Otoacoustic emissions recorded in people with chronic migraine

Lori Cameron, Wei Sun*

Department of Communicative Disorders and Sciences, State University of New York at Buffalo, 122 Cary Hall, 3435 Main Street, Buffalo, NY 14214, USA

Received 12 December 2014; revised 20 December 2014; accepted 9 January 2015

Abstract

Migraine, a moderate to severe chronic headache occurring on one or both sides, is a common disease affects young people. Although hearing loss in subjects with migraine is not rare, the correlation of migraine with hearing loss is not clear. In this study, we examined hearing loss in young migraine subjects to determine if migraine may be a factor in causing cochlear dysfunction. Seven college students with migraine and three age matched subjects without history of migraine were assessed using extended high frequency audiometry and distortion product otoacoustic emissions (DPOAEs). There was no significant difference in regular audiometry threshold between the migraine group and the control group. However, high frequency audiometry (9–16 kHz) showed thresholds at 25 dB nHL or higher in six out of twenty ears in the migraine group. The amplitude of DPOAEs were reduced for more than 10 dB in the migraine group in comparison with the control group. These data suggest that migraine may affect cochlear dysfunction evidenced by the reduced amplitude of DPOAE and high frequency pure-tone audiometry.

Copyright © 2015, PLA General Hospital Department of Otolaryngology Head and Neck Surgery. Production and hosting by Elsevier (Singapore) Pte Ltd. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

Keywords: Migraine; Hearing loss; Otoacoustic emission; High frequency audiometry

1. Introduction

Migraine is moderate to severe chronic headache which can occur on one or both sides of the head. The symptom of migraine can be exacerbated by physical movement. A headache must have at least two of the following characteristics for migraine: unilateral location, pulsating quality, moderate or severe pain intensity, and aggravation by routine physical activity. Additionally, during the course of the headache, patients have at least one of the following: nausea with/without vomiting, photophobia, and phonophobia. Most migraine sufferers experience the following symptoms during or two days prior the onset of a migraine: flashes of light, nausea, unusual sensitivity to light and sound, tingling in the limbs, cravings for sweets, drowsiness, extreme thirst, intense energy, fatigue, sweating, and irritability or depression. A migraine is known to last for short durations such as a couple hours up or longer periods of up to five days. Sometimes the symptoms can be so disabling that the individual cannot attend to normal daily activities. According to a 2001 report by the National Institute of Neurological Disorders and Stroke, more than 28 million people in the United States experience migraines. Statistical data reported by the Centers for Disease Control and Prevention (CDC) indicate that females have higher incidence than males of migraine or severe headache (Pleis et al., 2010). Adults aged 18–44 years are almost three times as likely as adults 65 years or older to experience migraines and it is known to run in families (Olsen and Lipton, 2004).

It is unclear exactly what causes migraine. Migraine may be caused by abnormal brain activity that is caused by stress, particular foods, environmental or unknown factors. A large number of medical experts now think the migraine attack begins in the brain, and includes a mixture of nerve pathways and
chemicals. These changes are thought to influence blood flow in the brain and neighboring tissues (Caplan, 1991). Previously, researchers have linked migraine to blood vessel dilation and constriction in the head (Caplan, 1991). Recent theories from scientists now think migraine may be caused by genetic abnormalities in particular cell populations in the brain. Interestingly, some individuals have also experienced a temporary sudden increase of hearing threshold (Viirre and Baloh, 1996). This leads to concerns that it may be a sign of further damage that could be happening in the inner ear. Lipkin et al. (1987) described a patient with sudden sensorineural hearing loss connected with typical migraine headache. Caplan (1991) described a sudden onset of a unilateral hearing loss and aural fullness as the preceding symptom of a cerebellar infarct in a patient who had migraine with visual aura and basilar artery stenosis. Viirre and Baloh (1996) described 13 patients with unexplained sudden hearing loss and neurological symptoms, all of whom had long-standing migraine headaches. Lee et al. (2000) described a patient with long-standing migraine with aura who experienced sudden hearing loss at the age of 50 years and onset of Meniere's syndrome at the age of 73 years. These authors suggested that migraine-associated vasospasm may have been the cause of sudden deafness.

Migraine is commonly reported in college students, especially in female students. However, there is no study on the hearing loss symptoms in this population. The objective of this study is to determine if changes in hearing occur at higher frequencies in young adults who suffer from migraine. Two specific aims of this study are: 1. To provide evidence regarding whether hearing loss is a symptom of migraine using extended high frequency audiometry; 2. To provide evidence demonstrating utility of extended high frequency air conduction audiometry and distortion product otoacoustic emissions (DPOAE) in the diagnosis of cochlear dysfunction in migraine.

2. Materials and methods

2.1. Subjects

Ten college age individuals (22–26 years old) participated in this study. All of the participants were recruited from the New York State University at Buffalo campus. Seven participants (five females and two males) who suffered from chronic migraine were recruited as the experimental group. Three participants who showed no signs of chronic migraine were recruited for the control group. No significant audiological issue or noise exposure history was reported by any of the participants. All of the chronic migraine participants were asked to complete an additional questionnaire prior to testing to ensure these criteria was met. Prior to testing, a consent form was completed by the participants.

2.2. Audiologic testing

All audiologic testing was carried out in a sound attenuation booth. Tympanometry was measured using the GSI TympStar Middle Ear Analyzer. Pure tone air conduction stimuli were delivered through a GSI-61 audiometer. Audiometric inclusion criteria for participation in the study were set as follows: pure-tone air conduction thresholds via insert earphone transducers at ≤ 25 dB HL bilaterally between 250 and 8000 Hz, pure-tone air conduction thresholds via high-frequency headphone transducers at 0–70 dB HL bilaterally between 9 and 16 kHz, and normal middle ear pressure and compliance.

2.3. DPOAE testing

Measurement of DPOAEs was carried out using the SCOUT DPOAE system. The probe was placed in each subject's right ear, and sealed through a foam ear-tip. Two pure tone signals, F1 and F2, were simultaneously presented to record the distortion product 2f1–f2, with parameters that produced the most robust emission (F2:F1 = 1.2, the intensity level of F1 is 10 dB higher than F2). F2 frequencies were set at 1797, 2147, 2566, 3080, 3640, 4363, 5133, 6160, 7280, 8726, 10313, 12319, and 14629 Hz. The same trials that were completed in right ear were then measured in the left ear.

3. Results

3.1. Migraine symptoms

The chronic migraine subjects were asked to complete a questionnaire about their history of migraine. All seven migraine subjects reported different migraine symptoms, such as visual or hearing changes, dizziness, fatigue, loss of appetite, nausea/vomiting, problems concentrating or numbness/weakness (Fig. 1A). In 5 out of the 7 migraine patients, there were reports of warning symptoms such as eye pain, blurred vision, seeing stars, tunnel vision or temporary blind spot (Fig. 1B). Only 3 of the 7 migraine patients reported temporary changes of hearing that resolved within a few hours from the episode.

3.2. Pure tone audiometry

All subjects in both the control group and the migraine group had pure tone thresholds lower than 25 dB HL from 250 to 8000 Hz. Six out of the fourteen ears in the migraine group had pure tone thresholds at 25 dB nHL or higher from 9000 to 16000 Hz. The averaged pure tone threshold is graphed in Fig. 2A. No statistical significant difference was found in the control group compared with the migraine group (two-way ANOVA test, P > 0.05). The hearing thresholds at 12 and 16 kHz in individual ears are plotted in Fig. 2B and C, respectively. Although two ears in the control group showed thresholds at 30–40 dB HL, the hearing threshold at neither frequency was statistically significant compared to the migraine group (Student's t test, P > 0.05).

3.3. Distortion product otoacoustic emissions (DPOAEs)

Analysis for DPOAE testing was carried out using the Prism analysis software. A two-way ANOVA with repeated
measure test showed that results were significantly different on the DPOAE amplitude when comparing migraine patients with the control subjects ($P < 0.05$) (Fig. 3A). DPOAEs of less than 10 dB SPL were found more often in the migraine subjects in comparison with the control subjects (Fig. 3B). A two-sided Fisher’s exact test was statistically significant when individual ears were compared at suppression levels less than 10 dB ($P < 0.05$, Fig. 3C). However, when DPOAE amplitude suppression was compared at 10 kHz, no significant difference was found (Student’s t-test, $P > 0.05$, Fig. 3D). Difference in length of migraine history was not statistically significant and it had no impact on DPOAE amplitude (Student’s t-test, Fig. 3E).

4. Discussion

Each participant’s hearing was assessed as part of inclusion criteria in the study. All participants had thresholds of less than 25 dB HL from 250 to 8000 Hz, indicative of normal hearing sensitivity. When high frequency audiometric thresholds were assessed at 9–16 kHz, there was a threshold increase of up to 40 dB HL in six subjects in the migraine group. However, the threshold increase in the migraine group was not statistically significant compared to the control group due to the large variability of threshold. We also found significantly reduced DPOAE amplitudes in the migraine group at 10 kHz. Therefore DPOAE is more sensitive to show early signs of cochlear dysfunction in the migraine group.

A study by Hamed et al. was published after completion of this study and assessed patients with migraine using routine diagnostic audiometry, transient evoked otoacoustic emissions (TEOAEs), DPOAEs, and auditory brainstem response (ABR). The data of the study suggest that migraine patients are at high risk of peripheral and/or central auditory dysfunction. The study suggests a compromise of blood supply of the auditory system in migraine patients. In comparison to our current study, the results are similar in that most of the patients had normal hearing but did show lowering of DPOAEs, indicating cochlear or peripheral auditory dysfunction.
In this study, we tried to use high frequency audiometry to detect an early sign of hearing loss in migraine patients. However, high frequency audiometry has no standard norms. Schechter et al. (Schechter et al., 1986) have studied 157 subjects aged 6−30 years and tried to report normative data of high-frequency (8−20 kHz) auditory thresholds in normal hearing individuals. This auditory threshold normative survey confirmed the gradual reduction of high-frequency sensitivity from young children until young adulthood. The data suggests that both intra-age category threshold inconsistency and inter-aural threshold differences tend to increase with both increasing age and test frequency. In our study we found a large variability in the hearing threshold at high frequencies, despite normal hearing thresholds in the regular range of audiometry.

OAEs are an objective measure for establishing cochlear function. OAEs are low level signals that are reflected by cochlear outer hair cells and are extremely sensitive to outer hair cell dysfunction. We found reduced amplitude of DPOAEs in migraine subjects showing hearing thresholds within normal limits. This result suggests that DPOAEs may be a better objective measure for early detection of auditory weakness in patients with migraine.

There are a few significant limitations that are associated with the outcomes of this study. One of the major shortcomings is the number of subjects who were able to be tested. The study was not funded which also limited the amount of participants who could be recruited. With only three participants in the control group and seven in the experimental group, the ability to generalize results to a larger population is limited. Future studies in this area should include more participants to increase the study's external validity.

References