Review Article

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OTOACOUSTIC EMISSIONS IN TYPE 2 DIABETES: A SYSTEMATIC REVIEW

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ABSTRACT

Outer row of hair cells in the organ of Corti are not directly involved in deciding the threshold of the acoustic stimulus, but their damage will increase the hearing threshold and may even cause the deafness. Type 2 diabetes is increasing globally at an alarming rate, which is affecting the normal hearing. In type 2 diabetes poor metabolic control, neuropathy or microangiopathy may affect the normal hearing. This study is focused on self enhancement of the knowledge in the particular field apart from providing the complete review on effect of type 2 diabetes on otoacoustic emissions to the readers. Articles published in English on otoacoustic emissions in type 2 diabetes were collected and reviewed from Pubmed, Google and Google Scholar. Hearing threshold is increased in type 2 diabetic patients and amplitude of otoacoustic emissions was decreased. Hearing loss is observed in type 2 diabetes and it is attributed to damage of outer row of hair cells in organ of Corti.

Keywords: otoacoustic emissions, type 2 diabetes, outer row of hair cells

INTRODUCTION

Otoacoustic emissions are low level acoustic signals originated from the outer row of hair cells of the cochlea[1,2] and they are used widely to study the cochlear function and hair cell micromechanics in humans[3]. Outer row of hair cells play a significant role in normal hearing when the sound stimulus intensity is bellow 60 dB[4] and they give an indirect idea about the functional status of inner hair cells[5]. Otoacoustic emissions are useful in screening for cochlear (outer hair cell) functioning, differentiation of cochlear versus retro cochlear auditory dysfunction and also in diagnosis of auditory dysfunction. Cellular metabolism in the outer row of hair cells increases dramatically when they are activated and the outer row of hair cells rapidly elongate during hyper-polarization and become shorter during depolarization. Change in outer hair cell length generates energy within the cochlea that contributes to hearing sensitivity and the ability to distinguish small differences in the frequencies of sounds[6]. Transient evoked otoacoustic emissions are not recorded with audiometric thresholds greater than 25 to 30 dB HL[7]. The absence of distortion product otoacoustic emissions with normal middle ear function is an indication of audiometric thresholds greater than 30 to 35 dB HL[8]. Globally 382 million people have diabetes and the number is set to rise beyond 592 million in less than 25 years. Currently 175 million undiagnosed diabetic cases are present across the globe and the number is increasing at a robust rate, a vast amount of people with diabetes are progressing towards complications unawares[9]. Like all the living cells, neuronal cells that are involved in auditory system also requires glucose for their survival and also for complex signaling process. And this suggests that the cochlea may also be a target organ for the damaging effects caused by hyperglycemia in type 2 diabetes[10]. In type 2 diabetes, hyperglycemia causes widespread tissue damage, most specifically injuring neural tissue[11, 12]. Type 2 diabetes effects extracellular matrix and results in thickening of the collagen fibers[13 - 15]. In diabetes, protein kinase C is up regulated and binds with PX2 receptors on mammalian hair cells, activating a down regulation of Na/K/ATPase causing elevated extracellular K+ and intracellular Na+ and Ca++ and resulting in excitotoxicity[16, 17]. This study is focused on self enhancement of the knowledge in the particular field for the authors apart from providing the complete review of articles on effect of type2 diabetes on otoacoustic emissions to the readers. Published articles in English on otoacoustic emissions in type 2 diabetes were collected and reviewed from Pubmed, Google and Google Scholar. Exclusion criteria: Otoacoustic emissions in type 1 diabetes were excluded.

OBSERVATION & CONCLUSION

Cochlear function is affected to different degrees at different frequency levels in type 2 diabetic patients[18]. Mean amplitudes of otoacoustic emissions were decreased in type 2 diabetes induced CAB/CaJ mouse model for lower and higher frequencies[19]. Hearing loss observed in type2 diabetes induced rhesus monkeys is predominantly cochlea[20]. Decreased amplitude of distortion product otoacoustic emissions were observed in type 2 diabetic patients when compared with the control subjects[21 - 27]. Right ear outer row of hair cells are damaged more than the left ear outer row of hair cells in type 2 diabetic patients[10]. Ears with damaged outer row of hair cells have reduced sensitivity as well as broader tuning than ears with normal outer row of hair cells[28 - 31]. Brain metabolites are also altered in type 2 diabetes; this may also have contributed for the hearing changes in diabetics[32]. All the studies have shown that hyperglycemia in type 2 diabetes decreases the amplitude of outer row of hair cells. They also have shown compelling evidence on increased hearing threshold in type 2 diabetics. Auditory screening with otoacoustic emissions is useful in early

detection of increased hearing threshold and decreased amplitude of outer row of hair cells, which is very essential in taking the prophylactic measures for preventing the further damage. So routine screening for hearing in type 2 diabetic patients with distortion product otoacoustic emissions is advisable. Longitudinal cohort studies are required on otoacosutic emissions for drawing the correlation between the hyperglycemia and functioning of outer row of hair cells in type 2 diabetics.

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