Brainstem Auditory Evoked Potentials and Electrocochleographic Findings in Patients with Idiopathic Sudden Sensorineural Hearing Loss

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ABSTRACT

Objective: To determine the status of cochlea, auditory pathway and hearing threshold by recording brainstem auditory evoked potential (BAEP) and electrocochleography in patients with idiopathic sudden sensorineural hearing loss (ISSNHL).

Study Design: Observational study.

Place and Duration of Study: Department of Clinical Physiology at King Abdul Aziz University Hospital, Riyadh, Saudi Arabia, from May 2002 to November 2007.

Methodology: Patients diagnosed with idiopathic sudden sensorineural hearing loss (ISSNHL) based on clinical features and pure tone audiometry were studied. Brainstem auditory evoked potential (BAEP) and electrocochleography (ECOG) was performed in them according to standard protocols for assessment of auditory pathway and hearing threshold.

Results: Out of 23 patients (14 males and 9 females) left ear was affected in 9 (39.1%) patients, right ear in 13 (56.5%) and both in 1 (4.3%). Absolute latency of wave I and wave V were significantly prolonged in affected ear compared to unaffected ears (p=0.0031), while interpeak I-V latency was significantly higher in affected ears versus unaffected ears (p=0.0544). Six patients (26.1%) had type II Diabetes mellitus, five cases (21.7%) had hypertension and 5 cases (21.7%) had dyslipidemia. ECOG revealed absence of summation potential (SP) and action potential (AP) response even at 95 dB in 17 out of 23 cases (73.9%).

Conclusion: Patients with ISSNHL had significant abnormalities in BAEP and ECOG recordings showing predominantly cochlear involvement. Thus, these tests provide useful diagnostic information in patients with ISSNHL in addition to pure tone audiometry.

Key words: Idiopathic sudden sensorineural hearing loss. Brainstem auditory evoked potential. Electrocochleography. Hearing loss. Cochlea.

INTRODUCTION

Idiopathic sudden sensorineural hearing loss (ISSNHL) has an incidence of approximately 20/100,000 people per year, where young and otherwise healthy people are often affected. ISSNHL is defined as the sudden onset of unilateral sensorineural hearing loss of > 30 dB over at least three contiguous audiometric frequencies.¹ ISSNHL is defined as deafness of sudden onset and unknown origin. The pathogenesis of ISSNHL is still controversial. Various potential causes include atherosclerosis, viral infection, vascular occlusion, cochlear membrane breaks, and neurologic disorders.²⁻⁴ Diabetes mellitus can cause microvascular changes involving sudden increase in blood viscosity and embolic and thrombotic episodes;⁵ and diabetes may be considered

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a major cause of ISSNHL. Since the incidence of Diabetes mellitus is increasing, the contributions of Diabetes to the occurrence, severity and outcome of ISSNHL warrant careful assessment.⁶

ISSNHL represents 1% of all sensorineural hearing loss cases and is usually unilateral with mild to severe temporary or permanent hearing loss.⁷ In most cases of ISSNHL, the cause is unknown ISSNHL is typically unilateral, with only 8% of the cases occurring bilaterally.⁸ The diagnosis is mostly limited to clinical presentation and audiogram findings.⁹⁻¹² Very few studies have evaluated brainstem auditory evoked potential (BAEP) and electrocochleography (ECOG) responses in patients with ISSNHL. Busaba *et al.* reported the significance of auditory brainstem response (ABR), but the details of ABR were not reported. Secondly it was reported that ABR was normal in 2/3 cases of ISSNHL.¹³

Brainstem auditory evoked potential (BAEP) and electrocochleography (ECOG) are transient responses, that provide valuable information on hearing thresholds and useful for differential diagnostics of auditory nerve pathway. ISSNHL is a well defined entity, but evaluation of this condition with brainstem auditory evoked potential (BAEP) and electrocochleography (ECOG) is sparse.

The aim of the present study was to assess the status of cochlea, auditory pathway and hearing threshold in patients with ISSNHL by recording brainstem auditory evoked potential (BAEP) and electrocochleography (ECOG).

METHODOLOGY

It was an observational study conducted in the Department of Clinical Physiology at King Abdul Aziz University Hospital, Riyadh, Saudi Arabia, from May 2002 to November 2007. Patients were diagnosed with ISSNHL and referred to the department for neuro-physiological check-up. In all the cases, brainstem auditory evoked potential (BAEP) and electro-cochleography (ECOG) was performed according to standard protocols.

Auditory function was determined by pure-tone audiometry from 125 Hz to 8 kHz, and was expressed as mean hearing levels from 250 Hz to 4 kHz.

Criteria for diagnosis of ISSNHL were sudden onset of sensorineural hearing loss (in 3 days or less) of unknown origin. The diagnosis of ISSNHL is made on the basis of perceptive hearing loss, etiology that remained unknown after clinical, laboratory and imaging studies, hearing loss occurring within 24 hours nonfluctuating, severity of the hearing loss averaging at least 30 dB HL for three subsequent one octave steps in frequency as shown in the standard pure-tone audiogram and blank otological history in an otherwise healthy individual.⁹⁻¹² All patients with history of trauma, infections and noise pollution were excluded.

BAEP and ECOG were done using an averaging technique using Nicolet Spirit (USA). A vertex to ipsilateral earlobe derivation was used. A total of 2000 click responses were recorded and averaged. The threshold of hearing, absolute peak latency for waves I, III and V, interpeak latency of I-III, III-V and I-V and latency intensity curve for wave V at 80 dB, 60 dB, 40 dB and 20 dB were used for clinical interpretation.

Hearing threshold level was determined from the recording of wave V threshold. Interpretation of ABR included absolute and interpeak intervals and presense/ absence of waves. Hearing threshold level was done at different frequencies. Table I shows normal reference range values of absolute and interpeak latencies of BAEP waves in clinical physiology laboratory at King Abdul Aziz University Hospital. Table II shows criteria used to assess the severity of hearing loss.

The data was analyzed by computer software program Statistical Package for Social Sciences (SPSS Version 10). Descriptive characteristics of the study patients were calculated as Mean \pm SD (standard deviation) for continuous variables and as percentages for categorical variables. The tests applied for statistical analysis was Student's t-test. A p-value of \leq 0.05 was taken as statistically significant.

RESULTS

A total of 23 cases who fulfilled the criteria for diagnosis of ISSNHL were finally selected for the study. Fourteen (60.9%) patients were males and 9 (39.1%) were females. Left ear was affected in 9 (39.1%) patients, right ear in 13 (56.5%) and both in 1 (4.3%) case. Therefore, the number of affected ears was 24 and unaffected ears were 22. The mean age of patients was 40.26 ± 10.66 years. Table III shows comparison of absolute and interpeak latencies between the unaffected (n=22) and affected ears (n=24). Absolute latencies of both wave I and wave V were significantly prolonged in affected ears compared to unaffected ears (p=0.0031), while in interpeak latency, I-V latency was

 Table I:
 Normal reference range values of absolute and interpeak latencies in milliseconds (ms) of BAEP at clinical physiology laboratory of King Saud University Hospital.

Wave	Latency (ms)		
Absolute latencies			
I	1.7 ± 0.15		
II	2.8 ± 0.17		
111	3.9 ± 0.19		
IV	5.1 ± 0.24		
V	5.7 ± 0.25		
Interpeak latencies			
1-111	2.1 ± 0.15		
1-V	4.0 ± 0.23		
III-V	1.9 ± 0.18		

Table II: Criteria used to assess different categories of hearing impairment according to the severity of hearing loss at clinical physiology laboratory of King Saud University Hospital (ABR is done at an increment of 10 dB).

Severity grade	Hearing loss in dB			
Mild	30,40			
Moderate	50,60			
High (severe)	70,80			
Very high (very severe)	> 80			
Profound	No waves are formed			

Table III: Comparison	of	absolute	and	interpeak	latencies	in
milliseconds (ms)	between	affecte	ed and unaf	fected ears	in
all ISSNHL pa	atien	ts.				

	Affected ear	Unaffected ear	p-value			
	(n=24)	(n=22)				
Absolute latency (ms)						
Wave I	2.15 ± 0.39	1.83 ± 0.15	0.0031			
Wave III	4.04 ± 0.54	3.82 ± 0.34	0.1272			
Wave V	6.46 ± 0.85	5.86 ± 0.32	0.0031			
Interpeak latencies (ms)						
1-111	2.11 ± 0.56	2.06 ± 0.24	0.7425			
I-V	4.39 ± 0.87	3.81 ± 0.81	0.0544			
III-V	2.36 ± 0.72	2.15 ± 0.57	0.3426			

significantly higher in affected ears versus unaffected ears (p=0.0544) indicating retrocochlear involvement of auditory pathway. Six patients (26.1%) had Diabetes mellitus (DM) type II, five cases (21.7%) had hypertension and 5 cases (21.7%) had dyslipidemia. Three patients (13.0%) among them had co-existent DM, dyslipidemia and hypertension. ECOG revealed absence of SP and AP response even at 95 dB in 17 out of 23 cases (73.9%) which indicated severe damage to cochlea. In case of affected ears mild, moderate, high, very high and profound hearig loss was observed in 1 (4.3%), 2 (8.6%), 10 (43.5%), 7 (30.4%) and 3 (13.0%) cases respectively. In 7 (31.8 %) patients, the unaffected ear had mild increase in hearing threshold and another 7 cases (31.8%) had moderate to high hearing threshold in the unaffected ear. In the remaining 8 unaffected ears (36.4%), the hearing threshold was normal.

DISCUSSION

The diagnosis of ISSNHL is based on clinical presentation and audiometry at present. The cause cannot be determined with the currently available methods of clinical diagnosis. This study aimed to assess auditory pathway and hearing threshold by recording of BAEP and ECOG in patients with ISSNHL. In most of the patients with ISSNHL loss of hearing occurs in isolation. The evidence for a significant number of cases of ISSNHL, being due to ischemia of the cochlea seems to be more compelling. Indeed one might imagine that a number of causes of ISSNHL could act through this common endpoint, something that better techniques for studying the microcirculation of the labyrinth might one day clarify. The absence of SP and AP response even at 95 dB in the majority of this study subjects, supports the evidence of involvement of cochlea in ISSNHL. Known associations between sudden deafness and prothrombotic conditions such as polycythemia, sickle cell anemia,¹⁴ and chronic myeloid leukaemia¹⁵ already seem to be consistent with this mechanism.

The database that is present on ABR and ECOG findings in ISSNHL is controversial. Busaba et al. studied 96 patients retrospectively with the diagnosis of ISSHL during a 2-year period. Their workup included ABR testing and gadolinium-enhanced magnetic resonance imaging (Gd-MRI) studies. Judgments of abnormality were based on the analysis of ABR wave latencies, interwave latencies, and inter-ear-interwave latencies. They reported that out of 65 cases, 51 had normal while 14 had abnormal ABR.13 ABR was abnormal in all the presently reported cases. ABR was done at different dB values starting from 80 dB up to the values for wave V threshold. It was observed that threshold of wave V was moderate in 8 cases, while severe in 12 cases and profound in 3 cases. Wave I was absent in 17 affected ears (73.9%). ECOG revealed absence of SP and AP

response even at 95 dB in 17 out of 23 cases (73.9%). This shows that in most of those cases peripheral auditory pathway was involved and most probably the lesion was of cochlear origin. Acute hearing loss could have been due to cochlear infarction alone or more proximal damage to the auditory nerve or central auditory pathways. This explanation is supported by the observation that ECOG revealed absence of SP and AP response even at 95 dB in 73.9% of cases. Some degree of recovery of hearing loss is common, though less likely in those most profoundly affected.⁶

A recent study shows that Diabetes mellitus, hypercholesterolemia and a high burden of cardio-vascular risk factors are associated with the risk of ISSNHL.¹⁶ The present data also shows the same trend.

Weng *et al.* showed that onset of ISSNHL is common in summer and higher serum albumin concentrations are favourable prognostic factors of SSNHL in diabetic patients.¹⁷ There is seasonal variation in incidence of ISSNHL. This is the first report from Saudi Arabia of ISSNHL and this area is very hot in summer and moderate in winter seasons. However, most of the subjects (16/23) presented in early summer weeks. Further prospective studies are needed to confirm the true effects of these prognostic factors.

Rarely an undiagnosed diabetic patient may present to an otolaryngologist with sudden deafness as the only symptom.¹⁸

In diabetic patients with SSNHL, hearing loss in the contralateral ear and the profound type hearing loss in the affected ear are commonly noted. However, in this study one diabetic patient had profound hearing loss. The poor prognosis of sudden deafness in diabetic patients may be caused by pre-existing microvascular lesions in the inner ear, and the PPG level could be a risk factor indicator for cochlear dysfunction in diabetic patients.¹⁹

ISSNHL may be a presenting symptom of infarction. Lee and Baloh found that 8% of patients with vertebrobasilar ischaemia had unilateral loss of hearing at presentation.²⁰ In almost one-third of patients, hearing loss in isolation was the initial presenting symptom of infarction. If hearing loss precedes the potentially more disabling symptoms of more widespread posterior circulation stroke, its recognition offers a window of opportunity for intervention in the hope of limiting long-term disability. It is a question which could lend itself to a future need for a randomized trial. If hearing loss is persistent and recovery does not occur ABR could provide a guide for future cochlear implant (CI) in these patients especially when it is bilateral.

For cochlear implantation (CI), case selection is essential before implantation and this work may help to

categorize these cases beforehand. Thus, this database may be helpful to form a base to be added to the list of current emerging indications for cochlear implants. Only one case was observed to be bilateral, but at the same time cases with unilateral involvement had mild to moderate hearing loss in the contralateral ear in many cases. Hence, these cases may be potential candidates for CI in future as they may not benefit from hearing aids.²¹ Recent experiences have shown that those patients who simultaneously use a CI and a contralateral HA (so-called bimodal stimulation) significantly improve in speech perception, both in guiet and noise. This improvement is not only allowed by stereophonic hearing, but also by the improved sound quality experienced by the patients on bimodal stimulation compared to their own only CI use.22 It can be further suggested that some residual hearing in contralateral ear can be an emerging indication for cochlear implants.²³

Recent reports show that hearing improvement rate significantly correlated with the net distortion product otoacoustic emissions (DPOAEs) in ISSNHL. Extended tests are thus needed for better assessment and prognosis in these cases.²⁴ Moreover, the significant predictors in the predictive model for improvement of idiopathic sudden sensorineural hearing loss, include distortion product otoacoustic emission, auditory brainstem response, vestibular evoked myogenic potential, and audiometric types.²⁵

It is suggested that BAEP and ECOG should be performed in patients presenting with ISSNHL. These tests could provide a useful guide for future cochlear implants in patients with ISSNHL especially when it is bilateral.

CONCLUSION

Patients with ISSNHL had significant abnormalities in BAEP and ECOG recordings showing cochlear involvement predominantly. Thus, these tests provide useful diagnostic information in patients with ISSNHL in addition to pure tone audiometry.

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