Brain Stem Auditory Evoked Responses and Visual Evoked Responses in Children with Tubercular Meningitis

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Thirty two patients between 6 months and 14 years of age with tubercular meningitis were evaluated for brain stem auditory evoked response (BAER) and Visual evoked responses (VER), within 7 days of admission. Absolute latencies and interpeak latencies were compared with values obtained from normal children. BAER abnormality was found in 56.25% and VER in 28% children, respectively. BAER abnormality correlated with Glasgow Coma Scale at admission and discharge, stage of meningitis, raised intracranial pressure, seizure activity, and poor outcome. VER abnormality correlated with abnormal fundus findings only.

Keywords: *Brainstem Auditory Evoked Responses (BAER),Tubercular meningitis (TBM). Visual Evoked Responses (VER).*

TUBERCULAR meningitis (TBM) is one of the major causes of mortality and morbidity in Indian children. It can cause long-term sequelae, like mental retardation, language and behavioral problems and impaired vision and hearing(1,2). Evoked response or potential is an electrical activity appearing in the EEG with appearance, disappearance or change in stimuli (visual, auditory). Brain stem auditory evoked response (BAER) studies may provide an objective non-invasive documentation of brain stem functions. In patients with optic neuritis the visual evoked response (VER) of involved eye has an increased latency period and decreased amplitude compared to normal. This occurs even when there is no decrease in visual acuity, colour perception or visual field defect.

The present study was undertaken to evaluate BAER and VER in patients with tubercular meningitis during acute phase and to asses correlation between abnormal evoked responses and certain selected clinical parameters.

Subjects and Methods

The study group comprised of consecutive cases of tubercular meningitis (TBM) admitted between December 2003 to August 2004 to the Department of Pediatrics, NSCB Medical College, Jabalpur. Diagnosis of TBM was based on history, clinical examination and CSF findings and sterile bacterial culture. 36 patients fulfilled the criteria. Four patients whose clinical condition did not allow them to be shifted to physiology lab were not included in the study. Each of the remaining 32 patients underwent ophthalmologic and ear examination to rule out primary pathology of vision and hearing before subjecting them to Evoked potentials (EP) studies. British Medical research council criteria were used to identify stage of TBM(3). Patients with abnormal sensorium were scored as per modified Glasgow coma scale(3). As soon as

clinical condition improved patients were shifted to physiology laboratory for evoked potential investigations. All patients were evaluated within 7 days of admission. 50% (n = 16) were evaluated within 24 hours of admission.

Brain stem auditory response (BAER) and visual evoked potentials (VEP) were performed in physiology laboratory. Recordings were obtained using an EP recorder Neurocare 2000 (Biotech India 2000 model). BAER was recorded with 0.1 ms rarefaction click stimulation delivered monoaurally at 95 db. Uniform stimulus strength was used in all patients because most of our patients had altered level of consciousness. The stimuli were delivered at the rate of 10 Hz. two thousand responses were averaged twice with a band pass filter of 100 Hz-3KHz. The latencies of different waves and interpeak latency were measured. VEP was recorded by flash method (LED goggle) in all patients, as most of them were not cooperative. Stimulus was given at the rate of 1 pps with a gain of 1-2 mv. Four hundred responses were averaged twice with a band pass filter of 2 Hz-180 KHz.

Controls 32 children matched in age and belonging to similar macro environment background were evaluated and data obtained from them was used to calculate normal values. Mean ± 2.5 SD of absolute latencies of individual waves and interpeak latencies of different waves in children below two years of age (n = 9) and above two years of age (n = 23), were calculated for both for BAER and VEP. The abnormality in each response was defined as absent response, prolonged latencies of individual waves (mean $\pm$ 2.5 SD) and prolonged interpeak latency between the waveforms (mean + 2.5 SD). Unpaired 't' test was used for quantitative variables. Pearson correlation was performed between abnormalities of evoked responses and clinical and investigational parameters.

Results

Thirty two patients with a mean age of 5.6 yr (11 months - 14 yr) were included in the study. Nine children were less than two years of age. Twentysix children were malnourished according to IAP classification. GCS Score at the time of admission ranged from 5-15 (mean 9.5). Presenting symptoms included fever (96%), drowsiness (75%), vomiting (65.6%), headache (31.2%) convulsions (68.2%), and neurological deficit (40.6%), more than two third (68.7%) were in stage III of TBM. Abnormal fundus findings were noted in 7 (21.8%) children. Papilledema was found in 4 patients and optic atrophy in three patients. On CT scan, 15/23 patients had hydro-cephalus. Four patients died during the hospital stay.

BAER abnormality was found in 56.2% of patients; these were categorized into three groups: (*i*) Bilateral absent response denoting sensorineural hearing loss (n = 2); (*ii*) prolonged interval with prolonged latency denoting brainstem dysfunction (n = 11); and (*iii*) prolonged latency only (n = 5). VEP were abnormal in 9 (28.1%) children; of these 4 had absent response. Absolute latencies and interpeak latencies of different waves of BAER and VER in patients were found to be greater than the upper limit of normal children (*Table I*). Most common abnormality in BAER was prolongation of wave III latency (n = 14) followed by prolongation of wave II latency (n = 10), wave II latency (n = 9) and interpeak latency I-III (n = 8). Two-tailed correlations of BAER and VEPs with clinical findings showed significant correlation between BAER abnormality and modified GCS Score at discharge (r = 0.87), mortality (r = 0.87), modified GCS at admission(r= 0.77), TBM

stage III (r = 0.68), seizure activity (r = 0.65) and raised ICP (r = 0.57). VEPs significantly correlated with abnormal fundus findings only(r = 0.787).

Discussion

Hearing impairment in bacterial meningitis is well-documented(4,5), but there is paucity of studies in tubercular meningitis. Kapoor, *et al.*(6) in a study on 50 children reported 52% prevalence of abnormality, which is similar to our observation. Topcu, *et al.*(7) found lesser frequency of BAER abnormality (24%) in TBM children at admission. Kalitha, *et al.*(8) reported 66% occurrence of BAER abnormality in adult patients with TBM.

The cause of BAER abnormality could be multifactorial. Bilateral and unilateral absent response may be due to involvement of VIIIth nerve due to basal exudates, toxic effects and vascular involvement. Disruption of brainstem function may be directly related to raised ICP, which is a common finding in TBM. Nagao, *et al.*(9,10) observed disruption of neural activity in rostral auditory brainstem following experimentally induced ICP in cats. Raised ICP can lead to compression ischemia by alteration in blood flow in the penetrating vessels of basilar and posterior cerebral artery producing transient BAER abnormality. In our study abnormal BAER had a correlation coefficient of 0.57 with raised ICP on bivariate analysis. Moreover, all 15 children having either type of hydrocephalus had some form of BAER abnormality.

The diversity of BAER abnormalities and lack of specific pattern in our study may be due to the diversity of pathophysilogical mechanism in TBM including hydrocephalus, infarctions, tuberculoma and varying degree of raised ICP.

VEP abnormalities in TBM can occur due to compression of optic nerve or entrapment of nerve by exudates and optochiasmatic

TABLE I–*BAER and VER Latencies in Patients with TBM and age Matched Controls*

Wave latency	Age < 2 years Latency-in Millisecs					Age> 2 years Latency in Millisecs				
		Cases		Control			Cases		Control	
BAER	N	Mean	S.D	Mean	SD	N	Mean	SD	Mean	SD
I	18	1.91*	0.28	1.72	0.1	42	1.87@	0.23	1.7	0.13
II	18	2.79	0.47	2.72	0.16	42	2.77*	0.26	2.72	0.18
III	18	3.95@	0.43	3.63	0.14	42	3.91@	0.32	3.63	0.22
IV	18	4.93*	0.46	4.81	0.23	42	4.78#	0.45	4.76	0.28
V	18	5.91@	0.52	5.62	0.25	42	5.79@	0.36	5.64	0.26
I-III	18	1.98*	0.34	1.91	0.11	42	1.88*	0.34	1.92	0.14
III-V	18	1.98*	0.24	1.92	0.19	42	1.88*	0.34	2.01	0.15
I-V	18	4.10*	0.42	3.9	0.23	42	3.9*	0.33	3.6	0.23
VER	N	Mean	SD	Mean	SD	N	Mean	SD	Mean	SD
N_75	16	76.91*	9.7	71.76	6.42	40	79.59@	10.1	69.36	6.32
P _100	16	105.97@	8.25	97.69	7.74	40	116.09@	13.4	97.65	6.48
P_135	16	143.08*	23.21	129.49	7.73	40	149.87@	18.4	128.5	10.55

P <0.05, @P <0.01, # Not significant, N is 2 times the number of patients as there are two ears, two eyes in each individual.

Key Messages

- Brain stem auditory evoked response (BAER) were found to be abnormal in 56.25% of 32 children with tubercular meningitis (TBM).
- Abnormal BAER had significantly positive correlation with modified Glasgow Coma Scale (GCS) score at discharge, modified GCS score at admission, TBM stage III, raised intracranial pressure, death and seizure activity.
- Abnormal BAER during acute phase of TBM seems to be correlated with the severity of disease.

arachnoiditis(11,12). Extrinsic compression of anterior visual pathways results in loss of amplitude, distortion of waveform and prolongation of P100 latency, even in a patient with normal visual acuity, field of vision and fundus examination(13).

We conclude that BAER and VER abnormality is often seen in acute phase of TBM. It is desirable to undertake more studies and to have longitudinal follow-up to define the long-term significance of evoked potential abnormality in patients with TBM.

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