Audiological screening in people with diabetes. First results

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Introduction

The relationship between diabetes mellitus (DM) and hypoacusia has been discussed since the work of Jordao, in 1857. Type II diabetes was considered a prevalent age-related medical condition, resulting in subclinical pathological changes. It was estimated that the incidence is about 9.6% of people in USA. (Cowie *et al*. 2006). Some studies has shown that the magnitude of hearing loss in patients with DM is related to the duration of the disease, age, and affects the auditory threshold to high frequencies. (Frisina *et al*. 2006). In a study of Tay HL *et al*. (1995) was found that there is a possible correlation between the duration of diabetes and hearing loss. The auditory system requires glucose and high-energy utilization for its complex signal processing. This suggests that the cochlea may also be a target organ for the ill effects of hyperglycemias. (Cullen and Cinnamond, 1993). Increased glucose exposure, even for short periods, initiates a metabolic cascade that could disrupt the cochlea both anatomically and physiologically (Jorgensen, 1961). Hearing depends on small blood vessels and nerves of the inner ear that are affected by high blood sugar level in diabetic patients. Outer hair cells modulate auditory reception in the inner ear: consequently, OAEs are commonly considered a useful index of cochlear function (Martin *et al.*, 1990). Well-established complications of diabetes, such as retinopathy, nephropathy, and peripheral neuropathy involve pathogenic changes to the microvasculature and sensory nerves (Acuña García, 1997). This conditions lead to a common symptoms in diabetic people that are tinnitus, dizziness and sensorineural hearing impairment, typically bilateral and progressive. Moreover, the specific pathologic effects of hyperglycemias and the complication associated with diabetes such as microvascular and neuropathic sorrows affecting also the ear including sclerosis of the internal auditory artery, thickened capillaries of the stria vascularis, atrophy of the spiral ganglion, and demyelination of the eighth cranial nerve, has been described among autopsied patients with diabetes (Lisowska *et al.*, 2001). Several studies are present in the international literature and the results are not unique. Compromised cochlear function has been measured using evoked otoacoustic emissions, a non-invasive method to assess damage to the outer hair cells of the cochlea, among patients with diabetes relative to healthy controls (Lisowska *et al.,* 2002). The aim of our study is to evaluate the topography of sensorineural hearing loss induced by diabetes, checking the sensitivity of audiological investigation to probe the damage.

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Methods

Selected subjects were divided in two groups: 40 patients with diabetes mellitus type 2 and 20 healthy controls. The examination with otoscopy was normal and tympanogramm was A type (i.e. without signs of inflammation in progress) in both groups.

In our study we rule out all subjects with a history of drugs able to influence the vascular reactivity, hearing loss or any middle/inner ear pathology or acoustic and cranial trauma; besides, any medical diseases which affect or are suspected to affect hearing (e.g. untreated hypertension, noise exposure, hypercholesterolemia, or use of ototoxic drug therapy), were excluded.

Hearing threshold were determined using:

- pure tone audiometry of frequencies 500, 1000, 2000, 4000, 6000 and 8000 Hz;
- relative impedance (stapedial reflex);
- Evoked Otoacoustic Emissions (TEOAEs and DPOAEs);
- Brainstem Auditory Evoked Potentials (BAEPs).

The Audiometric tests conformed to the specification in Amplaid A 321 (Acoustic test methods; basic pure tone and bone conduction threshold audiometry, International Organization for Standardization, Geneva, Switzerland). Impedence audiometry was performed for each tested ear. The tympanograms obtained were analysed for middle ear pressure and compliance values. The average threshold across the tested frequencies for each ear was evaluated.

Otoacoustic emissions (OAE) are sounds recorded in the external acoustic meatus that derive from the inner ear activity, specifically the movement of the outer hair cells. Testing of CEOAEs (Click Evoked OtoAcoustic Emissions) was accomplished using the ILO96 Otodinamycs analyzer (V6 ILO OAE Research).

Brainstem Auditory Evoked Potentials (BAEP) were accomplished using a OtoAccess program. The electrode impedance for the ear canal electrode, as well as the surface electrodes, was typically less than 5 kΩ.

Results

Analyzing the data obtained comparing to healthy subjects, diabetic patients showed an increase of the perception threshold at high frequency such as 4000-8000 Hz (P<0.01) (Table 1). We observed percentage about 16% of subjects with normal hearing and 83% of hearing loss; in a percentage (10%) of subjects with mean age of 40 and 50 years, we found already some degree of hearing impairment (+25 dB). The results from stapedial riflessometry showed that P2 and P4 were the parameters that more frequently increased in patients with diabetes. P2 78% for certainly increased and 8% with value probably increased. P4 certainly increased for 43% and 11% with value probably increased. TEOAEs reproducibility in both ears was significantly lower compared to control subjects, observed at the mean frequencies of 2-4 kHz (Figure 1). DPOAE intensity was reduced in diabetic patients as shown the Figure 2. The ABR showed average values of the latencies of waves V, with stimulus intensity of 60 dB and 100dB, and a significant increase in the values of ranges I-V, as shown in Table 2. According with the data in the literature, although many have not arrived at unequivocal conclusion, there is acknowledgement that diabetes cause a change in the speed of acoustic stimuli along the auditory pathways, regardless of type of diabetes and blood glucose.

A high incidence of impaired cochlear responses was found in diabetic patients, analyzing the results collected in our study.

Table 1.

Frequency	500	1000	2000	4000	8000	PTA
DM group	10.76±8.9	11.15±7.9	11.7±6.5	26.3±16.6	14.2±6.2	14.23±6.25
Control group	7±4.04	7.75±3.98	6.25±2.12	7±2.83	9.25±6.56	7.45±2.54
P	ns	ns	ns	<0.01	<0.01	<0.01

PTA, pure tone average.

Table 2.

dB nHL	wave I	wave III	wave V	IPL I-V	IPL I-III	IPL III-V
DM	1.6±0.13	3.8±0.18	5.66±0.26	4.06±0.19	2.26±0.24	1.79±0.14
CTRL	1.53±0.09	3.74±0.13	5.55±0.12	4.01±0.18	2.2±0.18	1.8±0.18
P	ns	ns	<0.01	<0.01	ns	ns

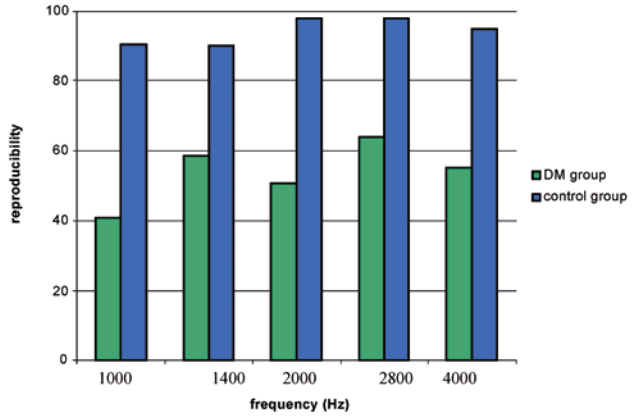

Figure 1. TEOAE test. Average of the reproducibility in the DM group versus control group in all frequency tested.

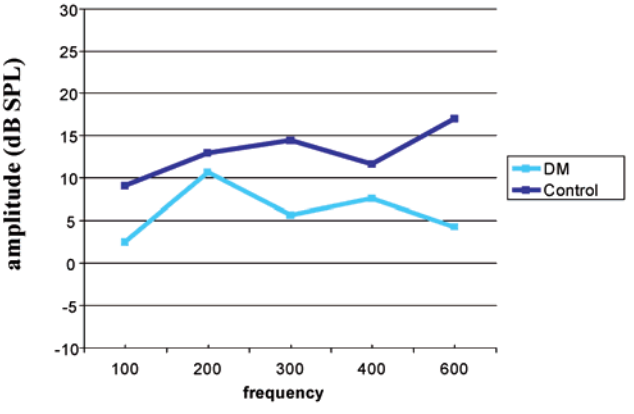

Figure 2. DPOAE amplitude in dB SPL.

Conclusions

Our report evaluates the association between diabetes and auditory dysfunction in patients, suggesting that diabetes could represent a risk factor for auditory pathway. To better understand the impact of auditory alterations in diabetes, we studied both cochlear function by recording TEOAEs and DPOAEs and neural transmission along the auditory pathway by recording ABRs. Adults with diabetes have a higher occurrence of hearing impairment than those without diabetes. Screening for this problem would allow for prevent an early hearing damage. From our primary results about study of functional brain-stem acoustic pathways of diabetic patients, we believe that the first diagnostic approach should be the impedence, that is easy and quick, whereas the ABR, that in our opinion should be recorded at least 2 rate of the stimulus, is a second level test for the subjects with normal responses in other audiological tests. The TEOAEs and DPOAEs have allowed a better understanding of the microvascular cochlear properties. These findings indicate a central disturbance in the auditory pathway and stress the idea of microvascular complications associated with diabetes. (Makishima and Tanaka, 1971)

In conclusion, the combined use of different procedures for monitoring the central and peripheral portions of the auditory pathway in diabetic patients showed the existence of the alterations in the cochlear micro-mechanisms and in the retrocochlear auditory pathway. Diabetes-related hearing loss has only been described as progressive, bilateral, sensorineural impairment with gradual onset predominantly affecting the higher frequencies. We observed stronger association between diabetes and high than low/mid frequency hearing impairment. Indeed the sufferance of the cochlear cells is major at the basal gyros, according to the theory of tonotopicity.

Compromised cochlear function has been demonstrated by a reduction of the OAEs values that could be attributed to a vulnerability of the hair cells to a glucose blood level (Sha *et al*., 2001).

Through the use of TEOAEs and DPOAEs, noninvasive techniques that give direct and objective information about the outer hair cell activity (Friedman *et al*., 1975), our results have contribute to underline the presence of subclinical alterations of the cochlear function in DM patients.

Our study may be contribute to focus on association of diabetes and hearing function, identifying an important public health problem that can be addressed. With the high prevalence of hearing impairment occurring among diabetic patients, screening for this condition may be justified. New studies could clarify whether the alteration of the auditory system could represent a useful means of staging and an early marker of ear dysfunction. The severity and the duration of the disease can contribute to the development of the decline of the neuronal and vascular function of the auditory pathway. Larger studies

will further help confirm the association and elucidate the auditory benefit of diabetes therapy.

References

Acuña García M, Herrero Laso JL, Durán Díez C, Menéndez Argüelles ME, Vallejo Valdezate LA, Díaz Suárez I, Pomar Blanco P. 1997. Diabetic complications and hypoacusia. An Otorrinolaringol Ibero Am 24(5):465-76.

Cowie CC, Rust KF, Byrd-Holt DD. 2006. Prevalence of diabetes and impaired fasting glucose in adults in the U.S. population: National Health And Nutrition Examination Survey 1999-2002. Diabetes Care 29(6):1263-1268

Cullen, J.R., Cinnamond M.J. 1993. "Hearing loss in diabetics", Journal of Laryngology and Otology, Volume: 107, pp. 179-182

Friedman SA, Schulman RH, Weiss S.1975.Hearing and diabetic neuropathy. Arch Intern Med. Apr;135(4):573-6.

Frisina ST, Mapes F, Kim SH, DR Frisina, RD Frisina. 2006. Characterization of hearing loss in aged type II diabetics. Hear Res. January; 211(1-2): 103-113.

Jorgensen MB. 1961. The inner ear in diabetes mellitus. Histological studies. Arch Otolaryngol 74:373-381.

Lisowska G, Namysłowski G, Morawski K, Strojek K. 2001. Cochlear dysfunction and diabetic microangiopathy. Scand Audiol Suppl (52):199-203

Lisowska G, Namysłowski G, Morawski K, Strojek K. 2002. Otoacoustic emissions and auditory brain stem responses in insulin dependent diabetic patients. Otolaryngol Pol 56(2):217-25.

Makishima K, Tanaka K. 1971. Pathological changes of the inner ear and central auditory pathway in diabetics. Ann Otol Rhinol Laryngol;80(2):218-228.

Martin, G.K., Probst R., Lonsbury-Martin B.L. 1990. "Otoacoustic emissions in human ears: normative findings", Ear Hear, Volume: 11, pp. 106-120

Sha SH, Taylor R, Forge A, Schacht J. 2001. Differential vulnerability of basal and apical hair cells is based on intrinsic susceptibility to free radicals. Hear Res 155:1-8.

Tay HL, Ray N, Ohri R, Frootko NJ. 1995. Diabetes mellitus and hearing loss. Clin Otolaryngol Allied Sci. Apr;20(2):130-4.