), Word Discrimination scores (WD) (table 4) and Acoustic Reflexes at different frequencies (Table 5) (P >0.05).

Table (1): Vitamin D level distribution among studied groups

	Group I (<12 ng/ml)	Group II (12-20 ng/ml)	Control (>20 ng/ml)	F	P
Vit D (ng/ml)	8.91±1.68# 6-11.3	16.48±2.09 12.1-19.4	25.52±6.40* 20.1-44	113.715	0.00**

***** Group significantly higher by LSD (least significant difference test), # Group significantly lower by LSD

Table (2): Pure Tone Audiogram distribution among studied groups

		Group I	Group II	Control	F	P
250 HZ	Rt	11.78±3.65 5-20	13.86±5.96 5-20	13.60±4.21 10-20	1.566	0.216
	Lt	12.14±3.45 10-20	13.86±4.61 5-20	13.8±4.15 10-20	1.522	0.225

		Group I	Group II	Control	F	P
500 HZ	Rt	13.03±4.25 10-25	13.40±4.11 5-20	14.4±3.90 10-20	0.578	0.564
	Lt	12.67±3.96 5-20	15.0±5.0 5-20	13.6±3.39 10-20	1.843	0.166
1000 Hz	Rt	11.96±3.68 5-20	12.72±4.11 5-20	13.6±4.21 10-20	1.079	0.345
	Lt	11.42±3.87 5-20	12.95±4.22 5-20	13.7±3.53 10-20	2.765	0.095
2000 Hz	Rt	12.67±3.46 10-20	14.31±4.16 10-20	13.0±3.53 10-20	1.303	0.278
	Lt	12.32±4.11 5-25	15.45±5.09 5-25	14.80±4.44 10-20	3.091	0.052
4000 Hz	Rt	12.32±4.4 5-20	13.40±4.36 5-20	14.40±3.0 10-20	1.704	0.189
	Lt	12.17±3.25 5-25	14.31±4.55 5-25	13.60±4.21 5-25	2.199	0.125
8000 Hz	Rt	13.03±3.42 10-20	13.86±4.34 10-20	15.20±4.44 10-20	1.893	0.158
	Lt	12.50±4.25 5-30	15.22±5.66 5-30	15.0±3.53 10-20	2.333	0.104

Table (3): SRT distribution among studied groups

	Group I	Group II	Control	F	P
Rt SRT	11.60±3.54 5-20	13.86±4.34 10-20	13.0±3.53 10-20	1.963	0.148
Lt SRT	12.85±4.36 5-25	13.63±4.23 5-20	13.80±3.89 10-20	0.325	0.724

Table (4): WDS distribution among studied groups

	Group I	Group II	Control	F	P
Rt WDS	96.28±3.91 88-100	97.63±3.18 92-100	96.96±3.70 92-100	0.851	0.431
Lt WDS	95.71±4.47 88-100	97.45±2.63 92-100	96.64±2.98 92-100	1.508	0.228

Table (5): Acoustic Reflexes distribution among studied groups

		Group I	Group II	Control	F	P
500 Hz	Rt	88.57±3.56 85-95	88.63±3.83 85-95	88.60±3.68 85-95	0.002	0.998
	Lt	88.57±4.04 85-95	87.72±3.35 85-95	88.20±3.78 85-95	0.309	0.735
1000 Hz	Rt	88.74±5.21 85-95	87.98±4.56 85-95	88.36±4.21 85-95	0.444	0.643
	Lt	89.64±3.58 85-95	88.63±3.15 85-95	88.63±3.43 85-95	0.535	0.588
	Rt	90.17±3.46 85-95	88.86±3.42 85-95	89.60±3.51 85-95	0.885	0.417

		Group I	Group II	Control	F	P
2000 Hz	Lt	88.75±3.22 85-95	87.95±3.67 85-95	88.40±3.45 85-95	0.330	0.720
4000 Hz	Rt	90.35±3.31 85-95	89.54±3.05 85-95	90.0±3.22 85-95	0.394	0.676
	Lt	91.78±4.55 85-100	90.45±4.05 85-100	91.20±4.39 85-100	0.574	0.566

DISCUSSION

Vitamin D deficiency is endemic in Egypt, and caused by several factors including age, clothing, obesity, sun exposure, and chronic illness [10]. Vitamin D is responsible for homeostasis and increased calcium, phosphate absorption and is a major factor for bone formation and proper structure. The imbalance in vitamin D levels has a negative effect on the skeletal system and is involved in infectious, inflammatory, and metabolic processes [11]. For vitamin D status evaluation, serum 25-hydroxyvitamin D level is recommended to be used. There is an international lack of consensus about the description of vitamin D sufficiency and deficiency. However, many guidelines have defined a 25-hydroxyvitamin D level of 20 ng/mL as the threshold of sufficiency, <20 ng/mL to stand in for vitamin D deficiency and <12 ng/ML to represent severe deficiency which should be evaded in all age groups [12]. In the current study, all the participating subjects had normal, or hearing loss considered with the age with excellent word discrimination and intact acoustic reflexes. Two subjects showed a threshold of 30 dB HL at 8 kHz frequency which was considered high frequency mild SNHL [9]. The pure tone average for 8 kHz for both study and control groups were 12.50±4.25, 15.22±5.66 and 15.0±3.53, respectively. Using ANOVA Test the comparison of the results didn't show a remarkable variation between deficient vitamin D cases and controls as regards PTA, SRT, WD and ARs (P >0.05). Which means no relation was found between hearing thresholds and vitamin D level. This result was in line with **Jeong et al., (2013)** who had a study performed on postmenopausal females with osteoporosis reported that they did not find correlation between hearing loss, and vitamin D status [13]. Moreover, previous reports who audio logically assessed groups with different vitamin D levels did not report any significant variation between these groups as regards hearing thresholds [14,15]. Contrary to the present study results, **Brookes (2020)** had reported that ten patients with bilateral cochlear deafness were correlated with deficiency of vitamin D. The pathogenesis was assumed to be localized cochlear demineralization causing secondary changes of morphology. Vitamin D had a significant role with parathyroid hormone calcium metabolism homeostasis [16]. **Ikeda et al., (1989)** suggested that deficiency of vitamin D may cause bilateral SNHL by and cochlear microcirculation and metabolism of calcium [6]. **Ghazavi et al., (2020)** found that the frequency of deficient vitamin D cases with sudden SNHL (SSNHL) was higher than normal cases. The highest rate of cases with no treatment response was SSNHL deficient vitamin D cases [16]. Moreover, higher intake of vitamin D was correlated with lowered odds of hearing difficulties [17]. It was reported that children with vitamin D deficiency were more likely to have SNHL than age-matched normal cases without vitamin D deficiency [8]. A cross sectional study on elderly adults reported that vitamin D could be a remarkable risk factor for hearing loss related to age as they found that low vitamin D status was correlated with low frequency hearing loss [18]. Additionally, **Berner et al. (2018)** in their study of 638 cases with diabetes reported a correlation between vitamin D deficiency and HL [19]. Mechanism of vitamin D deficiency that could cause HL is thought to be related to the role of vitamin D in absorption of calcium and bone structure. Vitamin D deficiency had a variety of adverse effects including hearing, bone structure, nerve transmission, cochlear sensitivity to chronic ischemic effects, degeneration of auditory structures, and lysosomal enzymes imbalance that leads to deafness, demineralization of cochlea, and otic capsule impaired bone remodeling and give place to degeneration in stria vascularis, cochlear hair cells, and spiral ligament [20,21]. Vitamin D increases innate immunity and decreases adaptive immunity giving evidence for its role in inhibiting autoinflammatory and autoimmune processes [22]. Previous studies reported that vitamin D has a clear effect on endothelial cells that prevents vascular leakage. The inner ear inflammatory processes were correlated with microvascular elevated permeability of blood-brain-barrier [20] may be the vitamin D deficiency was not enough to affect the cochlea in patients of the present study, or cochlea is less susceptible to effects of vitamin D deficiency. Lack of available data for certain variables of interest, like total calcium level, parathyroid hormone (PTH) and bone mineral density (BMD) may be a limitation in this work.

CONCLUSIONS

The present study did not find a relationship between hearing sensitivity and vitamin D status. For better judgment more studies with higher sample size, more variables and different age groups are recommended.

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