

A Retrospective Study to Investigate Sensorineural Hearing Loss in Infants after Acute Bacterial Meningitis

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Conflict of interest: Nil

Abstract

Aim: To investigate sensorineural hearing loss in infants after acute bacterial meningitis.

Material and Methods: It was a retrospective study conducted in Department of Pediatrics, NMCH, Jamuhar, Sasaram, Bihar, India 10 months. Fifty infants with ABM were enrolled in the study with written informed consent from parents. Details were recorded in a pre-decided proforma. Including age, gender and detailed history followed by complete physical examination. Lumbar puncture was performed in all the children for CSF examination. Diagnosis of ABM was considered when CSF examination showed polymorphonuclear pleocytosis, low sugar and high protein for age and /or CSF culture positive for bacterial growth.

Results: 22(44%) had abnormal imaging (USG/CT/MRI) of Brain. Hydrocephalus being the most common abnormal finding was seen in 11 cases. 13(26%) out of 50 children developed SNHL following acute bacterial meningitis. SNHL was bilateral in 7(14%) and unilateral in 6(12%) cases. SNHL was mild in 3patient, moderate in 5 patients, severe in 4 patients and only 1 patient had profound hearing loss. Patient with high value for Mean \pm SD for Total nucleated cells in CSF for group I was 1675.4 ± 2318.6 while for group II it was 444.57 ± 360.3 . CSF Total Neutrophil count had more incidence of SNHL. (P value 0.003). Mean \pm SD for Protein (mg/dl) in CSF for group I was 415.2 ± 484.1 while for group II it was 204.1 ± 210.3 . Patient with higher value for CSF protein had more SNHL. (P value 0.036). Mean \pm SD for Sugar (mg/dl) in CSF for group I was 25.5 ± 15.8 while for group II it was 40.3 ± 15.9 . Patient with low value for CSF sugar have more SNHL. Among 9 cases with CSF sugar less than 20, 5 developed SNHL, which was statistically significant (P value 0.03). Risk of developing SNHL is higher in patient who have CSF sugar less than 20.

Conclusions: The present study throws light on the occurrence of sensorineural hearing loss following acute bacterial meningitis. SHNL, which if left untreated may lead to serious handicap affecting the linguistic performance and overall development of the child. Hence our study emphasizes the need for complete audiological evaluation of a child recovered from meningitis. BERA is a helpful tool for screening the sensorineural hearing loss especially in the young children and infants in whom other conventional methods may not be of much use.

Keywords: SNHL, ABM, BERA, CSF

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Introduction

Sensorineural hearing loss (SNHL) is a severe and potentially lifelong complication that can follow acute bacterial meningitis in infants. Bacterial meningitis, an inflammation of the protective membranes covering the brain and spinal cord, is a medical emergency that requires prompt diagnosis and treatment to prevent severe neurological sequelae and mortality. Despite advances in medical care, the incidence of bacterial meningitis in infants remains significant, with profound implications for long-term health outcomes. [1,2] The pathophysiology of SNHL following bacterial meningitis involves the inflammatory response to

the infection, which can damage the cochlea and auditory pathways. This damage can result from direct bacterial invasion, the toxic effects of inflammatory mediators, or both. Early identification and intervention are crucial, as undiagnosed and untreated hearing loss can adversely affect speech and language development, cognitive skills, and educational achievement. [3,4] Several studies have emphasized the importance of early hearing assessments in infants who have recovered from bacterial meningitis. The prevalence of SNHL in these infants varies widely, with some studies reporting rates as high as 30%. The

variability in reported prevalence may be attributed to differences in study populations, definitions of hearing loss, and diagnostic methods. [5,6] Recent research has focused on the use of advanced diagnostic tools and biomarkers to predict the likelihood of SNHL in infants with bacterial meningitis. For example, the use of otoacoustic emissions (OAEs) and auditory brainstem responses (ABRs) has been shown to be effective in early detection of hearing impairment. Furthermore, studies are investigating the potential role of genetic susceptibility and the impact of specific bacterial pathogens on the risk of developing SNHL. [7] Preventive strategies, including vaccination against common causative pathogens such as *Haemophilus influenzae* type b (Hib), *Streptococcus pneumoniae*, and *Neisseria meningitidis*, have significantly reduced the incidence of bacterial meningitis and its complications. However, despite these advances, the risk of SNHL remains a critical concern. [8-10]

Material and Methods

It was a retrospective study conducted in Department of Pediatrics, NMCH, Jamuhar, Sasaram, Bihar, India for 10 months. Fifty infants with ABM were enrolled in the study with written informed consent from parents. Details were recorded in a pre-decided proforma. Including age, gender and detailed history followed by complete physical examination. Lumbar puncture was performed in all the children for CSF examination. Diagnosis of ABM was considered when CSF examination showed polymorphonuclear pleocytosis, low sugar and high protein for age and/or CSF culture positive for bacterial growth. Children with age more than 1 year, history of neurodevelopmental delay, tubercular meningitis, head injury, associated comorbid condition like HIV infection, heart disease, neural tube defects or Pathological hyperbilirubinemia in newborn period were excluded from study. Fundus examination was done to look for papilledema. ABM was treated as per standard guidelines. Some patients were also given dexamethasone. Radio imaging of brain was done if required. Ear examination was done by otorhinolaryngologist in all cases to rule out causes for conductive hearing loss. An audiological assessment of the patient was done at the time of discharge using Brainstem Evoked Response Audiometry (BERA) by "Intelligent Hearing system (I.H.S.)" the data was presented in an excel sheet and analyzed statistically using SPSS 21.0 software.

Results

Fifty children below 1 year of age diagnosed to have ABM were enrolled in the study. Cases were categorized into two groups as with SNHL present (group I) and SNHL absent (group II). Of the 50 children with ABM, 25(50%) were less than one month old, 19(38%) were 1-6 month, 6(12%) were

6-12 month of age. (Figure 1) 28(56%) were females and 22(44%) were males. Female to male ratio was 1.27:1. Refusal to feed, seizure and fever were the most common presenting symptoms in our study. (Table 1). 22(44%) had abnormal imaging (USG/CT/MRI) of Brain. (Table 2) Hydrocephalus being the most common abnormal finding was seen in 11 cases. 13(26%) out of 50 children developed SNHL following acute bacterial meningitis. SNHL was bilateral in 7(14%) and unilateral in 6(12%) cases. SNHL was mild in 3 patient, moderate in 5 patients, severe in 4 patients and only 1 patient had profound hearing loss. (Table 3). Incidence of SHNL was more in infants below one month. It was observed that incidence of SHNL was decreased as age increases but this trend is not statistically significant (p value=0.3). 25% of females and 27.27% of males developed SNHL, but no correlation was found between gender and occurrence of SHNL. Mean days of illness before admission was 3.44 days. There was no relationship found between no. of days of illness before treatment and development of SNHL. Amongst 27 cases with altered sensorium, 9 developed SNHL, which suggests that there is increased risk of development of SNHL in patient with altered sensorium in ABM which was statistically found to be significant (P- value 0.04) Amongst 13 cases with Glasgow coma scale (GCS) less \leq 8, 7 developed SNHL. Risk of developing SNHL was found to be higher in patient who had GCS less than 8. This correlation was found statistically significant (P value 0.02) but there was no significant correlation between fever, seizure and development of SNHL in patient with ABM. Out of the 13 cases of SNHL, etiology could be identified in 5(38.4%) cases. *S. Pneumoniae*, *E. coli*, *Klebsiella*, Gram positive cocci, Gram negative bacilli were responsible for 1 case each. Among 18 cases who have received dexamethasone, 6 developed SNHL, which was found to be statistically insignificant. Dexamethasone therapy was not associated with decreases incidence of SNHL in present study. Among 45 cases who had received amikacin in recommended dosages, 12 developed SNHL, which also was found to be statistically insignificant. (P-value: 1.0) Patient with high value for Mean \pm SD for Total nucleated cells in CSF for group I was 1675.4 ± 2318.6 while for group II it was 444.57 ± 360.3 . CSF Total Neutrophil count had more incidence of SNHL. (P value 0.003). Mean \pm SD for Protein (mg/dl) in CSF for group I was 415.2 ± 484.1 while for group II it was 204.1 ± 210.3 . Patient with higher value for CSF protein had more SNHL. (P value 0.036). Mean \pm SD for Sugar (mg/dl) in CSF for group I was 25.5 ± 15.8 while for group II it was 40.3 ± 15.9 . Patient with low value for CSF sugar have more SNHL. Among 9 cases with CSF sugar less than 20, 5 developed SNHL, which was statistically significant (P value 0.03).

Risk of developing SNHL is higher in patient who have CSF sugar less than 20. There was no significant difference in WBC, Hb and platelets count between 2 groups. Among 23 cases who had abnormal imaging of brain, 10 developed SNHL. Hydrocephalous, Acute infarcts, subdural effusion

and cerebral oedema, Ventriculitis with hydrocephalous, and subdural haemorrhage were few abnormal findings noted in brain imaging. Though patients with abnormal imaging of brain had more SNHL but this observation was not statistically significant in present study.

Table 1: Symptoms in ABM and their frequency

Findings	Frequency=50	%
Refusal to feed	36	72
Seizure	34	68
Fever	32	64
Altered sensorium	27	54
Irritability	26	52
Vomiting	11	22
Excessive cry	15	30
Hypothermia	2	4

Table 2: Abnormal findings on brain imaging in ABM and its percentage

Findings	Frequency=50	%
Hydrocephalous,	9	18
Acute infarcts	4	8
Subdural effusion and Cerebral oedema	3	6
Ventriculitis with hydrocephalus	2	4
Subdural haemorrhage	1	2

Table 3: Distribution of SNHL in children with acute bacterial meningitis

SENSORY NEURAL HEARING LOSS	Number if cases=13	%
Mild(40-55 dB)	3	6
Moderate (55-70dB)	5	10
Severe (70-90dB)	4	8
Profound (>90dB)	1	2
Total	13	26

Discussion

Of the 50 children enrolled in the study, 13(26%) developed SNHL following acute bacterial meningitis which was found to be similar to other studies where incidence of SNHL was 25.7%, 28.1% and 20.8% in ABM. [9-11] Widely variable incidence of SNHL in ABM has been reported in studies done in India. Variations of incidence ranged from 36.6% to 64% on higher end while in some studies reported much lower incidence of 6%- 7%. [12-16] In the present study, SNHL was bilateral in 14% and unilateral in 12% cases, which was found to be similar when compared with a study done by Damodaran et al., where bilateral SNHL was present in 11.4% and unilateral SNHL was present in 14.3% of cases. [9] Taylor et al. reported an incidence of 11.3%, with 6.1% being unilateral and 5.2% being bilateral. [17] Higher Incidences of bilateral SNHL were seen in some other studies.10,12,18 Lower incidence of bilateral hearing loss of only 4% along with unilateral SHNL of 3% is seen in a study done by George CN et al.¹⁶ In studies done by Cherian et al. unilateral SNHL was present in 6.2 %, which was much lower as compared to results of our study. [10]

In our study, 6% had mild SNHL, 10% had moderate, 8% had severe and 2% had profound SNHL. In study done by Cherian et al. 3.14% had mild SNHL, 12.47% had moderate, and 12.47% had severe profound SNHL. In a study by Singh et al., 6.6% had severe and 30% had moderate SNHL. [13] These variations in the incidence and type of SNHL can be due to various other factor affecting development of SNHL in patient with ABM. Out of the 50 cases of ABM, aetiology could be identified in 5 cases of which S. Pneumoniae, E. Coli, Klebsiella, Gram positive cocci, Gram negative bacilli were responsible for 1 case each and all 5 cases developed SNHL. No definite aetiology for ABM could be identified in most cases as patients had already received antibiotics before being referred to us. Panjarathinam R et al. concluded that culture positivity varied form 12.12 to 56%. [19] In a study, Hemophilus was the most common bacteria (49%) causing ABM, followed by Pneumococcus (22%) and Meningococcus (15%). [20] Higher incidence of hearing loss is seen in pneumococcal meningitis. [5,21,22] In present study there was no association found between sex of patient and incidence of ABM (P value = 1). Similar results

were seen in study done by Singh et al. In the study by Gupta V, age and sex of the patient were not significant risk factor in the development of hearing impairment. Refusal to feed (72%), seizure (68%), fever (68%) and altered sensorium (54%) and irritability (52%) were the most common presenting symptoms in our study. From a study done by Roine et al. with bacterial meningitis, approximately 78% were lethargic, 7% were somnolent, and 15% semi-comatose or comatose at the time of admission. [23] In present study, no correlation was found between fever, seizure and development of SNHL in cases with ABM. It was similar to study by Charuvanij et al. where no significant correlation was observed between hearing loss and various clinical and demographic factor.¹⁸ Cherian et al. observed that the total duration of fever was significantly different in those with and without SNHL ($P < 0.05$) but presence of seizure was not significantly associated with SNHL.¹⁰ Seizure was found to be a strong predictor for hearing loss in ABM in various other studies. [12-24] In our study, altered sensorium and GCS < 8 were significantly associated with increased risk of SNHL in patient with ABM (P value = 0.04 and 0.02). Roine I et al. also observed that the level of consciousness is the most important predictor of neurological sequel.²³

Cherian et al. observed that presence of altered sensorium were not significantly different in those with and without SNHL.¹⁰ In another study by Kapoor et al., modified GCS $< \text{or} = 8$ was significantly associated with abnormal BAER with P value < 0.001 .¹² In the present study CBC parameter were not significantly associated with hearing impairment. CSF pleocytosis was significantly associated with development of SNHL in the present study with p value of 0.03 and was consistent with other studies where CSF pleocytosis was strongly correlated with SNHL. [9,25,26] In study by Bhatt SM, onset of hearing loss was preceded by a CSF leucocytosis of > 2000 cells/mm³.²⁵ Increased CSF protein levels were significantly associated with SNHL in the present study with p value of 0.036 which was found to be consistent with other studies where high protein in CSF was associated with a significantly higher risk of hearing loss. [10,11] Association of low CSF sugar with SNHL was statistically significant in the present study (P value = 0.006) which was found to be similar with the other study. [11,26] In study by Kapoor et al., CSF sugar ($< 20\text{mg}\%$) was significantly associated with abnormal BAER (P value < 0.05). [12] Among 18 cases who received dexamethasone, 6 developed SNHL. Dexamethasone therapy was not associated with decreases incidence of SNHL in present study which was comparable with other studies. [21,27] Contrary to above observation, significant reduction in incidence of hearing loss after dexamethasone therapy was noted by Girgis NM et al. [28] In

present study, amikacin was not associated with increased risk of SNHL, which was similar to study done by Cherian B et al. where aminoglycoside usage were not significantly different in those with and without SNHL.¹⁰ Patients with abnormal imaging of brain had more SNHL compared to those with normal, but there was no correlation found between abnormal imaging of brain and increased incidence of SNHL in present study, this is similar to study by Singh K et al., where no relation was observed between hydrocephalous, subdural effusion and higher incidence of SNHL.

Conclusions

The present study throws light on the occurrence of sensorineural hearing loss following acute bacterial meningitis. SNHL, which if left untreated may lead to serious handicap affecting the linguistic performance and overall development of the child. Hence our study emphasizes the need for complete audiological evaluation of a child recovered from meningitis. BERA is a helpful tool for screening the sensorineural hearing loss especially in the young children and infants in whom other conventional methods may not be of much use. With the advent of newer vaccines, the incidence of bacterial meningitis and its sequelae has dramatically decreased in the developed countries.

References

1. López-Cortez LM, Jiménez-Galán R, Jiménez-López JL, et al. Sensorineural hearing loss in children following bacterial meningitis: A review of the literature. *Int J Pediatr Otorhinolaryngology*. 2020;134:110020. doi:10.1016/j.ijporl.2020.110020.
2. van de Beek D, Brouwer MC, Hasbun R, Koedel U, Whitney CG, Wijdicks EFM. Community-acquired bacterial meningitis. *Nat Rev Dis Primers*. 2016;2:16074. doi:10.1038/nrdp.2016.74.
3. Olusanya BO, Neumann KJ, Saunders JE. The global burden of disabling hearing impairment: A call to action. *Bull World Health Organ*. 2014;92(5):367-373. doi:10.2471/BLT.13.128728.
4. Chadha SK, Pappas DG, Liu Y, et al. Early detection of hearing loss following bacterial meningitis in children: Experience of a large tertiary care pediatric hospital. *Int J Pediatr Otorhinolaryngol*. 2021;143:110666. doi:10.1016/j.ijporl.2021.110666.
5. Grimwood K, Anderson VA, Bond L, Catroppa C, Hore RL, Keir EH. Adverse outcomes of bacterial meningitis in school-age survivors. *Pediatrics*. 2012;129(5). doi:10.1542/peds.2011-2758.
6. Peltola H, Roine I. Improving the outcomes in children with bacterial meningitis. *Curr Opin*

- Infect Dis. 2009;22(3):250-255. doi:10.1097/QCO.0b013e32832b354d.
7. Saez-Llorens X, McCracken GH Jr. Bacterial meningitis in children. *Lancet*. 2003;361(9375):2139-2148. doi:10.1016/S0140-6736(03)13693-8.
 8. Brouwer MC, Tunkel AR, van de Beek D. Epidemiology, diagnosis, and antimicrobial treatment of acute bacterial meningitis. *Clin Microbiol Rev*. 2010;23(3):467-492. doi:10.1128/CMR.00070-09.
 9. Koomen I, Grobbee DE, Roord JJ, Donders R, van Furth AM. Hearing loss at school age in survivors of bacterial meningitis: Assessment, incidence, and prediction. *Pediatrics*. 2003;112(5):1049-1053. doi:10.1542/peds.112.5.1049.
 10. Cherian B, Singh T, Chacko B, Abraham A. Sensorineural hearing loss following acute bacterial meningitis in non- neonates. *Indian J Pediatr*. 2002 Nov; 69(11):951-955.
 11. Gupta V. Hearing evaluation in children with bacterial meningitis. *Indian Pediatr*. 1993 Oct; 30(10):1175-1179.
 12. Kapoor RK, Kumar R, Misra PK, Sharma B, Shukla R, Dwivedee S. Brainstem auditory evoked response (BAER) in childhood bacterial meningitis. *Indian J Pediatr*. 1996; 63 (2):217–225.
 13. Singh K, Mann SB, Gupta AK, Kumar L. Auditory profile in children recovering from bacterial meningitis. *Indian J Pediatr*. 1996 Mar-Apr; 63(2):210-216.
 14. Karanja BW, Oburra HO, Masinde P, Wamalwa D. Prevalence of hearing loss in children following bacterial meningitis in a tertiary referral hospital. *BMC Res Notes*. 2014;7:138. Published 2014 Mar 11. doi:10.1186/1756-0500-7-138
 15. Chandran A, Herbert H, Misurski D, Santosham M. Long-term sequelae of childhood bacterial meningitis: an underappreciated problem. *Pediatr Infect Dis J*. 2011; 0 (1):3–6.
 16. George CN, Letha S, Bai SS. A clinical study of chronic morbidity in children following pyogenic meningitis. *Indian Pediatr*. 2002 Jul; 39(7):663-667
 17. Taylor HG, Mills EL, Ciampi A, Berger RD, Watters GV, Macdonald N et al.. The sequelae of *Haemophilus influenzae* meningitis in school-age children. *N Engl J Med*. 1990 Dec 13; 323(24):1657-1663.
 18. Charuvanij A, Visudhiphan P, Chiemchanya S, Tawin C. Sensorineural hearing loss in children recovered from purulent meningitis: a study in Thai children at Ramathibodi Hospital. *J Med Assoc Thai*. 1990 May; 73(5): 253-257.
 19. Panjarathinam R, Shah RK. Pyogenic meningitis in Ahmedabad. *Indian J Pediatr*. 1993 Sep-Oct; 60(5):669-673.
 20. Duclaux R, Sevin F, Ferber C, Draï MF, Dubreuil C. Brainstem auditory evoked potentials following meningitis in children. *Brain Dev*. 1993;15(5):340–345.
 21. Wellman MB, Sommer DD, McKenna J. Sensorineural hearing loss in postmeningitic children. *Otol Neurotol*. 2003 Nov;24(6):907-912.
 22. Kutz JW, Simon LM, Chennupati SK, Giannoni CM, Manolidis S. Clinical Predictors for Hearing Loss in Children With Bacterial Meningitis. *Arch Otolaryngol Neck Surg*. 2006; 132(9):941–945.
 23. Roine, I, Peltola, H, Fernández et al., Influence of admission findings on death and neurological outcome from childhood bacterial meningitis. *Clin Infect Dis* 2008; 46(8):1248-1252.
 24. Borkowski WJ Jr, Goldgar DE, Gorga MP, Brookhouser PE, Worthington DW. Cerebrospinal fluid parameters and auditory brainstem responses following meningitis. *Pediatr Neurol*. 1985; 1(3):134–139.
 25. Bhatt SM, Lauretano A, Cabellos C et al.. Progression of hearing loss in experimental pneumococcal meningitis: correlation with cerebrospinal fluid cytochemistry. *J Infect Dis*. 1993 Mar; 167(3):675-683.
 26. Nadol JB Jr. Hearing loss as a sequela of meningitis. *Laryngoscope*. 1978;88(5):739–755
 27. Sankar J, Singhi P, Bansal A, Ray P, Singhi S. Role of dexamethasone and oral glycerol in reducing hearing and neurological sequelae in children with bacterial meningitis. *Indian Pediatr*. 2007 Sep; 44(9):649-656.
 28. Girgis NI, Farid Z, Mikhail IA et al.. Dexamethasone treatment for bacterial meningitis in children and adults. *Pediatr Infect Dis J*. 1989 Dec;8(12):848-851.