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Auditory Impairment in Young Type 1 Diabetics

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Background and Aims. More attention has recently been focused on auditory impairment of young type 1 diabetics. This study aimed to evaluate auditory function of young type 1 diabetics and the correlation between clinical indexes and hearing impairment.

Methods. We evaluated the auditory function of 50 type 1 diabetics and 50 healthy subjects. Clinical indexes were measured along with analyzing their relation of auditory function.

Results. Type 1 diabetic patients demonstrated a deficit with elevated thresholds at right ear and left ear when compared to healthy controls ($p < 0.01$). The elevated auditory threshold was significantly related with HDL-cholesterol, diabetes duration, and systemic blood pressure ($p < 0.05$). Moreover, latencies of right ear (wave III, V and interwave I–V) and left ear (wave III, V and interwave I–III, I–V) in diabetic group significantly increased compared to those in control subjects ($p < 0.01$). Auditory brainstem response was significantly related with $GHbA_1C$ and microalbuminuria ($p < 0.01$). Furthermore, distortion product evoked otoacoustic emissions (DPOAE) of diabetes group were statistically significant in right ears at 4.0, 6.0 kHz and in left ears at 4.0, 6.0, 8.0 kHz ($p < 0.01$) compared with those of controls. Diabetic patients demonstrated lower amplitude responses of the right ear than the left ear at 8.0 kHz. Only triglyceride was positively correlated to the hearing impairment defined by DPOAE ($p < 0.01$). There was no significance of transient evoked otoacoustic emissions (TEOAE) between groups. TEOAE was associated with age and $GHbA_1C$ ($p < 0.01$).

Conclusions. Type 1 diabetics exerted higher auditory threshold, slower auditory conduction time and cochlear impairment. HDL-cholesterol, diabetes duration, systemic blood pressure, microalbuminuria, $GHbA_1C$, triglyceride, and age may affect the auditory function of type 1 diabetics.

Key Words: Type 1 diabetes, Hearing impairment, Auditory brainstem response, Otoacoustic emissions.

Introduction

Diabetes mellitus (DM) is a chronic metabolic diseases derived from lack of insulin secreted by the pancreas or from the ineffective use of available insulin, which is characterized by increased blood glucose levels (1). Type 1 DM is β cell destruction and absolute insulin deficiency caused by autoimmune response of pancreatic β cells. It mainly occurs in teenagers and accounts for 10–15% of diabetes (2).

Chronic hyperglycemia of diabetes can affect its metabolic balance and function leading to hearing impairment because the inner ear does not store energy (3). Many studies have indicated that hearing impairment is associated with DM (4–9). Both type 1 and type 2 DM can cause hearing loss (9–11). It has been shown that hearing loss of diabetes is bilateral sensorineural deafness in middle and high frequencies (11–15). Angiopathy and neuropathy of diabetes have been considered as reasons for hearing impairment

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because they affect vessel function in the inner ear (16). However, not all investigators believe that DM can cause sensorineural hearing impairment (6,17–19). Most recent studies focus on sensorineural hearing loss in type 2 DM; studies of hearing impairment in type 1 DM are very limited.

The most common methods of testing hearing impairment include pure tone audiometry, auditory brainstem response (ABR) and otoacoustic emissions (OAE). Okhovat et al. reported that type 1 diabetic patients had higher auditory thresholds than healthy group by pure tone audiometry (15). Dayem et al. found that type 1 diabetics had significantly lower signal at middle and high frequencies of transient otoacoustic emissions (20). The significantly delayed latencies of ABR peripheral transmission time (wave I) and central transmission time (interpeak latency I–V) in the type 1 diabetic were also reported (21). Therefore, we aimed to examine the auditory status in young type 1 diabetic patients and to determine the clinical factors associated with hearing loss in this study.

Materials and Methods

Subjects

A total of 50 type 1 diabetics from September 2013–September 2014 were enrolled in this study. They were from the Departments of Endocrinology and Nephrology, Qilu Hospital of Shandong University. An additional 50 healthy subjects representing the control group were from the physical examination center of Qilu Hospital. All subjects who met the following inclusion and exclusion criteria were enrolled. Inclusion criteria were as follows: patients diagnosed as type 1 diabetes according to the standard of the 2011 American Diabetes Association guidelines (22). Exclusion criteria were as follows: history of severe head trauma, neurological medical conditions, family history of severe hearing loss, occupational ototoxic medication and/or noise exposure, clinically overt hearing impairment such as otosclerosis, otitis media, and Meniere's disease. Our study was approved by the Ethics Committee of Qilu Hospital of Shandong University and signed informed consent was obtained from each subject.

Type 1 DM group consisted of 50 patients (24 females and 26 males, 100 ears tested) and ages ranged from 17–46 years with a mean of 25.56 years. Control group included 50 healthy subjects (24 females and 26 males, 100 ears tested) and ages ranged from 17–45 years with a mean of 27.56 years. All type 1 diabetic patients received a daily subcutaneous insulin injection to control blood glucose.

Clinical Examination

Body mass index (BMI) was calculated for each subject and overweight was defined as BMI 24.0–25 kg/m^2 and obesity was defined as BMI >25 kg/m^2. Blood pressure was measured in a sitting position after a 5-min rest using a mercury sphygmomanometer. Blood urea nitrogen (BUN), creatinine (Cr), triglyceride (TG), total cholesterol (TC), low-density lipoprotein cholesterol (LDL-c) and high-density lipoprotein cholesterol (HDL-c) were measured for all subjects. Glycosylated hemoglobin ($GHbA_1C$) level was measured and its level $<7.5\%$ was considered as good blood glucose (BG) control, whereas HbA1c$>7.5\%$ was considered as poor BG control.

An electromyogram was performed to identify peripheral neuropathy and an ophthalmological evaluation was used to identify diabetic retinopathy. Albumin level was measured for each subject. Microalbuminuria was defined as 20–300 mg/24 h and macroalbuminuria >300 mg/24 h.

Pure-tone Audiometry

Air- and bone-conduction auditory tests of pure-tone were performed for all ears in a soundproof room using a clinical audiometer (Hortmann, Leverkusen, Germany) with insert earphones at frequencies of 250, 500, 1000, 2000, 4000 and 8000 Hz. Pure-tone threshold >25 dB was defined as hearing impairment at any frequency (23).

Auditory Brainstem Response

Auditory brainstem response (ABR) used the Ep25 system (Audiometrics, Oceanside, CA) to record latencies of wave I, III, V and inter-wave latencies of I–III, III–V, I–V with disk electrodes placed on the mastoids and high forehead. This system utilized stimulus intensity 130 dB sound pressure level (SPL) with a rate of 11.0 and 80.1 clicks/sec, repetitions 2000, and filters 0.5–4 kHz. Abnormal ABR was defined as difference of latencies >0.4 msec at the same wave between the left and right side or the latencies more than mean +standard deviation (SD) of healthy group offered by our Ear-Nose-Throat Laboratory.

Otoacoustic Emissions

Distortion product evoked otoacoustic emissions (DPOAE) and transient evoked otoacoustic emissions (TEOAE) amplitudes of each subject were record by the Madsen Capellia system (GN Otometrics, Taastrup, Denmark) in a soundproof room. DPOAE were record for all ears with primary (f1) and secondary (f2) pure tone stimuli. The frequency ratio of f2/f1 was fixed at 1.22 with the stimulus levels at L1 = 65 dB SPL and L2 = 55 dB SPL. The 2f1–f2 DPOAE amplitude as a function of frequency was record in the 0.75–8 kHz range. TEOAE were evaluated at a level of 80 dB SPL with a 'non-linear' click stimulus and amplitudes between 1 and 4 kHz were record. Abnormal OAE were considered >20 dB SPL or ≤ 5 dB SPL.