Extended High Frequency Audiometry and Otoacoustic Emissions in Children with History of Otitis Media with Effusion

Original Article

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ABSTRACT

Introduction: The most common cause of hearing loss in infants and young children is Otitis media with effusion (OME), which is usually conductive. (OME) is an inflammation of the middle ear cavity associated with type [B] tympanogram with accumulation of effusion which can be serous, purulent, mucoid or a mixture of these fluids. Several methods were used to detect the effect of otitis media on auditory system.

Aim of the Study: To study the effect of long standing OME on cochlear function using Extended high frequency audiometry (EHF), Transient otoacoustic emissions (TOAEs), Distortion product otoacoustic emissions (DPOAEs) in children after resolution of OME.

Subjects and Methods: This work included two groups: control and study. This Control group included 20 children. Study group consisted of 40 children divided in to subgroup A (surgically treated group) and subgroup B (medically treated group), the hearing was evaluated using pure tone audiometry, speech audiometry and extended high frequency audiometry. The cochlear function was evaluated by using TEOAEs and DPOAEs.

Results: Showed significant affection in pure tone audiometry and Extended high frequency and also in OAEs between studied groups (subgroups A&B) and control group. On other hand, there was no significant difference between the study group subgroups A and subgroup B.

Conclusions: TEOAEs, DPOAEs and EHF tests are sensitive in identifying changes in cochlear function in children with a history of otitis media.

Key Words: Distortion product otoacoustic emission (DPOAEs), extended high frequency (EHF), otoacoustic emission (OAEs), otitis media with effusion (OME), transient otoacoustic emission (TOAEs).

Received: 25 September 2024, **Accepted:** 17 July 2025.

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ISSN: 2090-0740, 2025

INTRODUCTION

In developed countries, otitis media with effusion (OME) is the most common cause of hearing loss in children, affecting around 80% of preschool children[1]. OME is characterized by the presence of fluid in the middle ear without symptoms of acute ear infection[2]. It is prevalent among young children, with two peaks at the ages of two and five. By the age of 10, 80% of children experience at least one episode of OME[3].

OME, when left untreated for a long time, can lead to serious conditions like adhesive otitis media, disruption of the ossicular chain, retraction pockets, and cholesteatoma. While medical treatment has been commonly used to address this condition, its proven benefits are yet to be established. Surgical options for treatment include myringotomy with or without ventilation tube (VT) insertion, adenoidectomy, or both. There are differing views on the risks and advantages of VT insertion, thus making the management of OME a topic of controversy[4].

Multiple studies have indicated that occurrences of OME in children have notable lasting effects on both the peripheral and central auditory system[5]. Specifically, in relation to the peripheral system, researchers have discovered damage to the outer hair cells located at the cochlea's base. Recent human studies have also revealed that sensory deprivation during developmental periods results in enduring cellular deficiencies in the auditory cortex and decreased behavioral performance[6].

Years after the resolution of OM, an abnormal OAE response can still be identified. This suggests that the disease could have a prolonged impact on the otological and auditory status, contrary to previous beliefs. Therefore, it can be theorized that in children with persistent middle ear disease, OAEs may manifest with reduced amplitude at specific frequencies or could be entirely absent[7].

This study was done to analyze the long-term effect of otitis media in children who had a history of bilateral

DOI: 10.21608/ejentas.2025.323689.1781

OME by measuring their EHF audiometry, TEOAEs and DPOAEs response. Multiple researches had recognized involvement of the basal turn of the cochlea in OME in children, so we designed this research to evaluate whether there is long term cochlear affection after resolving of OME.

SUBJECTS AND METHODS

This work took place at the audio-vestibular medicine unit. Tanta University, after approved by the Research Ethics committee No. 34943/9/21. The idea of the research was explained in details to the parents and participants. Participation was voluntary and subjects may discontinue participation at any time without penalty or loss of benefits. An informed consent was obtained from all parents in this study.

Subjects:

The study included 60 children. Their age ranged from 4-18 years divided into two groups. The Control group which included 20 children with normal hearing with pure tone air conduction less than or equal to 15 dB HL at all audiometric tested frequencies from 250 Hz to 8000 Hz. The Study group included 40 children that had documented history of otitis media with effusion two episodes or more. Twenty subjects were managed by medical treatment and 20 were managed by surgical treatment. At the time of testing all children were having normal middle ear function determined by type (A) tympanograms and acoustic reflex at expected levels at 500,1000,2000 and 4000 Hz in both ears.

Inclusion criteria of the study group were age ranged from 4-18 years, no systemic diseases (e.g. any endocrinal, vascular, renal or neurological complaints) that can affect hearing. All children in this group should have a past history of otitis media with effusion (OME) two episodes or more.

Exclusion criteria of the study group were children with active middle ear disease or OME in time of testing, general health problems (e.g. any endocrinal, vascular, renal or neurological complaints) can affect hearing, congenital anomalies and syndromes. Also, history of sensorineural hearing loss (SNHL) before complaint of OME or positive family history of hereditary hearing loss.

Methods:

Equipment:

- Sound treated room: locally manufactured.
- Pure tone audiometry: Type GSI 61 clinical audiometry.
- Immittancemetry: interacoustic (AT235).
- Otoacoustic Emission (OAEs): Smart Intelligent Hearing System.

Procedure:

All subjects in this study were submitted to full audiological history, Otological examination, Pure tone audiometry, Extended high frequency up to 20.000 Hz, Speech audiometry including Speech Recognition Threshold (SRT) using Arabic bisylabic words for children and word discrimination score using Arabic Phonetically Balanced Words for children (PB-KG) (Elmahallawi *et al*.,1984)[8] and Immittancemetry including tympanometry and acoustic reflex threshold determination ipsilaterally and contralaterally in frequencies 500,1000, 2000 and 4000Hz.

Otoacoustic Emissions also were done including Transient Otoacoustic Emissions (TOAEs) a soft disposable probe tip was gently inserted into the outer portion of external ear canal using clicks stimulus (a wide band), 30 dB SPL and response was assessed by frequency using Fourier analysis function and the signal averaging methodology, the result was displayed on a screen as five frequencies 1000, 1500, 2000, 3000, 4000Hz. The signal to noise ratio and amplitude of each frequency was recorded.

Distortion Product of Otoacoustic Emissions (DPOAE) using a soft disposable probe tip which was gently inserted into the outer portion of external ear canal. Two pure tones were used slightly differ in frequency (usually f1/f2 = 1.20–1.25), and the OAE signal is measured at the third frequency f3 = 2f1 – f2, where f2 > f1. L1=65 dB and L2=55 dB stimulus frequencies were from 500 Hz to 8000 Hz at 10 frequencies /octave. OAE tests were performed in a quiet room.

The fundamental step was to make sure the noise levels in the external ear canal were at least lower than the 95th percentile for a normal subject before we even get started. (to get the noise down to at least -10 dB). If they were repeatable and OAE-to-noise-floor difference of at least 6 dB. It simply means they were present. Then comparing the OAE findings to normative data for that equipment. OAE itself was thought to have an amplitude that may vary from -10 dB SPL to +30 dB SPL in healthy ears.

RESULTS

The Control group consisted of 20 children (8 males and 12 females). Their age ranged from 4-9 years with mean and SD 5.21±1.44 years. The Study group consisted of 40 children. They were divided in two sub groups: Subgroup (A): Surgically treated by grommet tube insertion consisted of 20 children, 7 males and 13 females. Their age ranged from 4 to 6 years with mean and SD 4.80±0.77 years with mean numbers of episodes of otitis media 2.55±0.51, duration between surgery and assessment was 11 to 17 months, with mean 14.10±2.08 months. Subgroup (B):

Medically treated patients consisted of 20 children, they were 11 males and 9 females. Their age ranged from 4 to 7 years with mean 4.80+0.83 years, with mean number of episodes of otitis media 2.65±0.59 and the last attack of otitis media was 7-11 months (Table 1).

Table 1: Age and sex distribution among different groups:

Sociodemographic	Subgroup(A) (*N*=20)		Subgroup(B) (*N*=20)		control group (*N*=20)		*F* value	*P* value
Age								
Mean±SD	4.800.77±		4.80 ±0.83		5.211.44±		=0.987	0.379
Range	4-6		4 - 7		4 - 9			
	N	%	N	%	N	%	χ2	
Sex								0.414
Male	7	35.0	11	55.0	8	40.0	1.765	
Female	13	65.0	9	45.0	12	60.0		

F: One Way Analysis of Variance (ANOVA); χ2=chi square test for comparing between the studied groups.

PTA and extended high frequency results:

Comparison of PTA threshold between Rt and Lt ears in each group was done at each frequency and was insignificant (P≥0.05). Regarding pure tone average, the mean of PTA was 14.81+0.47 dB HL, 17.10+2.01dB HL and 17.40+1.94 dB HL at control group, Subgroup(A) and subgroup (B) respectively. By ANOVA test, there was a statistically significant difference between studied groups and post Hoc test showed a significant difference between the control group and subgroups A&B. There was no significant difference between both study subgroups A&B (Table 2).

Table 2: ANOVA and Post Hoc tests for Comparison of PTA average between studied subgroups and control groups:

Studied Groups	Pure tone audiometer average** (PTA)"dB"						ANOVA	
	Mean±SD			Range			*F*	*P*
Subgroup(A)	17.10	±	2.01	13	-	20		
Subgroup(B)	17.40	±	1.94	14	-	20	14.979	0.001*
Control group	14.81	±	0.47	13	-	15		
Post Hoc	A& B			C&A			C&B	
	1.000			0.001*			0.001*	

**Average of all frequency in both right and left ear. A: Surgically treated group; *P*≤0.05: significant; B: medically treated group; *P*≤0.001: Highly significant; C: healthy control group.

The PTA threshold of each frequency from 250 to 20000 Hz was done and comparison between each frequency among studied groups was done using ANOVA test. There was a statistically significant difference. Post Hoc test was done. There was statistically significant difference between study subgroups and control group and there was no statistically significant difference between both study subgroups (Tables 3,4).

Speech audiometry results

Comparison of speech recognition & speech discrimination tests between Rt and Lt ears was done at each group. There was no significant difference so, we add both ears, The mean of SRT was13.88+2.42, 14.75+1.12 and 17.00+2.51. at control group, subgroup (A) and subgroup (B) respectively. ANOVA test showed a significant difference between studied groups, post Hoc

test showed a significant difference between control group and subgroups A&B. There was no significant difference between both study subgroups. As regard SD%, it was 100% at all cases of control, study subgroups.

Table 3: ANOVA and Post Hoc tests for comparison of PTA at each frequency among the studied groups:

	Groups	Mean± SD			Range			ANOVA		Post Hoc	
								F	P		
250 Hz	Subgroup(A)	17.25	±	1.80	15	-	20			A& B	1.000
	(Subgroup(B	16.88	±	1.79	15	-	20	13.6	0.001*	C& A	0.001*
	Control group	15.00	±	٠.00	15	-	15			C& B	0.001*
٥٠٠Hz	Subgroup(A)	17.25	±	1.97	١٢	-	20			A& B	1.000
	Subgroup(B)	17.00	±	1.92	15	-	20	12.1	*0.001	C&A	0.001*
	Control group	15.00	±	.00	15	-	15			C& B	0.001*
1000 Hz	Subgroup(A)	15.88	±	3.47	10	-	20			A& B	0.291
	Subgroup(B)	17.13	±	2.03	13	-	20	4.6	0.014*	C& A	0.001*
	Control group	14.88	±	0.56	13	-	15			C& B	0.011*
2000 Hz	Subgroup(A)	16.38	±	3.39	10	-	20			A& B	1.000
	Subgroup(B)	16.88	±	2.67	10	-	20	4.2	0.021*	C& A	0.001*
	Control group	14.63	±	1.22	10	-	15			C& B	0.024*
4000 Hz	Subgroup(A)	17.25	±	2.13	13	-	20			A& B	0.827
	Subgroup(B)	16.50	±	2.74	10	-	20	9.6	*0.001	C& A	0.001*
	Control group	14.38	±	1.38	10	-	15			C& B	0.009*
8000 Hz	Subgroup(A)	17.88	±	2.47	13	-	20			A& B	1.000
	Subgroup(B)	17.25	±	2.68	13	-	20	12.9	*0.001	C& A	0.001*
	Control group	14.50	±	1.31	10	-	15			C& B	0.001*

**Average of each frequency in both right and left ear. A: Surgically; treated group; $P \leq 0.05$: significant; B: medically treated group; $P \leq 0.001$: Highly significant; C: healthy control group.

Table 4: ANOVA and Post Hoc tests for comparison of PTA at extended high frequencies among the studied groups:

	Groups	Mean± SD			Range			ANOVA		Post Hoc	
								F	*P*		
10000 Hz	Subgroup(A)	17.13	±	2.47	13	-	20			A& B	1.000
	Subgroup(B)	17.38	±	2.22	13	-	20	11.1	*0.001	C& A	0.001*
	Control group	14.63	±	1.22	10	-	15			C& B	0.001*
12000 Hz	Subgroup(A)	17.63	±	2.36	13	-	13			A& B	1.000
	Subgroup(B)	17.88	±	2.33	15	-	15	13.8	*0.001	C& A	0.001*
	Control group	15.00	±	.00	15	-	15			C& B	0.001*
14000 Hz	Subgroup(A)	17.00	±	2.76	10	-	20			A& B	0.296
	Subroup(B)	18.13	±	2.42	15	-	20	11.2	*0.001	C& A	0.013*
	Control group	15.00	±	.00	15	-	15			C& B	0.001*
16000 Hz	Subgroup(A)	16.88	±	2.80	10	-	20			A& B	0.320
	Subgroup(B)	18.00	±	2.51	15	-	20	9.8	*0.001	C& A	0.025*
	Control group	15.00	±	.00	15	-	15			C& B	0.001*
18000Hz	Subgroup(A)	17.13	±	2.03	13	-	20			A& B	0.393
	(Subgroup(B	18.00	±	2.38	15	-	20	14.6	*0.001	C& A	0.001*
	Control group	15.00	±	.00	15	-	15			C& B	0.001*
20000Hz	Subgroup(A)	17.50	±	2.29	15	-	20			A& B	1.000
	Subgroup(B)	18.00	±	2.38	15	-	20	14.2	*0.001	C& A	0.001*
	Control group	15.00	±	.00	15	-	15			C& B	0.001*

**Average of each frequency in both right and left ear. A: Surgically treated group; $P \leq 0.05$: significant; B: medically treated group; $P \leq 0.001$: Highly significant; C: healthy control group.

Acoustic reflex results:

Comparison of acoustic reflex threshold using t-test between Rt and Lt ears at each group. There was no statistically significant difference. So, we add both ears. ANOVA test was done between studied groups for ipsilateral and contralateral acoustic reflex test at frequencies 500,1000,2000and 4000 Hz, there was no statistically significant difference.

TOAEs results:

Comparison between Rt and Lt ears results of amplitude and SNR OF TOAEs was done. There was no significant difference. So, we add both ears. ANOVA test was done among groups to compare amplitude of TOAEs. There was a statistically significant difference at frequencies 1500,2000 ,3000 and 4000 Hz. Post Hoc test was done. There was a significant difference between subgroup (A) and control group, subgroup (B) and control group at 1500,2000, and 4000Hz, and also between subgroup (B) and control group at 3000Hz (Table 5).

ANOVA test was done to compare SNR of TOAEs among the three groups there was significant difference at all frequencies, Post Hoc test was done, there was significant difference between subgroup (A), subgroup (B) and control group at frequencies 1000,1500,2000 and 4000Hz. There was also a significant difference between subgroup (B) and control group at 3000Hz (Table 6).

Table 5: ANOVA AND Post Hoc tests for comparison of amplitude of TOAEs among studied groups:

	Groups	Mean± SD			Range			ANOVA		Post Hoc	
								F	*P*		
1000	Subgroup(A)	8.22	±	4.37	1.89	-	17.03			A& B	
	(Subgroup(B	7.04	±	4.33	0.44	-	19.53	1.3	0.272	C& A	
	Control group	6.02	±	4.12	0.06	-	14.32			C& B	
15000 average	Subgroup(A)	10.03	±	6.05	0.84	-	21.47			A& B	1.000
	Subgroup(B)	9.72	±	5.34	0.75	-	20.32	5.4	0.007*	C& A	.014*
	Control group	5.31	±	3.42	0.66	-	13.76			C& B	.024*
20000 average	Subgroup(A)	15.20	±	4.96	8.24	-	26.13			A& B	1.000
	Subgroup(B)	15.32	±	5.30	4.68	-	25.42	6.2	0.004*	C& A	.012*
	Control group	10.62	±	4.08	2.14	-	17.58			C& B	.009*
3000	Subgroup(A)	20.22	±	4.20	13.3	-	27.42			A& B	0.577
	Subgroup(B)	22.60	±	6.73	8.28	-	33.35	3.5	0.037*	C& A	0.573
	Control group	17.83	±	5.89	8.93	-	29.43			C& B	0.032*
4000	Subgroup(A)	20.23	±	4.36	9.99	-	26.76			A& B	1.000
	Subgroup(B)	20.07	±	6.80	8.98	-	29.44	6.8	0.002*	C& A	.006*
	Control group	13.99	±	6.87	2.03	-	28.65			C& B	.008*

**Average of each frequency in both right and left ear. A: Surgically treated group; $P \leq 0.05$: significant; B: medically treated group; $P \leq 0.001$: Highly significant; C: healthy control group.

Table 6: ANOVA and Post Hoc test for comparison of SNR of TOAEs among studied groups:

	Groups	Mean±SD			Range			ANOVA		Post Hoc	
								F	*P*		
1000 average	Subgroup(A)	5.05	±	2.29	1.12	-	8.60			A& B	1.000
	(Subgroup(B	4.60	±	3.10	0.98	-	13.13	24.6	0.001*	C& A	0.001*
	Control group	9.68	±	2.10	6.60	-	14.45			C& B	0.001*
15000 average	Subgroup(A)	6.52	±	3.82	0.91	-	12.64			A& B	1.000
	Subgroup(B)	6.33	±	3.14	1.33	-	11.37	12.9	0.001*	C& A	0.001*
	Control group	10.99	±	2.84	7.32	-	18.47			C& B	0.001*
20000 average	Subgroup(A)	6.83	±	5.18	0.33	-	16.55			A& B	1.000
	Subgroup(B)	7.16	±	3.65	1.54	-	11.49	8.7	0.001*	C& A	0.001*
	Control group	11.47	±	2.46	8.09	-	16.19			C& B	0.003*
3000 average	Subgroup(A)	7.56	±	4.46	1.53	-	16.00			A& B	0.773
	Subgroup(B)	6.22	±	3.94	1.03	-	13.01	5.7	0.006*	C& A	0.103
	Control group	10.09	±	2.33	7.07	-	15.96			C& B	0.005*
4000	Subgroup(A)	6.00	±	3.85	0.30	-	13.88			A& B	1.000
	Subgroup(B)	6.88	±	4.91	0.30	-	18.37	8.6	0.001*	C& A	0.001*
	Control group	10.84	±	2.71	6.29	-	14.97			C& B	0.007*

**Average of each frequency in both right and left ear. A: Surgically treated group; $P \leq 0.05$: significant; B: medically treated group; $P \leq 0.001$: Highly significant; C: healthy control group.

Results of DPOAEs:

Comparison between Rt and Lt ears results of distortion product and SNR, there was no statistically significant difference, so add both ears. Comparison between subgroup (A), subgroup (B) and control group regarding distortion product for each frequency average was done using ANOVA test. There was a significant difference at frequencies 499,704,1003,1409,2000,2822,3991 and 5649 Hz and post Hoc test was done. There was a significant difference between subgroup (A) and, subgroup (B) and control group at frequencies 704,1003,1409,2000,2822,3991Hz. Also, there was a significant difference between subgroup (B) and control group at 499, 5649Hz (Table 7).

Table 7: ANOVA and Post Hoc test for comparison of distortion product of DPOAEs among studied groups:

	Groups	Mean± SD			Range			ANOVA		Post Hoc	
								F	*P*		
375 average	Subgroup(A)	8.98	±	4.47	2.50	-	22.00			A& B	
	Subgroup(B)	8.78	±	4.28	2.00	-	16.50	2.3	0.108	C& A	
	Control group	11.15	±	2.58	7.00	-	17.50			C& B	
499 average	Subgroup(A)	8.90	±	3.42	4.00	-	15.50			A& B	1.000
	Subgroup(B)	8.45	±	3.51	2.00	-	14.00	3.9	0.025*	C& A	0.101
	Control group	11.10	±	2.57	7.50	-	17.00			C& B	0.033*
704	Subgroup(A)	7.93	±	4.12	0.50	-	15.00			A& B	1.000
	Subgroup(B)	8.45	±	4.68	2.00	-	17.50	7.6	0.001*	C& A	0.002*
	Control group	12.65	±	3.76	7.00	-	22.00			C& B	0.008*
1003 average	Subgroup(A)	7.20	±	4.49	1.50	-	19.00			A& B	0. 640
	Subgroup(B)	8.93	±	5.26	0.50	-	17.50	8.8	0.001*	C& A	0.001*
	Control group	12.80	±	2.93	8.00	-	20.00			C& B	0.019*
1409 average	Subgroup(A)	6.65	±	4.24	1.50	-	16.50			A& B	1.000
	Subgroup(B)	7.05	±	4.23	2.00	-	14.00	6.3	0.004*	C& A	0.006*
	Control group	10.35	±	1.93	7.50	-	15.50			C& B	0.017*
2000	Subgroup(A)	5.50	±	3.26	0.50	-	13.50			A& B	0.436
	(Subgroup(B	4.38	±	1.84	1.00	-	7.00	28.8	0.001*	C& A	0.001*
	Control group	9.85	±	1.84	6.00	-	13.50			C& B	0.001*
2822	Subgroup(A)	4.80	±	1.86	2.00	-	8.00			A& B	1.000
	Subgroup(B)	4.48	±	2.44	1.00	-	11.00	44	0.001*	C& A	0.001*
	Control group	10.23	±	2.19	7.00	-	15.00			C& B	0.001*
3991	Subgroup(A)	6.53	±	4.25	2.00	-	17.00			A& B	1.000
	Subgroup(B)	6.55	±	2.32	2.00	-	12.00	7.5	0.001*	C& A	0.004*
	Control group	9.73	±	1.87	6.50	-	14.00			C& B	0.004*
5649	Subgroup(A)	7.45	±	5.19	1.50	-	23.50			A& B	0.719
	Subgroup(B)	5.88	±	4.32	1.00	-	17.50	4.7	0.012*	C& A	0.201
	Control group	9.93	±	2.68	6.00	-	17.50			C& B	0.010*

**Average of each frequency in both right and left ear. A: Surgically treated group; $P \leq 0.05$: significant; B: medically treated group; $P \leq 0.001$: Highly significant; C: healthy control group.

Comparison between subgroup (A), group (B) and control group regarding SNR of DPOAE, was done by ANOVA test. There was a significant difference at all frequencies. Post Hoc was significant between subgroup (A) and subgroup (B) and control group at 375, 499,704, 10031409, 2000 and 2822 Hz. There was a significant difference between subgroup (A) and control group at 3991 Hz, and subgroup (B) and control group at 5649Hz (Table 8).

There was no correlation between PTA (basic &extended high frequency), acoustic reflex threshold &results of OAEs (TEOAEs & DPOAEs) and number of episodes in study subgroups (A&B).

Correlation was also done between all tests and duration between time of surgery and time of examination. There was a negative correlation of moderate degree between duration and threshold of PTA at all extended high frequencies in subgroup (A). There was no correlation between all tests and duration between last attack and examination of otitis media in subgroup (B).

Table 8: ANOVA and Post Hoc test for comparison of SNR of DPOAEs among studied groups:

	Groups	Mean± SD			Range			ANOVA		Post Hoc	
								F	*P*		
375 average	Subgroup(A)	5.45	±	2.10	1.50	-	9.00			A& B	0.392
	(Subgroup(B	6.95	±	4.09	1.00	-	17.00	15.8	0.001*	C& A	0.001*
	Control group	10.78	±	2.75	6.00	-	16.50			C& B	0.001*
499 average	Subgroup(A)	6.83	±	3.44	3.00	-	19.00			A& B	1.000
	Subgroup(B)	7.45	±	2.61	3.00	-	14.50	8.9	0.001*	C& A	0.001*
	Control group	10.40	±	2.45	5.00	-	14.50			C& B	0.006*
704 average	Subgroup(A)	7.13	±	3.89	1.50	-	15.00			A& B	1.000
	Subgroup(B)	8.23	±	3.92	3.00	-	19.50	11.2	0.001*	C& A	0.001*
	Control group	12.48	±	3.49	5.00	-	22.00			C& B	0.002*
1003 average	Subgroup(A)	7.33	±	3.84	2.00	-	15.50			A& B	0.355
	Subgroup(B)	9.33	±	4.72	3.00	-	21.00	10.7	0.001*	C& A	0.001*
	Control group	13.08	±	3.27	8.50	-	20.50			C& B	0.013*
1409 average	Subgroup(A)	6.75	±	4.08	2.50	-	15.00			A& B	0.355
	Subgroup(B)	8.73	±	3.42	3.50	-	16.00	6.	0.002*	C& A	0.001*
	Control group	11.03	±	3.35	6.50	-	17.50			C& B	0.013*
2000	Subgroup(A)	6.68	±	3.50	0.50	-	13.50			A& B	0.706
	(Subgroup(B	5.63	±	2.00	3.50	-	9.50	18.3	0.001*	C& A	0.001*
	Control group	10.65	±	2.60	6.00	-	14.50			C& B	0.001*
2822	Subgroup(A)	5.40	±	1.92	2.50	-	9.00			A& B	1.000
	Subgroup(B)	5.68	±	2.30	2.50	-	12.50	25.9	0.001*	C& A	0.001*
	Control group	10.03	±	2.58	5.50	-	16.00			C& B	0.001*
3991	Subgroup(A)	7.63	±	3.39	2.00	-	15.50			A& B	1.000
	Subgroup(B)	8.33	±	3.18	4.50	-	16.00	3.8	0.030*	C& A	0.032*
	Control group	10.10	±	2.16	7.00	-	15.00			C& B	0.189
5649	Subgroup(A)	9.18	±	6.37	2.50	-	28.50			A& B	0.958
	Subgroup(B)	7.60	±	3.99	3.50	-	16.50	3.2	0.048*	C& A	0.406
	Control group	11.55	±	4.17	5.50	-	23.00			C& B	0.044*

**: Average of each frequency in both right and left ear; A: Surgically treated group; $P \leq 0.05$: significant; B: medically treated group; $P \leq 0.001$: Highly significant; C: healthy control group.

DISCUSSION

There have been suggestions that inflammatory processes in the middle ear could lead to temporary or permanent sensorineural hearing loss in association with inner ear complications, resulting in the loss of outer hair cells from the basal turn of the cochlea. This could be due to the movement of antibiotics, toxins, or macromolecules from the middle ear into the perilymph through the semipermeable round window membrane. Furthermore, reactive inflammatory cells have been discovered in the perilymph close to the round window membrane[9].

OME might be the cause of hidden hearing loss, which could impair sound localization and alter the processing of auditory information. It is therefore highly recommended that patients with OME, who have normal audiograms, be monitored electro acoustically and electro physiologically to insure good audiological monitoring and intervention. The aim of the present study was to study the effect of long standing OME on cochlear function using EHF, TOAEs and DPOAEs in children after resolving of OME. The high sensitivity of the OAE test probably explains why OAE abnormalities could be detected even though other tests are normal[10]. Also, Extended High-frequency audiometry had been used to monitor episodes of aggravated OME, and can pick up the effects of OME on cochlear function[11].

In the present study, the number of females in both the control group and the surgical group was higher than males. Previous studies performed in children with a history of OME have shown a higher prevalence of pathology in females may due to genetic factors[12].

On the other hand, no significant difference in PTA between medically and surgically treated patients (subgroups A and B). Potentially adverse effects on the tympanic membrane are common after grommet insertion.

Thus, for the majority of children with a history of OME, an initial period of watchful waiting appears to be an appropriate management strategy. The clinician will need to base treatment decisions for these children on other evidence and indications of disability related to hearing impairment, as there is currently no evidence available for the subgroups of children with behavioral and learning problems, children with defined clinical syndromes, or children with speech or language delays[4].

PTA threshold in patients with previous history of OME (subgroups A and B) was significantly higher than normal hearing subjects. Analyzing the mean responses of control group, subgroup A and sub group B at frequencies from 250 Hz to 8000Hz, there was significant difference in hearing thresholds. Thus, it was found that OME in the subgroup A and subgroup B had caused a long-term negative effect despite the middle ear had recovered after the course of the disease[13].

In the current study, no significant difference in PTA between medically and surgically treated patients (subgroups A and B). In another study on children with recurrent OME with or without undergoing VT insertion in the first year of childhood had comparable hearing outcomes and middle-ear health status to those with no history of the disease. Although children who underwent VT insertion had an increased risk of abnormal middle-ear status and some elevation in hearing levels in their ear only, their audiometric results were still within normal limits, indicating that the impact of VT insertion in early childhood is unlikely to have clinically significant adverse impact on later hearing outcomes[14].

Speech discrimination was 100% for all groups and SRT mean was 17 in subgroup A, subgroup B and 15 in control group, this was agreed with[15].

Children with previous history of otitis media whether treated surgically [subgroup A] or medically (subgroup B) showed significantly higher hearing threshold level at all extended high frequencies than the control[16]. This result agreed with[17] who reported that children who have recovered from chronic OM have significantly poorer hearing in the EHF range compared with children without significant OM history. The EHF hearing losses that occur in children with OM histories are strongly frequency dependent, suggesting a preferential effect on the base of the cochlea. Middle ear impedance and reflectance differences do not account for the EHF hearing losses observed in children with OM history. The results support the hypothesis that OM-related EHF hearing losses are cochlear in origin.

According to Cowan and Wittes, 1994[18], both conventional and HF thresholds were highly impacted by the quantity of middle ear diseases that were currently active. A number of other variables, such as the appearance of the middle ear during intubation, the existence of tympanosclerosis, and the date of the most recent episode of otitis media, were also linked to worse hearing in the higher frequency range (HF) but lost significance when they were correlated with the quantity and frequency of OM.

There was a negative correlation of moderate degree between duration of time of surgery and time of examination and extended high frequency, this may be attributed to improvement in hearing threshold after tube insertion[19].

Specifically, the middle ear had probably not interfered with the responses of TEOAEs or DPOAEs. That is, the generation of otoacoustic emissions is related to the effect of otitis media toxins on the outer hair cells, while the transmission of otoacoustic emissions is related to the path taken through the middle ear, which may be altered due to sequelae in the tympanic–ossicular system[13]. In the present study Otoacoustic emissions was absent in two subjects. The amplitude and SNR of TEOAEs was significantly lower in both study subgroups than the control group.

The results of the present study agreed with[20] who attributed this to loss of outer hair cells at the base of the cochlea, caused by toxic substances permeating from the middle ear to the cochlea through the round window membrane, or by ultra-structural lesions in the inner ear of children with a history of OME.

The results also agreed with[21] who found a statistically significant difference in amplitude and SNR of TEOAEs between studied group and control group. There was robust response in the mean values of the control group compared to the studied group. These results show that TEAOEs are highly sensitive to cochlear changes, implying they can be used to monitor children with a history of OME, especially before and after medical treatment or surgical intervention.

In the present study, the DPOAEs was significantly lower in both study subgroups than normal. No significant difference between surgically treated and medically treated subgroups. This result agreed with[22] who found that DPOAE responses were significantly different at all frequencies tested, with the responses for the control group more robust than those in the studied group. It has already been found that a history of OME has the effect of lowering DPOAE amplitudes.

Campos *et al*.,2012[23] found reduced DPOAE amplitudes at 2002, 3174, and 4004 Hz. However, more work is still needed to clarify to what extent the reduction in TEOAE and DPOAE amplitudes interferes with higher-order auditory tasks. It is already known that episodes of OME in early infancy can have a negative impact on learning and communication[24].

Another study evaluated the functional status of the cochlea and concluded that repeated otitis media more than 2 episodes can cause changes in the peripheral structures of the auditory system. There were no TEOAE responses and DPOAEs response amplitudes were lower at all frequencies. The emission amplitude and the signal-to-noise ratio were statistically different between the two groups, and OAEs in the study group were statistically smaller compared to the control group[25].

Akdogan *et al*., 2006 studied spontaneous otoacoustic emissions (SOAEs), transiently evoked otoacoustic emissions (TEOAEs) and distortion product otoacoustic emissions (DPOAEs) that were recorded from 44 normal ears and 32 ears with past history middle-ear effusion. In 21 ears out of 32 otitis media with effusion (OME) ears, SOAEs, TEOAEs and DPOAEs were absent. In the 28 ears with middle-ear effusion, the response and wave reproducibility were diminished[21].

Tilanus *et al*.,1995[26] and Chang *et al*., 1998[27] studied TEOAE and found that the average band reproducibility below 2 kHz recovered significantly after tube placement whereas the average band reproducibility diminished at 5 kHz.

According to Richardson *et al*., 1996[28], TEOAE can be detected in 50% of ears following grommet insertion, although the responses were less than in normal ears. An increased noise level and myringotomy site bleeding may be linked to absent responses. It could also indicate a more inflammatory middle ear mucosa. However, after surgery, mean DPOAE amplitudes significantly improved, though they were still lower than in the control group, which is likely due to the cochlear affection.

Finally, OME guidelines from 2016 indicated that children who have the condition may experience delayed speech and language, hearing loss, and structural alterations to the tympanic membrane. OME can cause limited vocabulary, inattention, delayed reactions to auditory stimuli and limited vocabulary, all of which can affect a child's academic performance. Furthermore, auditory processing disorder may result from OME's inflicted auditory deprivation during crucial stages of a child's development of the central auditory nervous system[29].

For all these reasons, children with a history of OME should be regularly monitored, even if they have auditory thresholds within normal limits. Any absence of stimulation and/or changes in everyday sound stimuli run the risk of permanently affecting the central auditory nervous system.

CONCLUSION

Pure Tone Audiometry and Extended High Frequency thresholds remain elevated in children with history of Otitis Media with effusion even after resolution with no difference between surgically treated group and medically treated group. Transient Otoacoustic Emissions (TEOAEs) and Distortion Product Otoacoustic Emissions (DPOAEs) are affected in children with history of Otitis Media with effusion denoting hidden cochlear pathology even after resolution of OME.

CONFLICT OF INTERESTS

There are no conflicts of interest.

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