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Original Article

Otoacoustic emissions and auditory brainstem responses in patients with sudden sensorineural hearing loss. Do otoacoustic emissions have prognostic value?

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Abstract

BACKGROUND: Sudden sensorineural hearing loss (SSNHL) is a perplexing condition for patients and there are many controversies about its etiology, audiologic characteristics, prognostic factors, and treatment.

METHODS: In this prospective study, we performed some audiologic tests, including PTA, IA, ABR, and OAE (TEOAE) before beginning treatment of 53 patients with SSNHL. We assigned the patients randomly to two treatment groups: oral steroids + acyclovir vs. intravenous urographin. Twenty-eight patients underwent Magnetic Resonance Imaging (MRI) of the Brain.

RESULTS: Of 53 patients (22 female and 31 male), 22 (41.5%) had negative or no signal to noise ratio and overall correlation in TEOAE. Twenty-six patients (49%) had positive overall correlations less than 50%, and 5 patients (4.4%) had overall correlations >50%. Fifteen patients (28. 3%) responded completely or well, 20 (37.7%) responded partially, and 18 (33.9%) had poor or no response to the treatment. The mean values for overall correlation in 3 subgroups of patients (no response, partial response, and complete response) were – 3. 5% (± 1/16%), +11% (± 1/99%), and +36.6% (± 3/07%), respectively (P = 0.01). Twenty out of 52 patients had no reproducible wave in ABR (38.5%), and waves I, III, and V were absent in 40 (77%), 31 (59.6%) and 21 (40%) patients, respectively. There were some limitations (false positive and false negative results) in ABR use in our cases, but it may be useful in detecting site of lesion in SSNHL. Overall, according to the results of OAE, ABR, and brain MRI of these patients, 3 were affected by acoustic neurinomas, at least 1 had auditory neuropathy, and the site of lesion was cochlear in 6, and cochlear + retrocochlear in 13 patients.

CONCLUSIONS: ABR has limitations for use in SSNHL and seems not to obviate the need for brain MRI, but may help in determining the site of lesions such as ischemia or neuropathy. Overall correlation (and S/N ratio) in TEOAE is a valuable prognostic factor in SSNHL, hence we recommend performing TEOAE in every patient with SSNHL.

KEY WORDS: Sudden sensorineural hearing loss, pure tone audiometry, otoacoustic emissions, overall correlation, signal to noise ratio (S/N ratio), auditory brainstem responses, Interpeak latencies, retrocochlear lesion.

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Sudden sensorineural hearing loss (SSNHL) means loss of hearing more than 30 dB in 3 contiguous frequencies that occurs in less than 3 days. It is a controversial topic in otolaryngology, and more than 100 different causes may produce it [1, 2]. It is upon the clinicians to find the specific cause, evaluate prognostic factors and treat this stressful condition [1-3]. It is generally accepted that otoacoustic emissions (OAEs) reflect the preneural biomechanical processing activity of cochlea, especially outer hair cells (OHCs). These emissions could be recorded from the external auditory canal, spontaneously or after being evoked by some acoustic stimuli (e.g. clicks) [4].

The Auditory Brainstem Response (ABR) is composed of 5-7 waves produced by particular

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parts or stations in retrocochlear auditory pathway (from auditory nerve to brainstem and subcortex). It has been shown to possess excellent accuracy in indicating functional (software) integrity of the auditory tract [4]. It can also help detect retrocochlear lesions such as tumors.

Few studies have been conducted on the role of OAEs and ABR, especially on the prognostic value of OAEs in SSNHL. The aim of this study was to address the following questions: Can ABR and OAEs help us in detecting the site of lesion in SSNHL? Is there any difference in the outcome of SSNHL based on the results of OAEs tests?

Methods

This prospective study was performed in Kashani Hospital, Isfahan, Iran, between June 2003 and November 2005. Only 53 out of more than 85 patients with SSNHL who were referred to the Otolaryngology Clinic were included in the study. Patients with known causes of SNHL (such as Meniere's disease, acoustic trauma, otitis media, multiple sclerosis), patients who were more than 2 weeks past their onset of HL, and patients who were treated for SSNHL before being referred to us were excluded from the study.

Every patient underwent physical examinations and laboratory assessments such as PTA, IA (Amplaid 728 clinical, Amplaid 314 clinical), ABR (Amplaid MK22 clinical), and TEOAE (Capella, MADSEN clinical version 2.10, 2001). All tests were performed in identical conditions and by one skilled operator. All patients were recommended to undergo brain MRI with gadolinium, but only 28 opted to do so.

We assigned the patients randomly to two treatment groups: oral steroids (prednisone, 60 mg, PO daily for 10 days and then tapered) + acyclovir (800 mg, PO, tid for 7 days) vs. intravenous urographin 76% (2 cc for the first dose, and then 1 cc daily for 10 days). Post-treatment PTA and SDS were performed two weeks after termination of treatment. The patients were classified in three groups according to their response to treatment: complete or good response group (≥30 dB recovery in affected frequencies in PTA or ≥30% increase in SDS), partial or moderate response group (≥10 dB and ≤30 dB recovery in affected frequencies or ≥10% and <30% increase in SDS), and poor or no response group (≤9 dB recovery in PTA or ≤9% increase in SDS (1,8). We tried to determine site of lesion in these patients according to the results of ABR, OAEs, and MRI. Data, especially pretreatment test (OAEs and ABR) results of the three treatment response groups were analyzed with SPSS software using chi-square and ANOVA tests.

Results

In 53 patients (22 female and 31 male) with a mean age of 40.1 (± 15.3) years, right and left ears were involved somewhat equally and 4 patients (7.5%) had bilateral SSNHL. The mean value of hearing loss was ≥65 dB in 65% of patients, and the most common pattern in PTA curves was the flat pattern (70% of cases). After completion of investigations and treatments, of 28 patients who underwent brain MRI with gadolinium, 3 were found to have acoustic neurinomas. No significant difference was noted between the two treatment modalities. According to the PTA and SDS performed 2 weeks after completion of treatments, 15 patients (28.3%) responded completely, 20 (37.7%) responded partially, and 18 (33.9%) exhibited poor or no response. In ABR, 20 of 52 patients (38.5%) had no reproducible waves, and waves I, III, and V were absent in 40 (77%), 31 (59.6%), and 21 (40%) cases, respectively. Only 7 patients had normal ABR parameters (table 1). Interaural absolute latency differences of wave V (ILD5) were greater in retrocochlear causes (tumors) in SSNHL which were detectable in 31 patients (59.6%) (table 1). Of 53 patients, 22 (41.5%) had negative or nil signal to noise ratio.

Twenty-six patients (49%) had positive overall correlations less than 50% in TEOAE, and 5 (4.4%) had overall correlations >50%. Three patients had no wave or had abnormal interpeak latencies (IPLs) in ABR, while they had positive overall correlation (>40%) in TEOAE. One patient had normal brain MRI

and responded partially to the treatment (steroid + acyclovir) while the other two did not undergo MRI owing to financial restraints, but were treated completely with our treatment regimens. Thirteen patients had no wave in ABR (no waves at all or absent I or III waves) and attenuated (<20%) or negative overall correlations in TEOAEs, and normal MRI (table 2). ABR was normal in six patients with SSNHL but OAEs were abnormal (overall correlation less than 40%), and brain MRI was normal in four. Three of these patients responded completely, 2 responded partially, and only one displayed no response to the treatment.

Out of 28 patients undergoing MRI, 3 had acoustic neurinoma detected by MRI (table 3). Out of 22 patents with nil or negative overall correlation (and signal to noise ratio) in TEOAEs, 12 (54.5%) showed no response to therapy. Conversely, all of the 5 patients with overall correlation >50% in their TEOAE responded completely (3 patients) or partially (2 patients). On the other hand, of 31 patients with positive overall correlation and S/N ratio, only 6 did not respond to treatments and all had overall correlation less than 20% and had severe to profound hearing loss at their submitting. The mean values for each of the above-mentioned response groups in the two treatment modalities are summarized in table 4.

Table 1. IPL (I-III), IPL (1-V), IPL (III-V), and ILD-5 in 52 patients with SSNHL. 20 cases had not any wave (values in parenthesis are of contralateral ear).

Case no	IPL (1-III)	IPL (1-V)	IPL (III-V)	(ILD–5)	MRI results
2	-	-	2.12 (2.04)	+ 0.08	-
3	-	-	-	+ 0.1	Normal
4	-	-	1.92 (2.01)	+ 0.29	-
5	2.16 (1.87)	4.08(4.03)	1.92 (2.10)	- 0.28	Normal
8	1.92 (2.16)	4.08(3.4)	2.16 (1.74)	+ 0.12	Normal
11	1.56 (2.06)	3.24(3.74)	1.96 (1.72)	+ 0.08	-
12	-	-	1.92 (2.01)	+ 0.57	Acoustic Neurinoma
13	-	-	2.22 (1.98)	+ 0.24	Normal
14	2.4 (1.24)	4.08(3.66)	1.68 (1.92)	+ 0.12	-
17	2.01 (1.72)	3.69(3.45)	1.68 (1.72)	+ 0.14	Normal
18	-	-	1.96 (1.87)	+ 0.1	Normal
21	-	-	1.80 (1.92)	+ 0.48	-
22	-	-	-	+ 0.23	Normal
25	2.28 (2.08)	4.08(3.88)	1.80 (1.80)	-0.06	Normal
27	-	-	-	+ 0.2	-
33	-	-	-	- 0.06	Normal
35	1.76 (2.01)	3.78 (4.00)	-	- 0.05	-
36	1.72 (1.84)	3.88 (3.72)	2.16 (1.88)	+ 0.08	-
37	-	-	2.00 (1.92)	- 0.04	-
38	2.2 (2.0)	4.0 (3.92)	1.8 (1.92)	- 0.12	-
40	-	-	-	+ 0.02	-
41	2.00 (2.04)	4.0 (3.96)	2.0 (1.92)	+ 0.04	-
42	-	6.12 (4.16)	-	+ 1.8	Acoustic Neurinoma
43	2.20 (2.04)	4.00 (4.00)	1.80 (1.96)	+ 0.04	-
44		-	2.04 (2.58)	- 0.48	Normal
45	-	-	-	+ 0.09	Normal
46	-	-	2.04 (1.62)	+ 0.18	Normal
49	-	-	-	- 0.04	Normal
50	-	-	2.25(1.96)	+ 0.34	-
52	-	-	2.06 (2.11)	- 0.05	Acoustic Neurinoma
53	-	-	-	+ 0.15	Abnormal but Acoustic Neurinoma was ruled out
total	11 cases (1 FP)	12 cases (1 TP)	21 cases (2 FN, 1 FP)	31 cases (2 TP, 1 FN, 2 FP)	14 cases (8 cases with Normal MRI who had no wave)

*Cut off points: IPL I–III≥2.3 msec, IPL III–V≥2.1 msec, IPL I–V≥4.4 msec, ILD 5≥0.4 msec.
IPL: Interpeak Latency, ILD 5: Interaural absolute Latency Difference wave 5.
AN: Acoustic Neurinoma, FP: False Positive, FN: False Negative, TP: True Positive.

Table 2. 13 SSNHL patients with abnormal or absent ABR and TEOAE, and normal brain MRI.

Case number	Primary SRT	ABR	Overall correlation in TEOAE	Brain MRI	Treatment regimen	Type of response
1	N/A	No wave	+ 8%	Normal	C + A	No Response
7	50 dB	No wave	- 12%	Normal	Urographin	Moderate Response
9	N/A	No wave	- 3%	Normal	Urographin	No Response
24	N/A	No wave	+ 18%	Normal	Urographin	No Response
3	85 dB	Waves I & III absent	- 17%	Normal	C + A	No Response
22	65 dB	Waves I & III absent	- 3%	Normal	C + A	No Response
26	N/A	No wave	0	Normal	Urographin	No Response
28	80 dB	No wave	- 10%	Normal	C + A	Moderate Response
33	60 dB	Waves I & III absent	- 16%	Normal	C + A	No Response
31	65 dB	No wave	- 10%	Normal	C + A	No Response
45	80 dB	Waves I & III absent	- 34%	Normal	C + A	No Response
48	N/A	No wave	- 11%	Normal	Urographin	Moderate Response
49	80 dB	Waves I & III absent	- 21%	Normal	Urographin	No Response

N/A: was not able to test
C + A: Corticosteroid + Acyclovir
SRT: Speech Reception Threshold

Table 3. Three cases with acoustic neurinoma and primary presentation of SSNHL.

Case no	Sex–Age	Initial SRT	ABR	Overall Correlation in TEOAE
12	F-56	50	Wave I absent, IPL (III-V) = 1.92 (normal) ILD 5 = + 0.57 (Abnormal)	86%
42	M-24	35	Wave III absent, IPL (1-V) = 6.12 (Abnormal) ILD 5 = + 1.8 msec (Abnormal)	8%
52	F-53	70	Wave I absent, IPL (III-V) = 2.06 (normal) ILD 5 = - 0.05 (normal)	8%

ILD 5 = Interaural absolute Latency Difference of wave 5.
F: Female, M: Male.

Table 4. Mean overall correlation in TEOAE in different subgroups of treatment modality (oral steroid + acyclovir)

Subgroup	Number of cases	Mean age (years)	Mean initial SRT (dB)	Mean overall correlation (%)*
No response	11	38.63	77.27	- 3.5 (± 1.16)
Partial response	11	35.45	70.45	+ 11.0 (± 1.99)
Complete response	8	37.5	68.75	+ 36.6 (± 3.07)

*P = 0.01 according to ANOVA test.

Discussion

Sudden SNHL is not uncommon and has an overall incidence of 5-20 per 100,000 population per year [1,2,4]. More than 100 etiologies may result in SSNHL; albeit, in most cases it remains idiopathic. Several different pathogenic mechanisms have been proposed for idiopathic SSNHL, including labyrinthine viral infections, autoimmunity, vascular derangements and ischemic events in cochlea and neural pathways, and microtrauma to sensorineural structures [1,2,5,6]. Site of lesion and location of pathology could be at cochlea, auditory nerve and central neural pathway. In various studies performed on temporal bones of patients (or guinea pigs) with SSNHL, abnormalities have been suggested to be in cochlea [7], cochlear nerve cells and ganglia [8], or diffusely in both sensory end organs and ganglia [1].

But, how can we use noninvasive tests such as ABR and OAEs to determine the site of lesion in SSNHL? The ABR is a far-field record

of the synchronized responses of a large number of neurons in the lower portion of the auditory pathway. It consists of 5 positive peaks (waves I to V), and latency of individual peaks; IPL are stable and well documented [9,10]. Wave I is difficult to resolve with hearing loss >40-45 dB at higher frequencies, while wave III persists somewhat beyond that. IPL (or delays) such as IPL I-III or IPL I–V, and interaural absolute wave 5 latency difference (ILD 5) are considered the most sensitive measures of retrocochlear involvements. The basic assumption is that peripheral hearing problems (conductive or sensory) do not affect the neural transmission time as measured by IPLs, but tumors of the 8th cranial nerve do that [9]. There are 30-40% false negative results in ABR for detecting acoustic neurinomas, especially for smaller ones. Therefore it seems necessary to evaluate all SSNHL patients with brain MRI [2,5]. However, MRI cannot substitute ABR for determining software disturbances in neural pathways such as auditory neuropathy or diffuse ischemic events. Many variables including gender and age affect ABR recordings [11]. Even cochlear pathologies which cause hearing loss can affect ABR [9].

OAE is an easy and noninvasive test like ABR used in the evaluation of the hearing loss. There is a link between the presence of OAEs (spontaneous or evoked) and function of the OHCs of cochlea [4,10]. OAEs have proved to be sensitive indicators of change in the cochlea induced by many factors like hypoxia. They are vulnerable in detecting sensorineural hearing loss and idiopathic SSNHL [4,10]. Transient evoked OAEs (TEOAEs) are clinically popular evoked OAEs evoked by clicks and are typically analyzed in the frequency domains. Signal to noise ratios of more than 3-6 dB, coupled with reproducibility (overall correlation) of more than 70% [12] or 50% [13] are the main criteria used to determine weather a response is present within a specific frequency band. TEOAEs are generally not recorded in ears with audiometric thresholds greater than 30–35 dB [10,14]. The finding of significant EOAEs in an ear with audiometric thresholds worse than approximately 40 dB hearing loss suggests that the hearing loss may have a neural origin or may involve structures in cochlea other than the OHCs [10]. It may also show functional etiologies of SSNHL. Unfortunately, the amplitude of EOAEs does not strongly correlate with audiometric sensitively [10]. There are several known variables that affect the presence and the amplitude of OAEs including middle ear and external ear disorders, environmental and internal noise levels, number of averages, and primary tone characteristics [4]. Even retrocochlear diseases such as acoustic neurinomas influence OAEs, probably because of regional cochlear ischemia caused by the anatomic factors related to neural influence [1].

Given the unexplained etiology of SSNHL, various treatment regimens are recommended. Siegel listed 51 different drugs for treatment of SSNHL including vasodilators, diuretics, anticoagulants, plasma expanders, meglumin diatrizoate, carbogen (5% CO_2 and 95% O_2), steroids, rheologic agents, vitamins, and antivirals [1,2,5,6,15]. At present, systemic steroids are the most commonly employed treatment for SSNHL [1,2]. Treatment with oral antiviral drugs in conjunction with oral steroids has also proved to be more efficient [16]. Nonetheless, it is better to understand the pathophysiologic process of illness to choose the best treatment.
Can ABR and OAEs help us in detecting the site of lesion in SSNHL? Can the results of OAE tests influence the outcome of SSNHL?

A. Site of lesion

In a study of five patients with idiopathic SSNHL, Ota Y and Oda M used electrocochleogram (ABR) and TEOAE to determine the site of lesion; the lesions were located in both cochleae in three patients and in the retrocochlear pathway in two. The primary site of lesion had been suggested to be sensory hair cells [17].

Only 28 patients in our study afforded brain MRI. Nearly all other patients had normal physical examination (such as corneal reflex), normal indices in ABR, or responded completely to therapy (although this cannot rule out acoustic neurinomas). Of these 28 patients,

3 had acoustic neurinoma. Interestingly, ABR was abnormal in 2 patients and the overall correlation in TEOAE was reduced in two (table 3). Three patients had abnormal ABR, and normal or nearly normal TEOAE.

Auditory neuropathy was suspected in one of these patients who had normal MRI.

Six patients had normal ABR but abnormal OAE; cochlear pathology was suspected in four patients with normal MRI.

Thirteen patients had no waves in ABR (no waves at all or absent I or III waves) and attenuated or negative overall correlations in TEOAE and normal MRI (table 2). In this group of patients, primary SRT was mostly above 65 dB and this may have affected ABR and OAEs detection, but certain etiologies such as ischemia, which simultaneously affects neural pathway and cochlea, can be postulated. Interestingly, 10 of these patients showed no response to treatment (77%), and 3 only responded partially. None of our treatment modalities (steroids + acyclovir or urographin) had any significant effect on ischemia, and other treatment options such as vasodilators or carbogen might prove to be a better choice of therapy for these patients.

It should be noted that many variables such as primary SRT can affect ABR and OAE results but the mean values of SRT of these groups were not significantly different.

B. Prognostic value of OAEs

It is generally agreed that spontaneous recovery is common in SSNHL (30-65%) [2,5,6]. In various studies, placebos accomplished recovery rates of 14% [18], 25% [19] or 38% [2]. In fact, there is considerable controversy regarding the prognosis of SSNHL, and existing studies have not determined spontaneous recovery rates, the best therapeutic regimen, and the prognostic factors in recovery [5]. Negative prognostic factors include severe hearing loss, downsloping or flat audiograms, presence of vertigo, age (<15 years and >40 or 65 years), time from onset to initial visit, hearing loss in opposite ear, and elevated ESR (>25 mm in the first hour) [2, 5, 6,15]. It is generally agreed that patients with hearing loss >90 dB upon initial presentation have the poorest recovery regardless of therapy [1]. In our study, of 22 patients with nil or negative overall correlation and S/N ratio in pretreatment TEOAE, 12 (54.5%) showed no response to the treatment. Conversely, all of 5 patients with overall correlation >50% in TEOAE, responded completely (3 patients) or partially (2 patients).

Overall, in 3 treatment response groups, no response, partial response and good to complete response, the mean values of overall correlation were –13.15%, +16.76%, and +27.05% respectively. Meanwhile, there were no significant differences in mean initial SRT or age of patients in each subgroup. It may be concluded that positive overall correlation or S/N ratio in TEOAE may be a good independent prognostic factor for response to treatment in SSNHL.

Conclusion

We suggest using TEOAE and ABR to detect the site of lesion in patients with SSNHL; this may help in choosing the best treatment modalities. We also recommend using TEOAE as a new prognostic factor in SSNHL.

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