

## **Audiological changes in patients with otitis media with effusion (OME).**

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### **الملخص**

!!!يعد التهاب الأذن الوسطى الأنصباي (OME) مشكلة مرضية شائعة وخاصة في الأطفال وإذا لم يتم تشخيصها ومعالجتها مبكرا وبصورة مناسبة قد تؤدي الى مضاعفات خطيرة. تعلقت الدراسة الحالية ببعض جوانب هذه المشكلة وهي التغيرات السمعية المصاحبة لالتهاب الأذن الوسطى الأنصباي وتحديد التغيرات بالنغمة الصافية فيما يتعلق بنوع نقص السمع أي حسي عصبي أو توصيلي وشدته وتأثر كل تواتر على انفراد. شملت الدراسة خمسين حالة مرضية مشخصة سريريا تراوحت أعمارهم ما بين 4-15 سنة. كان متوسط العتبات الهوائية حوالي 31dBHL في الحالات المدروسة وبمعدل يتراوح ما بين 0-50 دسبل. وكان التواتر 500 Hz هو الأكثر تأثرا والتواتر 2000 Hz هو الأقل تأثرا مقارنة بالتوترات الأخرى فيما يتعلق بنقص السمع التوصيلي. كما وجد التواتر 1000 Hz هو الأكثر تأثرا بالنسبة لنقص السمع الحسي عصبي بينما التواتر 250 Hz هو الأقل تأثرا.

### **Abstract**

Otitis media with effusion (OME) is a common problem especially in children and if not diagnosed early and treated appropriately may lead to serious complications.

Our study was conducted to assess the audiological changes associated with OME regarding the type, intensity and each frequency affected. The study involved 50 patients and the age ranged from 4-15 years old. The average of air conduction thresholds in the submitted cases (conductive deafness) were 31dBHL in the range between (0-50) dBHL. 500Hz is the most frequency affected and the least affected one is 2000Hz. In bone conduction thresholds (sensorineural hearing loss), we found that the frequency 1000Hz is the most affected one, while the frequency 250Hz is the least affected.

### **Introduction**

Otitis media with effusion (OME) is a middle ear effusion (MEE) without signs & symptoms of infection <sup>(1)</sup>. The diagnosis of OME is difficult because the process is often asymptomatic, so-called silent otitis media <sup>(2)</sup>. Several investigators have shown that a high incidence of persistent MEE after an episode of acute otitis media <sup>(3)</sup>. In OME, the middle ear cleft contains fluid that decreases tympanic membrane mobility & this will result in a conductive hearing loss <sup>(4)</sup>, the conductive loss is maximal in the low frequencies <sup>(5)</sup>. It is estimated on screening studies that 17-41% of children between 2-3 years of age were found to have OME during a three months screening period <sup>(6)</sup>.

Many sequential studies have reported that between 20-50% of children will have an episode of OME at some time between the ages of 3 and 10 years <sup>(5)</sup>. The Eustachian tube has the functions of pressure regulation, protection and clearance <sup>(3)</sup>. Eustachian tube has a major role in the development of OME, Eustachian tube dysfunction exists when the tube does not open properly, is not patent, or is blocked in one way or another <sup>(7)</sup>. Eustachian tube obstruction may result from either intra or extraluminal forces that preclude tubal opening <sup>(6)</sup>. Cleft palate, craniofacial anomalies, congenital or acquired immune deficiencies, ciliary dysfunction, enlarged adenoids, sinusitis & Down syndrome may predispose a child to otitis media in general including otitis media with effusion <sup>(8)</sup>. OME may or may not be associated with a significant hearing loss & there is no hard evidence to correlate the effect of intermittent or even long-term hearing loss with speech or language development later in life <sup>(9)</sup>. The effect of the hearing loss produced by OME on speech, language, & cognitive development has been hotly debated. Studies show conflicting results & long-term adverse effects in the typical patient are likely to be subtle at most <sup>(10)</sup>. Some studies demonstrate evidence that even minimal or mild hearing losses in young children can impair speech and language development <sup>(3, 4)</sup>, & because a child cannot hear, he is likely to be labeled 'disobedient' or 'inattentive' both at home & at school <sup>(11)</sup>. The problem is often first noticed by a schoolteacher or visiting relative, but many cases are only discovered during routine audiometric screening <sup>(12)</sup>. The aim of the study was to study the type, intensity and each frequency affected in otitis media with effusion (OME).

### **Patients and Methods**

The study was conducted in Diwanayah Teaching Hospital & my private clinic. Fifty patients were clinically diagnosed with otitis media with effusion (OME) between July 2007 and December 2008 (28 males & 22 females). The reviewed data in the questionnaire form were age, sex, residence, duration of the disease, hearing impairment, speech delay, school delay, and ear fullness. Age of the patients in the study ranged from 4-15 years old. A control group of 24 (48 ears) age & gender-matched healthy persons were chosen randomly. Diagnosis was relying on clinical symptoms & signs where hearing impairment was the most important and most common symptom, microscopic & otoscopic examination with help of Siegle's speculum (the microscope used in our study was Hallpike Blackmore ear microscope & Karl Zeiss ear microscope), Pure tone audiogram (PTA), myringotomy & aspiration & tympanometry.

**Results**

Age of the patients in our study ranged from 4-15 years old, and the results showed that the most frequent age group affected was sixth years old then the fifth years old, the average age was 7.5 years old (table 1).

**Table -1: Distribution of patients with OME according to age**

Age(year)	4	5	6	7	8	9	10	11	12	13	14	15
Number	5	8	10	7	5	2	4	1	4	2	1	1
%	10	16	10	14	10	4	8	2	8	4	2	2

Regarding tympanometry, our results showed that, 90% of cases are type B, 6% are type C and 2% are type A.

The targets of our study (patients) were classified into five subgroups according to duration of the disease. Table (2) shows the distribution of patients according to duration of the disease. Most of the cases submitted in our study were more than 10 months at time of diagnosis.

**Table -2: Duration of the disease at time of diagnosis**

Duration(months)	1-3	6-4	7-9	>10	Unknown
Number	14	10	4	20	2
%	28	20	8	40	4

The distribution of the patients were also classified according to the complains of the patients, where hearing impairment was the most common symptom (table 3).

**Table -3: Distribution of patients according to presenting symptoms**

symptoms	Hearing impairment	Speech delay	School delay	incidental	Ear fullness
number	42	2	0	6	0
%	84	4	0	12	0

Regarding the conductive deafness, our results showed that the average of air conduction thresholds were 31dBHL in the range between (0-52) dBHL, (table 4).

**Table -4: Distribution according to average of air conduction threshold**

Range(dB)	0-20	21-40	>40
Number	28	40	32
%	28	40	32

We found four cases of mixed SNHL, which was considered to be etiologically related to OME (8% of our study), where there was no family history of hereditary deafness, or history of ototoxic drugs, trauma or craniofacial abnormalities, (table 5).

**Table -5: Distribution according to average of bone conduction threshold**

Average (dB)	0-10	11-20	>20
%	72	20	8

Regarding the average of air conduction threshold in each frequency, we found that the frequency 2000 Hz was the least affected one & 500 Hz was the most affected frequency, (table 6).

**Table -6: The average of air conduction threshold in each frequency**

Frequency (Hz)	250	500	1000	2000	4000	8000
Average in dB	31	33.4	33.2	24.4	29.8	32

Regarding the average of bone conduction threshold in each frequency, we found that the frequency 1000 Hz was the most affected one, (table 7).

**Table -7: The average of bone conduction threshold in each frequency**

Frequency (Hz)	250	500	1000	2000	4000
Average in (dB)	6.2	7.1	11	7	9

In comparison with above results, after performing pure tone audiogram in healthy individuals (control group) showed that the average of air conduction threshold was 10 dB & the average of bone conduction threshold was 9 dB.

## **Discussion**

This study indicated that OME was commonly associated with audiological changes. Many cases were excluded for inconvenience due to uncooperation of the patient or due to uncertain diagnosis. Although most cases affected by OME were below this age but they were unsuitable to do pure tone audiogram. There are many controversies regarding pathogenesis, audiological changes & the treatment of OME, but still, it is considered as one of the most common causes of hearing impairment in children, not only this, but OME may progress to a permanent chronic inflammatory diseases & this is common especially

with children. In our study, in contrast to acute otitis media, we found that the clinical manifestations of this condition in children were subtle which delay consultation of the physician especially in our communities as well as the underestimation of the problem by many doctors. The conductive deafness in OME results from disturbance of middle ear mechanism by preventing the motion of the ossicles Sensorineural Hearing Loss (SNHL) in OME is known to occur in various types of otitis media. Although the mechanism by which SNHL develops in association with (OME) is unknown accurately, several hypotheses have been advocated up to now like: Paparella (1980) assumes that some noxious agents, for example bacterial products such as endotoxins, might enter the inner ear through the round window membrane and exhibit their ototoxicity <sup>(13)</sup>. Yellon (1994) suggested the Quinolinic acid (QUIN) (is an endogenous metabolite) that exerts a neurotoxic effect by binding to specific neural receptors, (QUIN) was found in the middle ear effusions has the potential to cross the round window membrane and cause SNHL, possibly by binding to specific neural receptors in mammalian cochleae <sup>(14)</sup>. Another explanation is that accumulation of fluid in the middle ear may reduce oxygen supply to the inner ear through the round window membrane and disturb the cochlear function, in this connection; it has been shown that oxygen tension of the scala tympani depends on that of the middle ear drops under the condition of OME. SNHL might be due to the inhibition of the vibratory movements of the oval and\ or round windows caused by the presence of the effusion. This phenomenon was eliminated after the insertion of ventilation tubes in three cases out of four cases in our study. SNHL improvement was not achieved in one case, neither after medical treatment nor following insertion of ventilation tubes, and this may be due to the permanent damage caused by OME or may be due to other causes not related to this disease. Numerous short observation intervals are needed to accurately record the onset & the duration of OME, as the natural history is one of resolution. In comparison with other studies, T. J. Fria (1985) demonstrated that the average of air conduction threshold was 27dB, & there were 35 % of cases within normal hearing, 45% of cases had mild conductive hearing impairment & 20% had moderate conductive deafness, while the average of bone conduction threshold was 8dB <sup>(15)</sup>. In study of Thrasher (2007), the average of air conduction threshold was 28dB & there were 30% of cases within normal hearing, 50% of cases had mild conductive deafness & 20% had moderate conductive deafness, & most of these results were near to the results of our study <sup>(16)</sup>.

## **Conclusions**

Otitis media with effusion (OME) was the most common cause of hearing impairment among children & usually of conductive type, low frequencies were affected more than high frequencies and the frequency 500 Hz was the most affected one. Although, OME is a middle ear cleft disease but can cause Sensorineural hearing loss (SNHL) and the frequency 1000Hz is the most affected one.

## **References**

1. Cuneyt, M. Alper. 2001. Decision making in ear, nose & throat disorders W.B. Saunders company. USA. Pp.34-37
2. Rinaldo, F Canalis, Paul, R. Lambert. 2000. The ear, comprehensive otology. USA. PP 383-391
3. Byron, J. Baily. 1998 H&N surgery-otolaryngology second edition.USA. Pp.1297-1309
4. Bruce, W. Jafeck. 2005 ENT secrets, 3rd edition. USA pp. 67-71
5. John, B. Booth. 1997 Scott-Brown's otolaryngology sixth edition.UK 3/1/17, 3/3/10
6. LEE, K. J. 2003. Essential otolaryngology head & neck surgery. Eighth edition pp. 479-484
7. Stanley, A. Gelfand. 1997 Essentials of audiology. USA. Pp.186-194 Thieme.
8. Mary, Tally Bowden. 2006. Otolaryngology & facial plastic surgery, second edition, USA P.12
9. George, A. Gates. 1988 Current therapy in otolaryngology & H & N surgery sixth edition. USA P.12
10. Charles, W. Cummings. 2005. Cummings otolaryngology H&N surgery 4<sup>th</sup> edition review. USA P.320
11. Gerard, M. O'Doghue, Grant J. Bates, Antony A. Narula 1992. UK Pp 63-65
12. Martin, Burton. 2000. Hall & Colman's diseases of the ear, nose & throat. Fifteenth edition.UK Pp 3840
13. Paparella, M. M. 1980. Inner ear pathology & otitis media. Pp. 294, 253
14. Yellon, RF, Rose E. SNHL from quinolinic acid; a neurotoxin in middle ear effusions. Laryngoscope 1994 feb. 176- 181
15. Thomas, J. Fria 1985. Hearing acuity of children with otitis media with effusion. Arch otolaryngol. Volume 111(1), 6-10
16. Thrasher, R. D. 2007. Otitis media with effusion emedicine. USA