Post Meningitic Sensori-neural Hearing Loss in Children - Alterations in Hearing Level

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Summary

Post meningitic sensori-neural hearing loss was studied in forty new cases of bacterial meningitis and ten cases of viral meningitis treated at the Pediatric Institute, Kuala Lumpur Hospital from April 1991 to March 1992. Hearing assessment at 2 weeks, 3 months and 6 months following the diagnosis of meningitis using Brain Stem Evoked Response Audiometry showed that hearing loss was prevalent only in patients with bacterial meningitis. Hearing loss was detected in 32.5% of these patients during the acute phase of the disease, 22.8% after 3 months and 24.2% after 6 months. In 63.6% of the affected cases, hearing loss was bilateral. In 61.5% of the patients who had hearing loss during the acute phase of the disease, it was permanent, 16.7% had either partial or complete recovery and, 15.4% had deterioration in hearing level. In 2 cases the subsequent hearing level was unknown. The risk of developing sensori neural hearing loss was found to be significantly higher in patients who developed other neurological sequelae. The study highlights the importance of performing repeated hearing assessment in children with bacterial meningitis and the difficulty in appropriate selection of hearing aids in the early stages.

Key Words: Bacterial meningitis, Sensori-neural hearing loss, Alteration of hearing level

Introduction

The mortality rate in meningitis has significantly decreased since the introduction of antibiotic therapy and adequate treatment of its complications. Unfortunately the reduction of mortality rate is not accompanied by a similar decrease in the immediate or long term sequelae of the disease, with sensori-neural hearing loss being one of the commonest complications[1].

Meningitis is the leading cause of acquired sensori-neural hearing loss. Several studies done on school age deaf children have shown meningitis to be the responsible causative factor in 8-24% of cases with severe deafness[2]. Meningitic hearing loss is often permanent, but it has a remarkable peculiarity that there can be improvement or progression[3,4,5,6,7]. Fewer articles report on late onset of hearing loss and deterioration of hearing level[6]. This possible alteration of hearing level has made it difficult for the audiologists in deciding on the appropriate selection of hearing aids at early stage.

Early intervention of hearing loss is crucial especially in children where it will jeopardise speech development. This paper presents the audiometric findings in a group of patients with meningitis during the acute phase at 3 months and 6 months after the onset of the illness. Realizing the importance of early detection of hearing loss in these cases, an attempt was made to find the possible risk factors in developing sensori-neural loss as a complication of meningitis.

Materials and Methods

The study group consisted of 50 patients with meningitis admitted to the Pediatric Institute, Kuala Lumpur Hospital between April 1991 to March 1992. Of the 50 patients, 29 were girls and 21 were boys. The age ranged between 5 days to 11-years-old; 64% of the patients were under 6 months and 90% were under 2 years of age.

The diagnosis of meningitis was based on the presence of signs of infection and meningeal irritation. The type of the meningitis was based on the following criteria;

i) Bacterial, when

   a) cerebrospinal fluid (CSF) culture isolated bacteria or latex agglutination test was positive for a specific antigen.
   b) in cases with negative CSF culture and negative latex agglutination test, there was a total leucocyte count of over 1000/microliter, glucose levels were below 40 mg/deciliter and positive blood culture or bacteria was present in CSF by Gram staining.

ii) Viral, when

   a) CSF was aseptic by culture, staining and latex agglutination test and
   b) CSF total leukocyte count was less than 1000/microliter and glucose levels were normal (50-85 mg/deciliter).

Patients reported by parents as having hearing loss prior to the onset of meningitis were excluded. All patients underwent otoscopic and tympanometric examination to ensure normal middle ear condition. The hearing was assessed by brain stem evoked response audiometry (BSER). The first hearing assessment was done within two weeks of the diagnosis of meningitis, as soon as the patient's condition was stable. Irrespective of the first BSER results, all patients were scheduled for the second and third hearing assessments at 3 and 6 months following the diagnosis.

BSER test was done using Amplaid MK 10 Multi-sensory system using ipsilateral recording. Each tracing consisted of 2048 sweeps. Two superimposed tracings were obtained at threshold level to ensure repeatability of the waves. The threshold of hearing was based on the lowest intensity stimuli with wave V. The hearing level was classified as

i) *Normal hearing*
   When wave V was present at stimulus of 30 dB HL or below

ii) *Mild hearing loss*
   When wave V was present at stimulus of 35 to 50 dbHL

iii) *Moderate hearing loss*
   When wave V was present at stimulus of 55 to 70 dbHL

iv) *Severe hearing loss*
   When wave V was present at stimulus of 75 to 90 dbHL

v) *Profound hearing loss*
   When wave V was present at stimulus of 90 db HL and above

The diagnosis of sensori-neural hearing loss was made using the following criteria;

i) Normal otoscopic finding
ii) Normal tympanogram
iii) Elevation of hearing threshold by BSER

Results

Causative Organisms

Out of 50 patients 40 had bacterial meningitis and 10 suffered from viral meningitis. Organisms were cultured from the cerebrospinal fluid (CSF) of 75% of the patients with bacterial meningitis. *Haemophilus influenzae* was the leading cause of meningitis (42%). *Streptococcus pneumoniae* was found in 10 per cent of the patients whereas, *Flavobacterium sp., Neisseria meningitidis, Mycobacterium tuberculosis* and *Escherichia coli* were the responsible organisms in 2 to 7 per cent of the cases. The distribution of the causative organisms in patients with bacterial meningitis is shown in Table I.

Post Meningitic Hearing Loss

Sensori-neural hearing loss was observed only among the patients with bacterial meningitis. Table II shows

Table I
Causative organism of bacterial meningitis

Organism	No. of cases	Percent (%)
Haemophilus influenzae	17	42.5
Streptococcus pneumoniae	4	10.0
Escherichia coli	3	7.5
Mycobacterium tuberculosis	3	7.5
Neisseria meningitidis	2	5.0
Flavobacterium sp.	1	2.5
No organism	10	25.0
Total	40	100.0

the number of patients with hearing loss at 2 weeks, 3 months and 6 months after the diagnosis of meningitis according to the causative organism.

i) **Hearing Loss During the Acute Phase**

During the acute phase of the illness, sensori-neural hearing loss was found in 32.5% of the bacterial meningitic cases. In 63.6% of them, the hearing loss was bilateral and the remaining patients had unilateral loss.

ii) **Hearing Loss at 3 Month Follow up**

Accurate incidence of hearing loss at 3 month was difficult to obtain because 5 of the patients defaulted follow up. Of those 5 patients, 2 showed normal hearing and 3 had hearing loss during the acute phase. Out of the 35 patients left 22.8% had hearing loss on the second assessment. All of them were also found to have hearing loss during the acute phase. Although 62.5% showed no alteration of hearing level it is interesting to note that 37.5% of the patients shows either full or partial recovery of normal hearing. Of the 2 cases who recovered normal hearing, one had bilateral minimal hearing loss during the acute phase, while the other case had bilateral profound hearing loss. One patient showed a partial recovery from profound to moderate hearing loss. In 25% of the patients with hearing loss, the hearing level was found to deteriorate from mild to profound in both ears.

iii) **Hearing Loss at 6 Month Follow up**

Two patients who showed normal hearing during the second hearing assessment defaulted follow up for the third hearing assessment done at 6 month after the diagnosis. The hearing level of the remaining 33 patients remained the same as the in the previous assessment at 3 month. The

Table II
Frequency of patients with hearing loss during acute phase, at 3 months and 6 months after diagnosis according to causative organisms

Etiology	No. of cases	No. of cases with hearing loss		
		Acute	3 months	6 months
Haemophilus influenzae	17	4	1	1
No organism	10	2	2	2
Streptococcus pneumoniae	4	2	2	2
Escherichia coli	3	3	2	2
Mycobacterium tuberculosis	3	1	1	1
Neisseria meningitidis	2	0	0	0
Flavobacterium sp.	1	1	–	–
Total	40	13	8	8

incidence of hearing loss at 6 month follow up was therefore 24.2%.

Association Between Hearing Loss and Laboratory Findings

The association between meningitic hearing loss and laboratory findings, such as the initial sugar and protein levels as well as white blood cells count in cerebrospinal fluid were analysed to find the risk factors for developing sensori-neural hearing loss in bacterial meningitis. The mean initial CSF sugar, protein and WBC counts of patients who developed sensori-neural hearing loss and those with normal hearing were compared. Statistical analysis did not show any significant difference in the means of initial CSF sugar, protein and WBC counts between the two groups of patients. Table III summarizes these findings.

Association of Hearing Loss and The Interval Between The Onset of Meningeal Symptoms and Initiation of Treatment

The approximate interval between the onset of meningeal irritation and the initiation of appropriate therapy in hospital was assessed from the history. This interval ranged from 6 hours to 240 hours with mean interval of 75.8 hours ± 32.1. The mean interval between the occurrence of meningeal symptoms and initiation of treatment for the patients with hearing loss was 78.8 hours whereas the mean interval for the normal hearing group was 70.9 hours. No significant difference were found when these means were compared (p=0.75).

Association Between Hearing Loss and Aminoglycosides Treatment

Aminoglycosides has been postulated as one of the responsible factor for hearing loss in meningitic patients. In this study 18 out of 40 patients with bacterial meningitis received aminoglycosides in the course of their treatment. The incidence of hearing loss in patients treated and not treated by aminoglycosides were 27.8% and 27.3% respectively when measured during the acute phase. Statistical test of significance showed no significant difference in the incidence of hearing loss between the two groups of patients (p=0.75).

Association Between Hearing Loss and Other Neurological Sequelae

Twenty percent of the patients with bacterial meningitis had other neurological sequelae such as subdural effusions, hydrocephalus, hypertonia and hemiparesis. The incidence of hearing loss in patients with other neurological sequelae was found to be much higher (62.5%) compared to those without other neurological sequalae (18.8%). These differences in incidence was statistically significant (p=0.01). Therefore, the presence of other neurological complications put the patients at higher risk of developing hearing impairment.

Discussion

Haemophilus influenzae was the leading cause of meningitis in this study. The incidence of meningitic sensori-neural hearing loss varied from 32.5% to 22.8% and 24.2% at 2 weeks, three months and 6 months following the diagnosis. Improvement and recovery of meningitic hearing loss have been reported to occur[4,5,8]. In this study, 3 patients had either partial or complete recovery of hearing level. Complete recovery of hearing function was seen in 2 patients; one had initially a bilateral profound hearing loss and another had mild hearing loss. One patient in whom

Table III
Association between Hearing loss and laboratory findings

Laboratory test	Hearing loss n=13	Normal hearing n=27	Statistical analysis
CSF sugar level (Gm/ml)	1.02	1.13	p = 0.66
CSF protein level (Gm/ml)	3.19	2.23	p = 0.08
CSF WBC count (cell/µl)	927.1	738.2	p = 0.46

the hearing partially recovered, improvement was from profound to moderate. Deterioration of hearing function from mild to profound was noted in 2 cases. It is interesting to note that the alteration of hearing level occurred within 3 months of diagnosis. Hearing level was found to be stable after that period of time. The finding of this study agree with that of others that majority of meningitic hearing loss remain stable[3,5,6].

There are different explanations for the mechanism of meningitic hearing loss. The first mechanism of hearing loss is by direct bacterial invasion of the inner ear causing suppurative labyrinthitis. Subsequent fibroblastic proliferation and ossification are responsible for the occurrence of permanent hearing loss[9]. The second mechanism of meningitic hearing loss is by serous or toxic labyrinthitis. In this phenomenon the damage of the labyrinth is due to diffusion of toxic materials through the cochlear aqueduct. This theory is supported by the observations that hearing loss occurred at the same time as the toxic reaction[1]. Hearing loss can also be due to vascular occlusion by septic embolus which are frequently seen in bacterial meningitis. Other mechanisms include meningeal irritation resulting in neuritis of the auditory nerve. The resolution of labyrinthitis and transient neuritis are thought to be the cause of complete or partial improvement of hearing loss in meningitic cases. Deterioration of the hearing level could be due to secondary endolymphatic hydrops following labyrinthitis. Temporal bone studies have shown that endolymphatic hydrops might be present after labyrinthitis. Platenga and Browning[10] found that in secondary endolymphatic hydrops, the endolymphatic duct was obliterated by bone or fibrous material. Cochlear damage by ototoxic drugs used for the treatment of meningitis has also been postulated to cause hearing loss in these cases[1]. The findings of this study however, does not support the possibility of this theory.

Nadol[6] found a positive correlation between CSF pleocytosis and low sugar concentration with hearing loss. He suggested that this positive correlation may relate hearing loss to the severity of the infection rather than directly to pleocytosis and low CSF sugar level. He also postulated that low CSF sugar concentration may predispose cochlear neuroepithelium to subsequent damage. This study however, did not show any significant correlation between the CSF sugar and white blood cell count with the incidence of hearing loss. It is also interesting to note that the interval between the occurrence of meningeal irritation and the initiation of treatment did not correlate with the incidence of hearing loss. This suggest that the risk of developing hearing loss was not reduced by early treatment of meningitis.

Conclusion

Bacterial meningitis is a significant cause of acquired sensori-neural hearing loss. None of the laboratory findings can be used as an indicator of high risk to developing sensori-neural hearing loss in patients with meningitis. The possibility of improvement and progression of hearing loss in these cases presents a challenge in the management of hearing loss in such cases, especially in children where early intervention is crucial. Stability of hearing level after three months following meningitis suggests that selection of amplification devise can be done with a higher degree of certainty at this period. Repeated hearing assessment is important in all patients with meningitis to ensure a definite level of the permanent degree of hearing loss.

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