



Cochlear Affection in Obese Children

Rafeek Mohamed A*

Department of Otolaryngology, Minia University, Egypt

Abstract

Objective: For assessment of hearing of obese children and to detect even minor or early changes in the cochlea of obese children then to determine if obesity is a risk factor for hearing or not.

Methods: The 39 children were examined in this research, 10 of them were normal weight children while the others were obese children with BMI equal to or more than 95 percentile. All children were subjected to the following tests as BMI assessment, laboratory investigation in the form of evaluation of blood hemoglobin, blood lipid profile and blood glucose level. Hearing assessment by pure tone audiometry, tympanometry, acoustic reflex, extended high-frequency assessment and Otoacoustic Emission (OAE) assessment.

Results: There is no statistically significant difference either in pure tone assessment and extended high frequencies assessment or laboratory test. However, there is the difference in 20 KHz, and transient evoked otoacoustic emission.

Conclusion: Obesity by itself is a risk factor for hearing, so hearing screening of obese children is considered.

Keywords: Obesity; Cochlea; Children

Introduction

Obesity is not as simple symptoms, but it is a chronic disease. Obesity is not only chronic disease, but it is an epidemic raising grave affecting all the public health [1]. Obesity has the greatest harmful effect on health even more than tobacco [2].

Excessive fat in the body collected to form adipose tissue. Adipose tissue is not only as simple tissue, but it acts like endocrine gland as it enhances secretion of offensive and inflammatory adipokines as plasminogen activator inhibitor-1 & interleukine-6 and c-reactive protein, while decrease production of protective adipokines as adiponectin [3].

All these changes create a state of chronic low-grade inflammation, recently called meta-inflammation and impairment of immune response. All these factors make obesity as a risk factor for major chronic disease [4].

Otoacoustic Emission (OAE) is a fascinating auditory phenomenon. OAE is sound energy that originates in the cochlea then propagates through the middle ear and into the ear canal where it could be measured using a sensitive microphone. Kemp first described OAE [5]. OAE is a preneural phenomenon; it can be measured even when eighth nerve activities is chemically blocked [6]. OAE is vulnerable to subtle changes in the cochlea which cause damage to outer hair cells or cause hearing loss [7].

Extended High-Frequency Audiometry (EHFA) can detect early changes in hearing which almost occur in high frequency in the beginning. So, EHFA and OAE used in the monitoring of hearing associated with ototoxicity. Ototoxicity is a chemical injury to the labyrinth as a side effect of pharmacotherapy. An ototoxic insult usually affects the high frequencies early then progress to low frequencies. So, the key is to monitor hearing by assessment of high frequencies by extended High-Frequency Audiometry (HFA) or by otoacoustic emission [8,9].

The primary target of this research was to detect any subtle changes in the cochlea of obese children and to detect even minor changes in the hearing of those children by OAE and HFA. For the best of our knowledge, the studies to examine the effect of obesity on hearing of children are quite scarce. So, the current work was addressed to investigate the hearing of obese children, hence to determine if obesity, by itself is a risk factor for hearing in obese children or not.

OPEN ACCESS

*Correspondence:

Rafeek Mohamed A, Department of Otolaryngology, Minia University, Minia, Egypt, Tel: +20-1000432269; E-mail: rma1hfa2002@yahoo.com

Received Date: 15 May 2020

Accepted Date: 09 Jun 2020

Published Date: 16 Jun 2020

Citation:

Rafeek Mohamed A. Cochlear Affection in Obese Children. Am J Otolaryngol Head Neck Surg. 2020; 3(5): 1100.

Copyright © 2020 Rafeek Mohamed A. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Patients and Methods

A total number of (39) subjects were examined in this study. All subjects were selected from outpatient clinic of E.N.T Department, Minia University Hospital from 6-2015 to 4-2016.

The study group was divided into two subgroups, their age range from 6 to 17 years. The control group (group A) consisted of 10 children, four boys, and six girls their mean age 11.5 ± 4.05 while the second group (group B) consisted of 29 children, 13 boys, and 16 girls. Their mean age 10.62 ± 2.77 . The first group has average weight while the second group composed of obese children with Body Mass Index (BMI) more than 95th percentile according to Center for Disease Control and Prevention (CDC) criteria of obesity in children. While the control group their age from 6 years to 17 years, composed of average weight children according to CDC criteria.

Both groups have no history of systemic diseases, medical diseases or immunological diseases that affect hearing. There was no history of hearing loss or delayed speech and language development. And no consanguinity between parents. Both groups have type (A) tympanogram and intact acoustic reflex with the normal acoustic reflex threshold.

The only difference between both groups in BMI as obese children have BMI more than 95th percentile while the other control group has average weight according to CDC criteria.

Parents of the participated children had been informed about the study aims and the detailed procedures to be used, before taking part. They gave written consent for their children participate in the study, and the ethical research committee approved all procedures at Minia University.

All children participated in the current work were subjected to the following, full history taking including prenatal, perinatal, neonatal, postnatal, developmental and family history & otological examination & tympanometry and acoustic reflex testing. According to the child age and reliability either conditioned play audiometry or conventional audiometry was performed using audiometer Madsen Astera and sound treated room amplisilence. Air conduction threshold was measured at frequencies 0.25 KHz, 0.5 KHz, 1 KHz, 2 KHz, 4 KHz, 8 KHz. Bone conduction thresholds was measured in frequencies 0.5 KHz, 1 KHz, 2 KHz and 4 KHz.

Examination includes determination of hearing threshold for extended high frequency audiometry in the frequency range (9 KHz, 10 KHz, 11 KHz, 200 KHz, 12 KHz, 500 KHz, 14 KHz, 18 KHz, 20 KHz). Laboratory investigations that include; Complete Blood Picture (CBP), lipid profile and blood sugar.

BMI calculated according to birth date, date of measurement, Sex, Height, and Weight. (Centers for Disease Control and Prevention), www.cdc.gov/healthyweight/assessing/bmi/index.html. Before measurement of the height and weight, the children were asked to remove outer clothing (e.g., coats), shoes, and removable hair accessories and to remove personal items from their pockets. The height was measured to the nearest inch using a weighted measuring tape attached to the wall. Children placed their backs and heels against the wall. Then a measuring triangle was placed on the children head to form a right angle with the wall. The height measurement was taken from the lower edge of the triangle. The weight also measured to the nearest gram using an electronic scale placed on an uncarpeted floor. The scale was zero balanced before each child was weighted.

Table 1: Mean and SD of BMI of group (A) and group (B).

	Obese (n=58)	Control (n=20)	P value
Range	(20.5-47.3)	(14.6-25.1)	<0.001*
Mean ± SD	30.69 ± 7.09	18.6 ± 4.19	

Table 2: Mean and SD of hemoglobin, cholesterol, triglycerides & blood sugar of group (A) and group (B).

	Obese (n=58)	Control (n=20)	P value
Hemoglobin			
Range	(10.4-1205)	(11.8-13.6)	0.405
Mean ± SD	53.88 ± 219.44	12.65 ± 0.53	
Cholesterol			
Range	(107.69-208.02)	(107.6-200)	0.3
Mean ± SD	168.29 ± 26.87	160.83 ± 29.45	
Triglycerides			
Range	(52.21-265)	(50.93-196)	0.335
Mean ± SD	151.86 ± 49.69	138.81 ± 58	
Blood sugar			
Range	(81.7-110)	(90-110)	0.478
Mean ± SD	95.07 ± 5.94	96.14 ± 5.2	

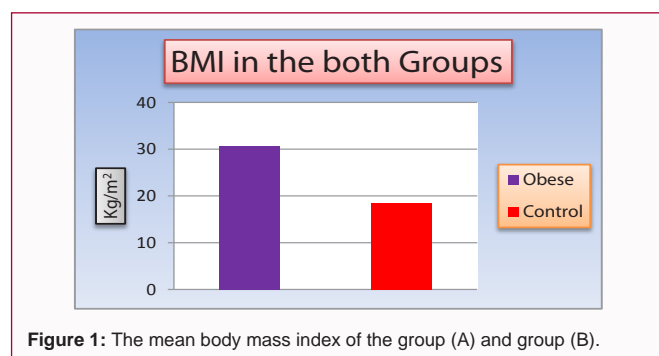


Figure 1: The mean body mass index of the group (A) and group (B).

Measurement of Transient Evoked Otoacoustic Emission (TEOAE) using Smart Intelligent Hearing System (IHS), Stimuli were 75 us rectangular clicks presented at a peak level of 80 dB pSPL. A total number of 1,024 sweeps were averaged using a 500 Hz to 6000 Hz bandpass. The level of TEOAE was spectrally analyzed and automatically determined at different frequency bands.

Distortion Product Otoacoustic Emission (DPOAE) was recorded using the IHS two channels evoked potential recording apparatus with Smart EP software, version 4.5 with smart OAE 4.5 software. Two tones were used: $L_1=65$ dB (SPL) and $L_2=55$ dB SPL, while f_2/f_1 was 1.22. Both the amplitude of the response of the Distortion Product (DP) at $2f_1-f_2$ and background Noise (Ns) were obtained at nine points corresponding to f_2 frequencies of 553 Hz, 783 Hz, 1105 Hz, 1560 Hz, 2211 Hz, 3125 Hz, 4416 Hz, 6250 Hz and 8837 Hz. These measurements were used to build a DP-gram by displaying the DP against the f_2 frequency. The Signal-to-Noise Ratio (SNR) was measured (SNR=DP-Ns) at each of these nine points. DPOAE was considered normal, thereby reflecting normal cochlear function, if the SNR was >3 dB SPL on at least 70% of the tested frequencies.

Results

There is statistically significance ($P<0.05$) between the two groups as regarding BMI as shown in Table 1 and Figure 1, while there was no statistically significance ($P>0.05$) between the two groups as

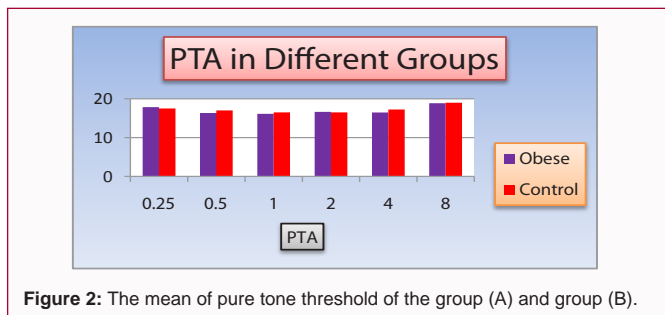


Figure 2: The mean of pure tone threshold of the group (A) and group (B).

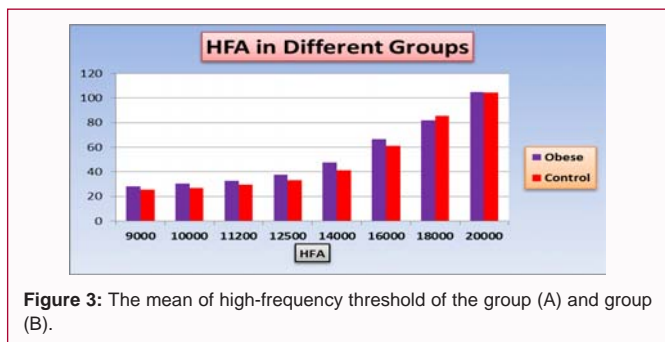


Figure 3: The mean of high-frequency threshold of the group (A) and group (B).

Table 3: Mean and SD of pure tone threshold of group (A) and group (B).

	Obese (n=58)	Control (n=20)	P value
PTA 0.25	17.84 ± 3.75	17.5 ± 3.44	0.719
Mean ± SD			
PTA 0.5	16.21 ± 4.01	17 ± 2.99	0.421
Mean ± SD			
PTA 1	16.12 ± 3.38	16.5 ± 2.85	0.655
Mean ± SD			
PTA 2	16.63 ± 3.55	16.5 ± 3.28	0.879
Mean ± SD			
PTA 4	16.46 ± 3.74	17.25 ± 3.43	0.412
Mean ± SD			
PTA 8	18.87 ± 4.08	19 ± 2.61	0.902
Mean ± SD			

regarding Laboratory investigations as shown in Table 2.

As regards the audiological evaluation, there is no statistically significant difference ($P > 0.05$) between the 2 groups as seeing PTA in all frequencies (from 0.25 to 8) as shown in Table 3 and Figure 2, HFA, the same as PTA showing no statistically significant difference ($P > 0.05$) between the 2 groups regarding HFA in all frequencies (from 9,000 to 20,000) as shown in Table 4 and Figure 3.

However, there is some percentage of ears in both groups with no response at 18 KHz and 20 KHz. There is a statistically significant difference ($P < 0.001$) between the two groups, in this percent, only at 20,000 frequencies as shown in Table 5 and Figure 4.

Transient OAE is showing a statistically significant difference ($P < 0.001$) between both groups as shown in Table 6 and Figure 5 while DPOAE is showing no statistically significant difference between both groups as all ears with normal (pass) response in both groups.

There is a statistically significant correlation between OAE and

Table 4: Mean and SD of High-frequency threshold of the group (A) and group (B).

	Obese (n=58)	Control (n=20)	P value
HFA 9000	28.36 ± 8.81	25.75 ± 7.65	0.241
Mean ± SD			
HFA 10000	30.34 ± 8.82	26.75 ± 7.65	0.109
Mean ± SD			
HFA 11200	32.67 ± 9.51	29.5 ± 5.82	0.166
Mean ± SD			
HFA 12500	37.58 ± 10.72	33.25 ± 8.62	0.107
Mean ± SD			
HFA 14000	47.75 ± 12.91	41.25 ± 14.76	0.065
Mean ± SD			
HFA 16000	66.72 ± 17.78	61.25 ± 19.45	0.25
Mean ± SD			
HFA 18000	81.98 ± 19.86	85.75 ± 19.81	0.467
Mean ± SD			
HFA 20000	105.17 ± 14.95	104.5 ± 10.87	0.854
Mean ± SD			

Table 5: The percentage of NR at (18000 & 20000) frequencies between the two groups.

No response (NR)	Obese	Control	P Value
HFA18000	8 (13.8%)	3 (15%)	0.894
HFA20000	35 (60.3%)	7 (35%)	0.049*

Table 6: Mean of Transient & Distortion OAE of the group (A) and group (B).

Transient OAE	Obese (n=58)	Control (n=20)	P value
Abnormal (Refer)	34 (58.6%)	1 (5%)	<0.001*
Normal (pass)	24 (41.4%)	19 (95%)	

Table 7: Correlation between BMI & OAE.

	OAE	
BMI	R-value	-0.284
	P value	0.012*

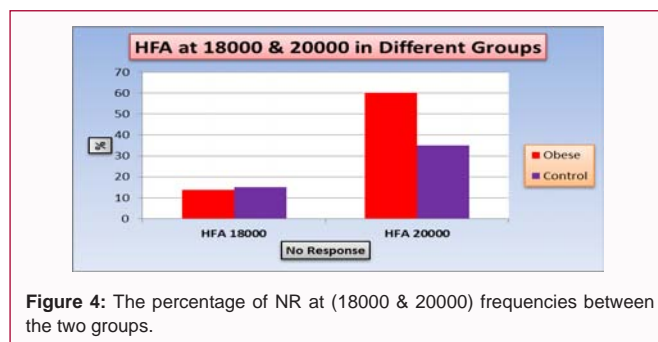


Figure 4: The percentage of NR at (18000 & 20000) frequencies between the two groups.

Body Mass Index (BMI). This correlation was negative, when BMI increase, the normal response to OAE (The Pass response) decrease as revealed from the Table 7.

Discussion

Kim et al. [10], reported that childhood obesity might be associated with otitis media with effusion, However, in the current study, there is no statistically significant difference between both

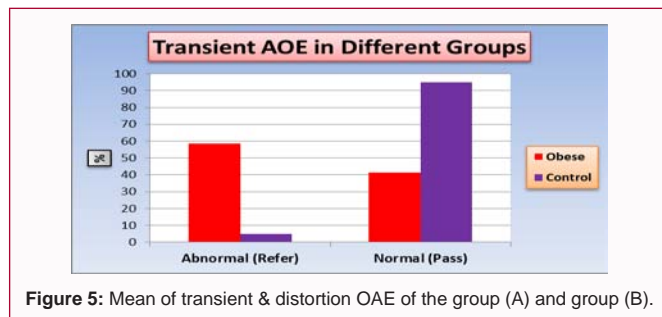


Figure 5: Mean of transient & distortion OAE of the group (A) and group (B).

groups neither in tympanogram nor acoustic reflex as both groups have type (A) tympanogram and intact acoustic reflex with the normal acoustic reflex threshold, reflecting normal middle ear function in both groups.

As regards the hearing sensitivity, there is no statistically significant difference in pure tone audiometry between both groups. Which is not matched with Lalwani et al. [11], whom reported that obesity in childhood is associated with high hearing thresholds across all frequencies in pure tone audiometry. This difference might be due to different age range in either study. In Lalwani study they use older age range from 12 years to 19 years, while in current study from 6 years to 17 years. Beside it could be attributed to the different method of calculation of hearing threshold. In our study, we examine each frequency separately while Lalwani examines the average hearing threshold.

Obesity leads to excessive formation of adipose tissue. Adipose tissue in obesity not act as just a simple tissue but serves as endocrine gland as it secretes offensive adipokine as cytokine and interleukin in all tissues of the body which enter almost all tissues in the body in a state of chronic inflammation Andrew and Martin [12]. Rosenhall and Sundh [13], and Evans et al. [14], reported that the comorbidities of obesity as diabetes hyperlipidemia had been reported to be negatively associated with the peripheral hearing function. However, in the current study, there is no statistical difference between both groups in blood glucose level or blood lipid profile. So in this study, the systemic effect of obesity was not affecting the cochlear either directly or indirectly [15].

Adipose tissue in obesity leads to excessive secretion of offensive adipokines as cytokines while decreasing protective adipokines as adiponectin. There is a close correlation between adiponectin deficiency and increase hearing threshold. Adiponectin knock-out in mice 15 leads to endothelial changes in the wall of cochlear blood vessels so lead to decrease blood flow in stria-vascularis. This result could explain why there is more abnormal response in OAE in the obese group. Adiponectin knock-out also leads to cochlear hair cells damage especially in the basal turn of the cochlea which leads to cochlear apoptotic activity; this result could explain the absent response in 20,000 KHz in obese children.

Obesity is not a simple disease but is epidemic of the 21st century Andrew and Martin [12]. So, early detection of any subtle affection

of the cochlea must be detected. So, screening of obese children is mandatory even if there is no complaint as regards hearing sensitivity. There is future ongoing research to determine the blood level of adiponectin of obese children and if adiponectin supplementation in obese children could be used for the prevention and management of hearing loss.

References

- Ogden C, Carroll M, Kit B, Flegal KM. Prevalence of obesity and trends in body mass index among US children and adolescents, 1999-2010. *JAMA*. 2012;307(5):483-90.
- Sturm R. The effects of obesity, smoking, and problem drinking on chronic medical problem and health care costs. *Health Aff (Millwood)*. 2002;21(2):245-53.
- Kishida K, Funahashi T, Shimomura I. Adiponectin as a routine clinical biomarker. *Best Pract Res Clin Endocrinol Metab*. 2014;28(1):119-30.
- Olsen K, Danielsen K, Wilsgaard T, Sangvik M, Sollid J, Thune I, et al. Obesity and *Staphylococcus aureus* nasal colonization among women and men in a general population. *PLoS One*. 2013;8(5).
- Kemp DT. Stimulated acoustic emissions from within the human auditory system. *J Acoust Soc Am*. 1978;64(5):1386-91.
- Arts HA. Influence of perilymphatic tetrodotoxin and calcium concentration on hair cell function. *Assoc Res Otolaryngol Abstr*. 1990;13:194.
- Dallos P, Harris D. Properties of auditory nerve responses in the absence of outer hair cells. *J Neurophysiol*. 1978;41(2):365-83.
- Ress BD, Sridhar. KS, Balkany TJ, Waxman GM, Stagner BB, Lounsbury-Martin BL. Effects of cis-platinum chemotherapy on otoacoustic emissions: The development of an objective screening protocol. Third place-resident clinical science award 1998. *Otolaryngol Head Neck Surg*. 1999;121(6):693-701.
- Van der Hulst RJ, Dreschler WA, Urbanus NA. High-frequency audiometry in prospective clinical research of ototoxicity due to platinum derivatives. *Ann Otol Rhinol Laryngol*. 1988;97(2 Pt 1):133-7.
- Kim JB, Park DC, CHaCI, Yeo SG. Relationship between pediatric obesity and otitis media with effusion. *Arch Otolaryngol Head Neck Surg*. 2007;133(4):379-82.
- Lalwani AK, Katz K, Liu YH, Kim S, Weitzman M. Obesity is associated with a sensorineural hearing loss in adolescents. *Laryngoscope*. 2013;123(12):3178-84.
- Greenberg AS, Obin MS. Obesity and the role of adipose tissue in inflammation and metabolism. *Am J Clin Nutr*. 2006;83(2):461S-5.
- Rosenhall U, Sundh V. Age-related hearing loss and blood pressure. *Noise Health*. 2006;8(31):88-94.
- Evans MB, Tonini R, Shope CD, Oghalai JS, Jerger JF, Insull W Jr., et al. Dyslipidemia and auditory function. *Otol Neurotol*. 2006;27(5):609-14.
- Tanigawa T, Shibata R, Ouchi N, Kondo K, Ishii M, Katahira N, et al. Adiponectin deficiency exacerbates age-related hearing impairment. *Cell Death Dis*. 2014;5(4):e1189.