

Sensorineural Hearing Loss Following Acute Bacterial Meningitis—A Prospective Evaluation

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Sensorineural hearing loss (SNHL) is the most common neuropsychological sequelae of acute bacterial meningitis (ABM). The effect of SNHL on language is well recognized and therefore early detection is mandatory. However, hearing assessment is difficult in infants and young children. Auditory brain stem evoked responses (ABER) can help to assess hearing in this population. There is paucity of data from India in this respect. The present study was designed to evaluate the incidence of SNHL following ABM and to identify the clinical correlates of SNHL.

Subjects and Methods

This prospective study was conducted in Kalawati Saran Children's Hospital from

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Received for publication: April 10, 1995;

Accepted: February 5, 1996

September 1992 to May 1993. Children in the age group of 1 month to 12 years with clinical signs and symptoms suggestive of ABM and with CSF polymorphonuclear pleocytosis were enrolled in the study. Patients with history suggestive of neurodevelopmental delay or hearing impairment prior to illness were excluded from the study. A detailed history and clinical examination was done at admission and the children were followed till discharge from the hospital. CSF was studied for direct microscopy, biochemical examination, Gram stain, culture and latex agglutination test using Biomerieux slidex meningitis kit.

All patients received the initial treatment as per the standard protocol for ABM which at the time of study comprised of ampicillin (400 mg/kg/day) and chloramphenicol (100 mg/kg/day) in four divided doses intravenously. The antibiotic regime was modified later if necessary depending upon the culture report or poor clinical response. This most often consisted of third generation cephalosporins.

Complete audiological assessment was done before discharge after the patient was clinically cured of meningitis. Otoscopic examination and tympanogram were done to exclude conductive deafness. Auditory brainstem evoked responses (ABER) were recorded in all patients to assess for SNHL. The ABER was elicited on OTE Biomedica using rarefaction click stimulus monoaurally at the rate of 20 stimuli per second. The ABER were recorded by using 2048 stimulus presentations and two tracings were obtained at each intensity level. The starting intensity of the stimulus was 30dBHL. Hearing threshold

was estimated by the weakest stimulus that produced wave V. ANOVA test was applied to detect difference in means and Chi square test to detect difference in proportions.

Results

A total of 50 children with suspected ABM were initially enrolled in the study. Of these, 6 cases were thought to be aseptic or tuberculous meningitis by the CSF and clinical picture and hence excluded from final analysis. Eighty per cent of the patient were infants with 35 cases (79.55%) aged between 1 month to 1 year, 3 (6.81%) between 1-5 years and 6 (13.64%) more than 5 years of age. The main presenting symptoms were fever (100%), seizures (63.9%), altered sensorium (56.8%), and respiratory symptoms (52.9%). A large majority of the patients had received treatment before they were hospitalized. However, the nature of medicines taken was not known and therefore the exact number of partially treated ABM among these patients could not be ascertained.

The etiological organisms were identified in 84% of cases by using different diagnostic methods. *H. influenzae* was the commonest etiologic organism (34.09%), followed by *Strep. Pneumoniae* (27.7%), *N. meningitidis* (6.89%), *Staph. aureus* (6.89%), *E. coli* (4.55%) and others (4.55%). Case fatality was 15.9% and all the 7 patients died during the acute phase. Two patients left the hospital without completing 2 weeks of antibiotic therapy. Consequently, audiological assessment was done only in 35 cases at the time of discharge. Nine out of 35 patients (25.7%) had hearing threshold >30 dB suggesting SNHL and the organism isolated in these cases included *Strep. pneumoniae* (n=4), *H. influenzae* (n=2), *N. meningitidis* (n=1) and *Staph. aureus* (n=1). SNHL was bilateral in 4 cases (11.4%) and unilateral in 5 (14.3%). None of these cases had conductive deafness.

The clinical and CSF parameters of the patients without SNHL (Group I) were compared with the patients who had evidence of SNHL (Group II). CSF pleocytosis (p=0.003), poor response to treatment (p=0.043) and prolonged hospital stay (p=0.0022) were strongly correlated with SNHL (Table I).

Discussion

Our results suggest audiological sequelae in 25.7% of 35 cases of ABM in whom ABER was recorded at discharge. Incidence of SNHL in ABM has been reported to range from 10-47% by other workers(1-3). Carroll and Carroll observed hearing loss in 32.2% of patients with purulent meningitis(1). A much higher incidence (47%) of peripheral hearing loss was found in another study in which hearing was tested immediately following meningitis by ABER(2). On the other hand, the incidence of SNHL was 10.3% in another prospective study(3). Administration of dexamethasone has been shown to reduce the incidence of SNHL in ABM due to *H. influenzae*(4) as well as *Strep. Pneumoniae* (5). Though these two organisms were isolated in more than 50% of cases, our treatment protocol did not include dexamethasone in view of the fact that antibacterial agents need to administered much earlier than the microbiological reports are available. Therefore, it is difficult to comment if the outcome would have been different if steroids were used in those cases.

The mechanism of VIIIth nerve damage and the time at which it occurs has been a subject of many studies(6,7). Indeed it is believed that SNHL occurs in the early phase and is not related to the severity of illness(8). On the other hand some reports have found correlation between severity of infection and audiological outcome(1). The most likely mechanism of VIIIth nerve I damage is perhaps suppurative labyrinthitis which may take place by direct spread of infection from the subarachnoid

TABLE I—Clinical and CSF Correlates in SNHL.

Parameter	Group I (n=26) Mean ± S.D.	Group II (n=9) Mean ± S.D.	p value
Age (mo)	24.58±38.73	16.00±26.96	0.55
Fever duration (days)	4.69±6.14	3.44±3.05	0.85
CSF: Mean cell counts/ cu mm	1071.96±1325.25	3786.11±2808.55	0.003
Protein (mg/dl)	230.81±194.84	561.29±901.17	0.23
Sugar (mg/dl)	43.30±33.23	26.00±17.70	0.19
<i>Course after admission</i>			
Fever (duration in days)	2.73±1.91	4.22±2.33	0.06
Altered sensorium (duration in days)	4.83±5.15	8.57±5.62	0.24
Poor response to treatment (in %)	26.92	66.67	0.043
Hospital stay (duration in days)	14.85±6.52	25.56±13.45	0.0022

space through cochlear aqueduct. Toxic or serous labyrinthitis due to the inflammatory cytokine production is another likely cause(9). Perhaps both the agent factor and natural host defence to the offending organism have a role in the etiologic mechanism of SNHL.

Several clinical and CSF parameters have been studied to determine if they can predict impending hearing impairment. Low CSF glucose(3,10), raised CSF white cell count(11) and severe neurological deficits(3) have been found to be risk factors for SNHL. Factors during treatment like prolonged hospital stay(12) and delayed CSF sterilization(13) have also been associated with SNHL. In the present study prolonged hospital stay, CSF pleocytosis and poor response to treatment

were significantly higher in patients with SNHL. All these indices suggest that infection was severe and long drawn. Though the mean CSF sugar was lower in group II the difference was not statistically significant. This is in sharp contrast to most other studies (3,10,11). Pneumococcus is notorious for causing grave neuroaudiological sequelae (1, 12,14). The sample size was not large enough to detect this difference although 4 out of 9 cases with SNHL were due to *Strep. pneumoniae*.

Our results highlight the problem of hearing impairment following ABM and emphasise the need for complete audiological evaluation after recovery. Hearing impairment needs to be identified and treated as quickly as possible to

prevent further handicap. ABER helps in detecting SNHL in young infants who could have been missed by routine testing. However, it only tests the high frequency of auditory spectrum so that selective low frequency deafness can be missed. Conversely, select high frequency deafness may cause only a minimal influence on auditory function and language development. As most of our patients were small infants, the impact on the language development could not be studied by short term follow up. However, given the high incidence of audiological sequelae and absence of reliable clinical predictors, SNHL should be assumed to be a possibility in every case of ABM unless proved otherwise.

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