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Original Article

Sensorineural component in chronic suppurative otitis media

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Abstract:

A cross sectional study was conducted at the Department of Otolaryngology and Head-Neck surgery, Dhaka Medical College Hospital, Dhaka during the period of July 2005 to June 2007 to determine whether chronic suppurative otitis media (CSOM) can cause sensorineural hearing loss (SNHL) and to note its degree and its relation to duration of disease.

130 patients with unilateral CSOM were included in the study age ranging from 11 to 50 years after exclusion of other possible causes of SNHL such as meningitis, head injury, previous ear surgery, and chronic noise exposure. The use of unilateral CSOM cases provided a contralateral ear to serve as a control thus excluded common variables, such as presbycusis, parenteral ototoxic medications, metabolic disorders, and the effect of sex or genetics. Bone conduction threshold elevation between diseased and control ear was considered as the indicator of inner ear damage.

In this series, CSOM is seen to be associated with sensorineural hearing loss. The study showed a bone conduction threshold elevation from 4.1dB to 10.7dB across frequency ranges. Significant threshold elevation was observed in relation to the disease duration. The average bone conduction loss at 4 kHz was higher than the average at the speech frequency range.

This study suggests that CSOM can cause significant bone conduction threshold elevation and it should be considered when managing this problem. Early detection and prompt treatment may limit this potential handicap.

Key words: *CSOM, Sensorineural hearing loss, Bone conduction*

Introduction:

Chronic suppurative otitis media is persistent inflammation of the middle ear or mastoid cavity, clinically manifested with deafness and discharge through a persistent defect in the tympanic membrane. CSOM is typically a persistent disease, insidious in onset often capable of causing severe destruction and irreversible sequelae.

Hearing loss associated with CSOM is matter of concern globally, particularly in children because of its long term effect on development of essential skill in speech, language and social interaction. Hearing disability in adults also has its bearing on the individual and on the society.

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Chronic suppurative otitis media is fairly common disease in our country especially in rural community[1]. WHO in 2004 mentioned, the worldwide prevalence of chronic suppurative otitis media is 65-330 million people and 39-200 million (60%) suffer from clinically significant hearing impairment[2]. It is observed CSOM frequently affects children in developing countries[3]. In a study in our country, it was found 34.56% of school children had different grades of hearing impairment and 16.95% having chronic suppurative otitis media had mild to moderate hearing loss (41- 60 dB)[1].

Tuning fork tests and an air and bone conduction audiograph usually show a mainly conductive hearing loss…(but) cochlear degeneration can develop very insidiously in chronic otitis media[4]. A study showing positive association also. The degree of loss was small 5 dB (Bone conduction threshold difference at 0.5, 1, 2 kHz) but individual variation was large and clinically significant[5]. It is found in a study that mean increase in preoperative bone conduction threshold between diseased and control (contrlateral normal) ears ranging from 5.2 dB to 9.02 dB at different frequencies. Their

study population included individuals with unilateral chronic otitis media, no history of head injury, meningitis, previous otological surgery, systemic ototoxic drug therapy. The use of unilateral CSOM cases provided a contralateral ear as a control thus eliminating common variables like presbycusis, parenteral ototoxic medications, metabolic disorders, and the effect of sex or genetics[6].

In these studies, just mentioned, deterioration in bone conduction (BCT) was used as the measurement of inner ear changes. To find out the pathological basis of inner ear affection Cureoglu *et al.* in 2004 studied fifteen human temporal bones with unilateral chronic otitis media and compared with opposite normal temporal bones. Loss of hair cells was common in basal turn of cochlea and there was also significant reduction of area of stria vascularis and spiral ligament compared to normal side[7]. In a study, among 195 patients with monolateral chronic otitis media (COM) disease duration showed significant influence upon sensorineural hearing loss and an average increase of 5.5 dB for every 10 years was found by Cusimano *et al*. Similar view is shared by another study.

In this study, which is the first of its kind in our country, was carried out over a limited period of time and in limited number of patients with unilateral CSOM. General objective was to evaluate the relationship between chronic suppurative otitis media (CSOM) and sensorineural hearing loss. Its specific objectives was I). to find out degree of sensorineural hearing loss in CSOM, II). to correlate the degree of hearing loss with duration of disease.

Methods:

This cross-sectional study was done in Department of Otolaryngology and Head-Neck Surgery, Dhaka Medical College Hospital, Dhaka during the period July 2005 to June 2007 (2 years). The patients suffering from Chronic Suppurative Otitis Media who attended the outpatient department and/or admitted in the Indoor of Department of Otolaryngology and Head-Neck Surgery, Dhaka Medical College Hospital, Dhaka, during the study period were the study population. Purposive sampling was done using the inclusion criteria: I) Unilateral chronic suppurative otitis media (Normal tympanic membrane in contra lateral ear) II) Patients with age ranging from 11 to 50 years and exclusion criteria : patients with H/O of head injury, meningitis, previous ear surgery, chronic noise exposure.

Result:

The findings of the study obtained from data analyses are documented below.

Age distribution:

Age distribution demonstrates that over 35% of the patients were 20 – 30 years of age followed by 32.3% between 30 – 40 years, 20.8% between 40 years or above and 11.5% below 20 years of age. The mean age of the patients was 31.1 ± 9.8 years and the lowest and highest ages were 11 and 50 years respectively.

Occupation:

Distribution of the patients by occupation shows in Figure-1.

Fig.-1: *Distribution of patient by occupation (n = 130)*

Presenting complaints:

Mode of clinical presentation demonstrates that deafness was commonest symptom (93.8%), followed by ear discharge (84.6%), tinnitus (39.2%), earache (28.5%) and dizziness (6.2%) (Table-I).

Table-I

Distribution of patients by presenting complains (n = 130)

Presenting complains	Frequency	Percentage
Deafness	122	93.8
Ear discharge	110	84.6
Tinnitus	51	39.2
Earache	37	28.5
Dizziness	08	6.2

Table-II

Distribution of patients by presence of risk factors (n = 130)

Risk factors	Frequency	Percentage
Ototoxic drug	58	44.6
DM	22	16.9
HTN	14	10.8
Renal disease	05	3.9
Family history of hearing impairment	03	2.3

Bone conduction:

Table-IV shows the comparison of bone conduction between diseased and healthy ears at different frequencies. At all the frequencies the bone conduction was observed to be significantly higher in the diseased ears than those in the contralateral healthy ears ($p < 0.001$).

Table-III

Bone conduction between diseased and healthy ears (n = 130)

Frequency (Hz)	Bone conduction (dB) Diseased Ear (Hz)	Healthy Ear (n = 130)	p-value
500	8.0 ± 9.1	3.9 ± 6.4	< 0.001
1000	10.4 ± 10.4	4.0 ± 6.3	< 0.001
2000	18.9 ± 10.9	8.2 ± 6.4	< 0.001
4000	17.1 ± 12.1	7.3 ± 8.4	< 0.001

Data were analyzed using paired t-Test and were presented as mean ± SD.

Table-IV

Bone conduction between diseased and healthy ears (n = 130) in speech frequency and 4kHz

Bone conduction (dB)	Mean ± SD
Bone conduction diseased ear, speech frequency	12.32 ± 9.28
Bone conduction healthy ear, speech frequency	5.39 ± 5.48
Bone conduction, diseased ear, 4 kHz	17.12 ± 12.18
Bone conduction, healthy ear, 4 kHz	7.35 ± 8.04

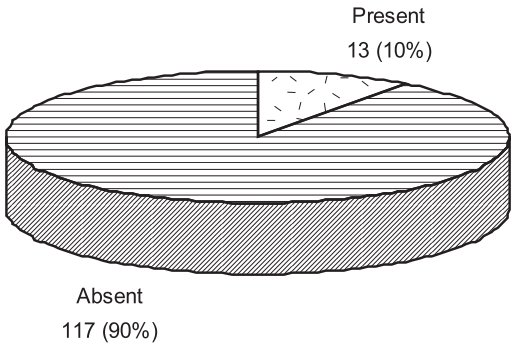

Fig.-2: *Sensorineural hearing loss detected among the study subjects.*

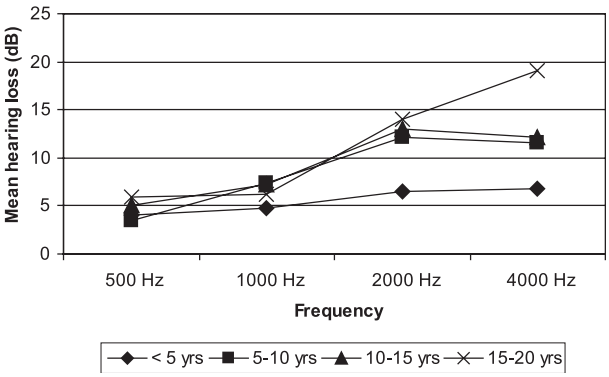

Fig.-3: *Bone conduction loss related to duration of disease (n = 130). Fig. compares the hearing loss experienced by the patients relative to duration of disease.*

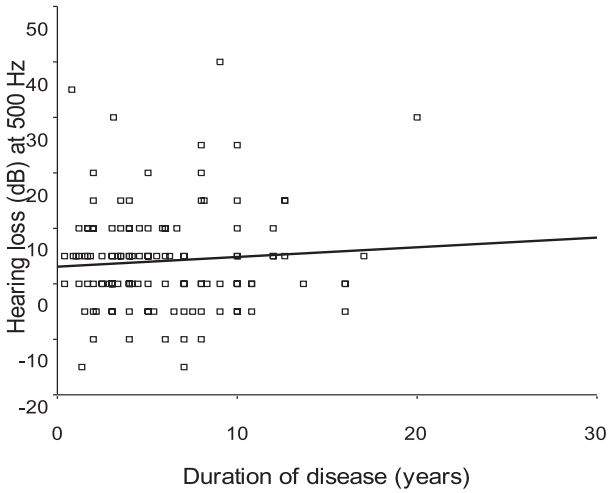

Fig.-4: *Correlation between duration of diseases and hearing loss (dB) at 500 Hz. No significant correlation between duration of illness and hearing loss at 500 Hzs was observed ($r = 0.074$, $p < 0.403$)*

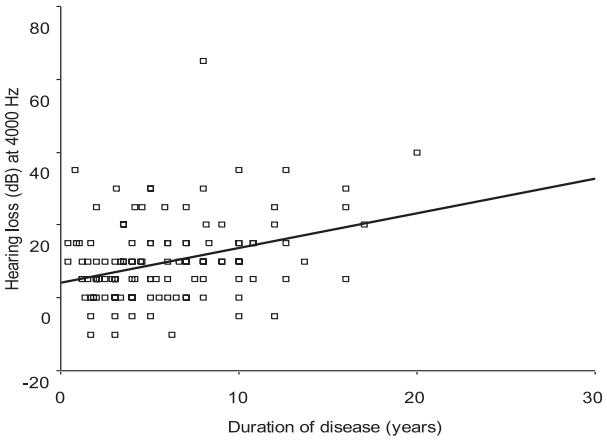

Fig.-5: *Correlation between duration of disease and hearing loss at 4000 Hz Duration of illness and hearing loss at 4000 Hzs was observed to be significantly correlated (r = 0.335, p < 0.001).*

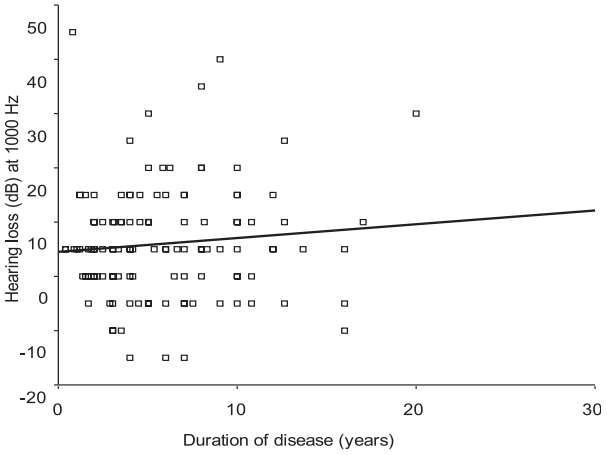

Fig.-6: *Correlation between duration of disease and hearing loss at 1000 Hz. Duration of illness and hearing loss at 1000 Hzs was not observed to be correlated (r = 0.096, p < 0.277).*

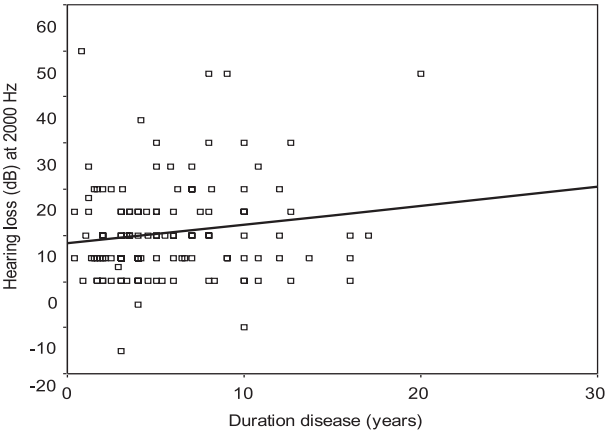

Fig.-7: *Correlation between duration of disease and hearing loss at 2000 Hz. No significant correlation between duration of illness and hearing loss at 2000 Hzs was observed (r = 0.149, p = < 0.092)*

Discussion:

In this present series, about 68% of patients were in third and fourth decades. Mean age was 31.1 ± 9.8 years. This age range matches the samples in Cusimano *et al*[8], it was 31.2±16, in El Sayed[10] it was 35.3 years and age range was also similar in studies by Paparella *et al*[11]. Among respondents, by occupation 30.8% were housewife, followed by 23.1% students, 22.3% service holders and 13.1% business. Highest 79% of the patients were in the middle income group. The prevalence of CSOM is more in people of lower socioeconomic group. In my series, patients who sought treatment in the place of study and those who maintained the subsequent visits after relevant investigations could be included in the study. This probably resulted in the outcome of highest representation of middle income group of people in my series. Among them 84(65%) were from urban area and 46(35%) were from rural area. Higher proportion of urban patients may be the reflection of inclusion of defined group of patients in an urban referral hospital.

Presence of risk factors for developing sensorineural hearing loss (SNHL) in CSOM and their relative incidence was investigated. It was found that 10.8% of patients in my series were hypertensive. The studies that relate to vascular disorders and hearing are inconclusive. This is illustrated by one of the largest and most recent human studies, by Gates *et al*. which followed 1662 patients from the Framingham Heart Cohort study[12]. After adjusting for age the only risk factor associated with hearing loss was systolic blood pressure in women and that was a small effect (1dB low frequency elevation for every 20mm Hg); there was no effect with diastolic pressure.

Another risk factor, Diabetes Mellitus (DM) was present in 22(16.9%) of patients. Finding the association between hearing loss and diabetes mellitus differed from literature to literature. Taylor and Irwin[13] found diabetics as a whole were deafer particularly in the lower frequencies than the controls and gradually approach each other in the middle range(1- 4 kHz) and were similar at 8 kHz. In contrast, Virtaniemi *et al* hearing level tended to be worse in diabetic patients than in controls but the difference was statistically different only in frequencies of 6kHz and 8kHz. Microvascular complication and disease duration was associated with elevated hearing thresholds. In this series, 3(13.6%) out of 22 diabetic patients had developed sensorineural hearing loss.

Being a systemic disease it is expected to have bilateral affection rather than unilateral. So unilateral sensorineural hearing loss of these three patients of current series cannot be solely explained either by diabetes or CSOM.

Possible role of ototoxicity relating to certain therapeutic agents both topical and systemic were enquired. In our series of 130 patients of CSOM 44.6% gave history of using any of the ototoxic drugs. Most of the patients did use different topical aural medications, patients received these topical drops usually for 7-10 days and none of the patients did use the drug for more than two weeks in a single occasion. Among the sample patients 44.6% could mention the name of the preparation but most of them (52.3%) could not. Patients treated with ototoxic antibiotics (containing aminoglycosides) were not excluded from the present study because the majority of patients received these medications. Though ototoxic antibiotics ear drops accepted as risk factors but it is related to duration of treatment and duration of disease also[14]. But Linder *et al*. mentioned possible ototoxic side effects of ototopical preparations in continued use beyond two weeks[15]. Family history may sometimes play a role. But in this series 2.3% gave history of acquired deafness of one of their parents. So it is not expected to carry any significant relevance in this present series.

Levine *et al.* in 1989 in their study found the mean bone conduction difference for the pure-tone average was 9.1 dB and ranged from 5.6 to 12.8dB across the frequency range[16]. In the present series bone conduction measurement in diseased and uninvolved contralateral side shows there is both absolute and relative elevation of bone conduction in diseased side and this elevation is significantly higher ($p<0.001$) at all frequencies. In 500Hz, 1000Hz, 2000Hz,4000Hz bone conduction threshold in diseased ear is 8.0, 10.4, 18.9 and 17.1dB respectively and threshold difference is 4.1, 6.0, 10.7 and 9.7dB respectively.

Mean bone conduction threshold in Speech frequencies (500,1000,2000 Hz) and separately at 4000Hz is shown in Table-III. It shows 6.93dB difference in speech frequencies which increases to 9.77dB at 4000Hz. It demonstrates that higher frequency is more susceptible than lower frequencies. It conforms with findings of Papp et al.[17], who reported among 121 patients a threshold elevation from 5.91dB to 8.8dB at speech frequencies and 4kHz respectively. Similar result is observed by MacAndie and O'Reilly[6] in which bone conduction difference of the diseased and control ears ranged from 5.24 to 9.02 Hz across frequency range. The difference tended to increase with increasing frequency.

Paparella *et al.* in 1984 described a large series of 607 patients with unilateral CSOM from six centers of five countries across the globe. Bone conduction impairment (30dB or greater at two or more frequencies in 1,2 and 4 kHz) was 11% in Minnesota, 23% in Italy, 12% in Finland, 30% in New York, 18% in Sweden, 11% in Korea with an average of 18 percent[11]. Similar result was found by Vartainen and Karjalainen[18] who used the criteria of 30dB or more bone conduction loss. In their series of 874 chronically infected ears SNHL was 1.6% in control ears and 18% in chronically infected ears. An average hearing impairment of 25dB or greater over the frequencies 0.5,1,2 and 4 kHz is presumed as working definition for sensorineural pathology. Applying the same threshold levels in bone conduction(BCT), in this series 10% had developed sensorineural hearing loss among 130 patients (Fig. 2). So findings of this study confirms with the other literatures.

In general, it is shown that the duration curve slopes upwards to the right meaning progressive duration of disease causes progressive bone conduction threshold deterioration (Fig. 3). It is more evident as frequency is increased from smaller to higher one (e.g. 0.5kHz, 1, 2, 4kHz). Significant correlation with duration of disease is found only in 4kHz (Fig. 4-7).Cusimano *et al.* observed statistically significant increase in the sensorineural component in the hearing loss with respect to those of the healthy ears (0.5kHz, 1, 2, 4kHz) related to duration of pathology[7]. Similar effect of duration also reported by El Sayed[10], Vartainen and Karjalainen[18]. The duration of infection was frequently based on a vague subjective opinion, and it may be that a better correlation of duration of infection with threshold difference would have been found with more accurate data.

The findings of a BCT difference between normal and infected ear requires an explanation of what might be causing it. Walby *et al.* although admits possibility of invasion of inner ear through different routes, assumes mechanics of sound transmission as a plausible explanation for the hearing loss[19]. In the other way, a number of microbial sub-products, products of tissue degradation, tissue enzymes and/or humoral factors

are generated or involved in the suppurative process and their effects can be deleterious to cochlear function, if contact with the structures of the inner ear is possible. Closer location of the hair cells responsible for high frequency hearing (to the base of the cochlea) and the round window probably explains this. According to Papp *et al* (2003) harmful agents (like inflammatory mediators) penetrating the round window membrane may reach these cells in a higher concentration, in this way[17].

References:

1. Amin MN, Datta PG, Amin AS. Incidence of hearing impairment amongst the school going children. Journal of Bangladesh College of Physicians and Surgeons. 1992; 10(1): 23-25.
2. WHO. Chronic suppurative otitis media. burden of illness and anagement options. 2004. CHILD_HEALTH/ISBN_92_4_159158_7.pdf (last accessed 8 November 2005) http://www.who.int/child-adolescent-health/New_Publications/.
3. Rupa V, Jacob A, Josheph A .Chronic suppurative otitis media: prevalence and practice among rural south Indian children. International Journal of Pediatric Otorhinolaryngology. 1999; 48(3): 17-221.
4. Paparella MM, Bradley DR and Hoel R. Sensorineural hearing loss in chronic otitis media and mastoiditis. Trans. Am. Acad. Ophthalmol. Otolaryngol. 1970; 74: 108–115.
5. Blakley BW, Kim S. Does chronic otitis media cause sensorineural hearing loss. J of Otolaryngology. 1998; 27(1): 17-20.
6. MacAndie C, O'Reilly BF. Sensorineural hearing loss in chronic otitis media. Clin Otolaryngol Allied Sci. 1999; 24(3): 220-2.
7. Cureoglu S, Schachern PA, Paparella MM, Lindgren BR. Cochlear changes in chronic otitis media. Laryngoscope. 2004; 114(4): 622-6.
8. Cusimano F, Cocita VC, and D'Amico A. Sensorineural hearing loss in chronic otitis media. J. Laryngol. Otol. 1989;103: 158–163.
9. Redaelli de Zinis LO, Campovecchi C, Parrinello G, Antonelli AR. Predisposing factors for inner ear hearing loss association with chronic otitis media. Int J Audiol. 2005; 44(10): 593-8.
10. El-Sayed Y. Bone conduction impairment in uncomplicated chronic suppurative otitis media. Am J Otolaryngol. 1998;19(3): 149-53.
11. Paparella MM, Morizono T, Le CT, Mancini F, Sipila P, Choo YB, Liden G, Kim CS. Sensorineural hearing loss in otitis media. Ann Otol Rhinol Laryngol.1984; 93: 623-9.
12. Ludman H & Wright T. Diseases of the Ear, 6th edn, London. Arnold;1998.
13. Kerr AG, Booth JB. Scott-Brown's Ototlaryngology. 5th Ed.vol.3. Oxford. Butterworth-Heinemann; 1997.
14. Podoshin L, Fradis M, Ben David J. Ototoxicity of ear drops in patients suffering from chronic otitis media. J Laryngol Otol. 1989;103(1): 46-50.
15. Linder TE, Zwicky S, Brändle P . Ototoxicity of ear drops: a clinical perspective. Am J Otol. 1995;16(5): 653-7.
16. Levine BA, Shelton C, Berliner KI, Sheehy JL. Sensorineural hearing loss in chronic otitis media. Is it clinically significant? Arch Otolaryngol Head Neck Surg.1989; 115(7): 814-6.
17. Papp Z, Rezes S, Jókay I, Sziklai I. Sensorineural hearing loss in chronic otitis media. Otol Neurotol. 2003; 24(2): 141-4.
18. Vartainen E and Karjalainen S. Factors influencing sensorineural hearing loss in chronic otitis media. Am. J. Otolaryngol. 1987; 8: 13–15.
19. Walby AP, Barrera A and Schunecht HF. Cochlear function in chronic suppurative otitis media. Ann. Otol. Rhinol. Laryngol. 1983. 92(Suppl. 103): 3-19.