ORIGINAL ARTICLE

Sensorineural Hearing Loss in Chronic Suppurative Otitis Media

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ABSTRACT

Aim: To determine that chronic suppurative otitis media (CSOM) is associated with sensorineural hearing loss.

Methods: 53 patients with unilateral chronic suppurative otitis media were included in the study. The diseased ear served as case and normal ear served as control. Patients with systemic disease, previous history of trauma, meningitis and other CNS infections were excluded from the study to eliminate other contributing factors resulting in SNHL.

Results: All patients after history and examination were subjected to pure tone audiometery. Air and bone conduction thresholds were recorded at four test frequencies i.e. .5 kHz, 1 kHz, 2 kHz, and 4 kHz. SNHL was defined as difference in bone conduction thresholds between diseased and control (normal) ears. All the data was analyzed statistically (student's t test). There was significant bone conduction threshold difference between diseased and control ears p 0.001.

Conclusion: It was concluded that CSOM results in statistically significant SNHL.

Keywords: Hearing loss, Ch cuppurative otitis media, sensorineural

INTRODUCTION

Chronic suppurative otitis media (CSOM) is very common in developing countries especially in Asia and Africa. CSOM usually begins in childhood as tympanic membrane perforation due to acute suppurative otitis media (ASOM) or as a sequel of less common otitis media with effusion. CSOM is defined as chronic discharge from an ear through a persistent perforation in tympanic membrane.

Hearing loss is one of the common feature of CSOM and most often the presenting feature in our society. Hearing loss in CSOM is usually conductive in nature due to tympanic membrane perforation or damage to ossicles[1]**.** But sensorineural hearing loss (SNHL) is also reported in patients suffering from CSOM[2,3,4,5]**.** There have been different studies on the subject, many clinical and some histological one to correlate the association between SNHL and CSOM. This question still remains controversial. The majority agree that CSOM is associated with some degree of SNHL. Most authors agree that bacterial toxins are the causative factor of this neural type of deafness, and also the fact that round window membrane is the pathway which transmits toxins from middle ear to inner ear[6]**.** The question of concern is whether this SNHL due to CSOM is significant clinically. Another question is whether the type of disease, its severity, and duration correlates with the severity of SNHL.

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This study was carried out using strict criteria to eliminate contributing factors. The most important criteria were unilateral SCOM, the diseased ear served as the case and the normal ear served as the control. Other causes of SNHL were eliminated by selecting patients with no previous history of head trauma, meningitis, noise trauma, or noise exposure. Traumatic perforations were also excluded from the study and also patients having systemic diseases like diabetes.

PATIENTS AND METHODS

The study population consisted of 53 cases of unilateral CSOM selected through non-probability purposive sampling technique. These patients presented in the out-patient department of Ghurki Trust Teaching Hospital, Lahore. The duration of study was from 01 March 2013 to 31 October 2013. Patients were included in the study having unilateral CSOM, no previous history of head trauma, meningitis or intracranial infections, labyrinthine fistula, traumatic tympanic membrane perforation, noise exposure, or any systemic disease like diabetes. Patients were excluded from the study if any of the audiogram showed air conduction threshold more than 25dB, and significant air-bone gap (i.e., more than 10 dB) in any of the test frequencies in normal ear.

Patients included in the study were subjected to detailed history and examination. The examination included examination under microscope for all the patients. Afterwards patients were subjected to Audiological evaluation. Audiometery was carried out in sound proof room in out-patient department of

Ghurki Trust Teaching Hospital. Air and bone conduction thresholds were estimated for both diseased and control ears at four test frequencies i.e. .5 kHz, 1 kHz, 2 kHz, and 4 kHz. All the finding of history, examination and audiometery were recorded on a Performa specially designed for this purpose. Paired students t-test was used to analyze the differences between bone conduction thresholds of diseased ears and normal ears at the four test frequencies i.e. .5 kHz, 1 kHz, 2 kHz, and 4 kHz. WINKS 4.80a windows version of KWIKSTAT program was used to perform statistical analysis.

RESULTS

53 patients were included in the study including 32 male and 21 female patients, with male to female ratio of 1.5:1. The age of patients ranged from 13 to 62 years with a mean age of 26.3 years. The most common presenting symptom was ear discharge and deafness. Other symptoms were vertigo and tinnitus. The average duration of ear discharge in our patients was 12.7 years, deafness was present for an average of .7 years before presentation. These two symptoms were seen in all the patients. Vertigo was present in 21 of 53 patients and tinnitus was seen in 17 of 53 patients. The average of duration of these symptoms before presentation was 1 month.

After detailed history and examination all patients underwent pure tone audiometery. The air and bone conduction thresholds were recorded for four test frequencies i.e. .5 kHz, 1 kHz, 2 kHz, and 4 kHz.

The mean values for hearing threshold testing on bone conduction audiometery for normal ears in all patients were 6.13 at .5 kHz, 4.15 at 1 kHz, 10.56 at 2 kHz, and 8.20 at 4 kHz. The mean values of hearing threshold on bone conduction testing in diseased ears in patients at different frequencies tested were 28.58 at .5 kHz, 29.05 at 1 kHz, 34.43 at 2 kHz, and 34.90 at 4 kHz. The mean difference between diseased and normal ears in patients at four test frequencies were 22.45 at .5 kHz, 24.90 at 1 kHz, 23.86 at 2 kHz, and 26.69 at 4 kHz. The p values were significant <0.001, at all the test frequencies. The details of statistical analysis are summarized in table.

P value for Bone conduction (BC) thresholds (n=53)

	.5 kHz	**1 kHz**	**2 kHz**	**4 kHz**
Normal ears (mean BC thresholds) SD	6.13208 6.09551	4.15094 7.12092	10.56604 7.38113	8.20755 5.97224
Diseased ears (mean BC thresholds) SD	28.58491 15.01209	29.0566 16.55575	34.43396 17.85789	34.90566 19.67257
Difference between diseased and normal ears (mean BC thresholds) SD	22.45283 13.88907	24.90566 15.97746	23.86792 19.15628	26.69811 18.83489
Calculated t with 52 D.F. P value	11.7689 <0.001	11.34823 <0.001	9.07071 <0.001	10.31942 <0.001

DISCUSSION

CSOM is commonly encountered in our everyday ENT practice. The fact that CSOM causes conductive hearing loss is well established but many studies have reported SNHL associated with CSOM. Some investigators believe that round window membrane is a pathway for bacterial toxins from middle ear to inner ear resulting in cochlear damage. Some other believe that other factors like systemic diseases like diabetes, or noise exposure, previous head trauma, and so forth may be the contributors in causing SNHL in patients seen with CSOM. In this study strict inclusion criteria was imposed to take care of these factors.

Sensorineural hearing loss was defined as a statistically significant difference between the bone conduction thresholds of diseased ears and normal ears.[7] We found that bone conduction thresholds were elevated in diseased ears at all test frequencies, and more at high frequencies i.e. 2 kHz and 4 kHz. The average difference between the diseased and control ears were more than 20 dB at all test frequencies, a borderline threshold defined by WHO. The results of our study indicate a statistically significant component of SNHL in patients with CSOM. Bone conduction thresholds of diseased ears were elevated significantly compared to normal, control ears.

Many previous studies have indicated the element of SNHL in patients with chronic middle ear disease, but the magnitude of hearing loss varied from 5 dB to 30 dB in various studies.[7,8,] Most previous studies studied absolute bone conduction thresholds of diseased ears, and not a difference in threshold between diseased ears and normal, control ears. More recent studies report a difference in threshold between diseased and normal ears.

The cause of SNHL in CSOM is proven but what actually results in neural deafness is not clearly

known. Paparella reported that round window membrane acts as a conduit for toxins to reach inner ear from middle ear. He also reported serofibrinous precipitates and inflammatory cells located in inner ears near round window membrane[9]. While others reported labyrinthitis, infiltration of round window with inflammatory cells, and cochlear hydrops[10]. Still others did not find any pathology attributable to SNHL in temporal bones with CSOM[11].

Our study categorically indicates that there is SNHL associated with CSOM and the degree of hearing loss is significant statistically. As with other studies our study had also some limitations, that it did not study the nature of disease process (safe of unsafe CSOM) associated with hearing loss, more over the duration of disease and degree of hearing loss was not studied.

Another point of concern is that we used deterioration in bone conduction thresholds as a measure of inner ear damage. This has been seen with concern that bone conduction threshold is not synonym with cochlear function and might be altered by middle ear cleft changes occurring in subjects with chronic middle ear infection.[12] It has been shown that mechanical obstruction of oval window area may result in elevated bone conduction thresholds with incorrect estimation of relative SNHL. Moreover it has also been demonstrated that ossicular necrosis lead to increase in bone conduction levels along with air conduction thresholds[13,14].

CONCLUSION

CSOM is associated with SNHL and this neural hearing loss is statistically significant. It is therefore noted that all ENT surgeons should implement early medical and surgical management of any form of CSOM to prevent SNHL which is irreversible in most of the patients.

Acknowledgement: I am thankful to Dr Muhammad Rafique (Ph D) Pakistan Science Foundation for his support and help in carrying out statistical analysis for the study.

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