Journal of the Neurological Sciences 339 (2014) 176–182

Contents lists available at ScienceDirect

Journal of the Neurological Sciences

journal homepage: www.elsevier.com/locate/jns

Long-term prognosis for hearing recovery in stroke patients presenting vertigo and acute hearing loss

Hyun-Ah Kim [a,b,1], Byung-Chan Lee [a,1], Jeong-Ho Hong [a,b], Chang-Ki Yeo [c], Hyon-Ah Yi [a,b], Hyung Lee [a,b,*]

[a] Department of Neurology, Keimyung University School of Medicine, Daegu, Republic of Korea
[b] Brain Research Institute, Keimyung University School of Medicine, Daegu, Republic of Korea
[c] Department of Otorhinolaryngology, Keimyung University School of Medicine, Daegu, Republic of Korea

ARTICLE INFO

Article history:
Received 4 December 2013
Received in revised form 15 January 2014
Accepted 12 February 2014
Available online 19 February 2014

Keywords:
Hearing loss
Long-term outcome
Stroke
Prognostic factor
Vertigo
Vertebrobasilar territory

ABSTRACT

Background and purpose: Vertebrobasilar ischemic stroke (VBIS) can cause acute hearing loss (AHL) because the vertebrobasilar system supplies most of the auditory system including the inner ear. The aim of this study was to assess the long-term prognosis of AHL associated with VBIS.
Methods: Over 12.5 years, 62 patients with AHL of a vascular cause who were followed for at least 1 year (mean, 49.2 months; SD, 24.4 months) were enrolled in this study. Quantitative audiovestibular function testing was performed during the acute (mostly within 10 days after symptom onset) and last follow-up periods in all patients.
Results: On the last follow-up, approximately 65% (39/62) of the patients showed a partial (n = 24) or complete (n = 15) hearing recovery. All but 2 (97%) patients had acute vertigo and 56 (56/62, 90%) had a unilateral canal paresis to caloric stimulation on the side of the AHL. The most commonly infarcted territory on brain MRI was in the distribution of the anterior inferior cerebellar artery (55/62, 89%). Multivariable analysis showed that multiple risk factors for stroke [odds ratio (OR) 10.46, 95% confidence interval (CI) 1.72 to 13.7, p = 0.011] and profound hearing loss [OR 3.92, 95% CI 1.03 to 14.97, p < 0.046] predicted a poor outcome for recovery of hearing loss.
Conclusions: Acute hearing loss associated with posterior circulation ischemic stroke exhibits a relatively good long-term outcome. Two or more risk factors for stroke and profound hearing loss are adverse prognostic factors for recovery of hearing loss of a vascular cause.

1. Introduction

Vertebrobasilar ischemic stroke (VBIS) can cause acute hearing loss (AHL) because the vertebrobasilar system supplies most of the auditory system including the inner ear [1]. Previous studies [2–11] have shown that AHL can occur as a sign of VBIS mainly involving the anterior inferior cerebellar artery (AICA) territory. The symptoms of VBIS can be devastating, and some forms of VBIS are associated with high mortality rates [12]. Recent reports [13,14] have emphasized that AHL may serve as a window to prevent the progression of AHL into more widespread areas of infarction in the posterior circulation.

The detailed spectrum of audiovestibular loss due to VBIS has recently been addressed [11]. However, the outcome of hearing loss as a sign of VBIS has not been well studied. To the best of our knowledge, there have been no prior reports that focus specifically on the factors predicting the long-term outcome of hearing loss in a large sample population of patients with AHL of a vascular cause. Therefore, we sought to assess the long-term outcome of AHL of a vascular cause and to identify the prognostic factors predicting a poor outcome in this type of AHL.

2. Patients and methods

From January 2000 to June 2012, we identified 62 patients with AHL due to VBIS who were followed for at least 1 year from the acute stroke registry at Keimyung University Dongsan Medical Center. We defined AHL of a vascular cause as follows: 1) patients noted a definite change in hearing at the time of VBIS; (2) pure tone audiogram also documented AHL of sensorineural type corresponding to the patient's symptomatic hearing loss; and (3) diffusion-weighted brain MRI showed acute infarct(s) in the posterior circulation. Our definitions of risk factors for stroke were as follows. (1) Hypertension was deemed present when the patient had been undergoing treatment with antihypertensive agents,

* Corresponding author at: Department of Neurology, Keimyung University School of Medicine, 56 Dalseong-ro, Jung-gu, Daegu 700-712, Republic of Korea. Tel.: +82 53 250 7835; fax: +82 53 250 7840.
E-mail address: hlee@dsmc.or.kr (H. Lee).
[1] Two authors equally contributed as co-first authors to this work.

http://dx.doi.org/10.1016/j.jns.2014.02.010
0022-510X/

or their blood pressure was either ≥140 mm Hg systolic or ≥90 mm Hg diastolic on at least two occasions after the acute phase of their ischemic stroke. (2) Diabetes mellitus was deemed present when the patient had been receiving medication for diabetes, had an elevated fasting glucose level ≥126 mg/dL (7.0 mmol/L) or a 2-hour plasma glucose ≥200 mg/dL (11.1 mmol/L) during their oral glucose tolerance test, or had a plasma glucose level ≥200 mg/dL (11.1 mmol/L), along with classic symptoms of hyperglycemia, a hypoglycemic crisis, or a hemoglobin A1c >6.5% [15]. (3) Current smokers were those who regularly smoked at least one cigarette per day at the time of presentation. (4) Previous stroke or TIA was defined as a history of sudden onset of focal neurologic deficits without reference to the type of stroke (i.e., ischemic and hemorrhagic attacks) or duration of symptoms (i.e., stroke or TIA). (5) Dyslipidemia was determined to be present if the patient had been taking lipid-lowering agents or had total cholesterol >240 mg/dL (6.21 mmol/L), TG >200 mg/dL (2.26 mmol/L), or LDL cholesterol >160 mg/dL (4.14 mmol/L).

The average age of the patients was 63.0 years (SD, 11.8; range, 27 to 92 years). Each patient underwent a complete history and neurological examination by the senior author (H.L.). In all patients, initial neurotological evaluations were performed during acute periods after symptom onset (audiometric assessment: 5.4 ± 5.4 days, vestibular assessment: 8.1 ± 6.8 days) and the mean follow-up periods were 49.2 months (±24.4 months, range: 12–110 months). A pure-tone average (PTA) was performed using air- and bone-conducted signals in an acoustic booth. Hearing level was calculated as an average hearing level for three different frequencies ranges [i.e., 3 pure tone average (PTA) with 500, 1000, and 2000 Hz. A PTA > 25 dB was regarded as indicative of hearing loss. The degree of hearing loss was divided into four categories: mild (26–40 dB), moderate (41–70 dB), severe (71–90 dB), and profound (>90 dB). Vertigo was defined as a sensation of self-motion when no self-motion is occurring. Video-oculography (VOG) (SMI, Teltow, Germany, resolution of 0.1°, sampling rate of 60 Hz) was used to record spontaneous and gaze-evoked nystagmus, and caloric responses. Caloric testing was performed with 30 °C-cold and 44 °C-warm irrigations of each ear for 20 s. Asymmetry of vestibular function was calculated using the Jongkees' formula and caloric paresis (CP) was defined by the response difference of 25% or more between the ears. Details regarding the audiovestibular tests including pure tone audiogram, speech discrimination test, and auditory brainstem response have been previously published [3,16,17].

In all of the patients, MRI (including diffusion-weighted images) and magnetic resonance angiography (MRA) of the vertebral and basilar arteries were performed shortly after the onset of symptoms (mean interval: 2.6 days, range: 1–11 days). The vascular territories in the posterior circulation were determined according to the previously validated MR-anatomical templates for the diagnosis of the arterial territories [18]. Infarction within the vertebrobasilar territory, which is responsible for hearing loss, was divided into three categories; 1) isolated AICA territory infarction, 2) AICA plus infarction, and 3) non-AICA territory posterior circulation infarction. The vascular territory was determined by a consensus between the two neurologists (H.L., and H.A.K.).

Hearing outcome measures included both the final PTAs and the difference between the initial and final PTAs. Hearing improvement was calculated as an absolute improvement (difference between the initial and final PTAs). Based on the PTAs on the last follow-up, we divided the patients into three groups; complete improvement, partial improvement, and no improvement according to the modified Siegel's criteria [19]. Complete improvement was defined as recovery in the affected ear to a PTA ≤ 25 dB and partial improvement was defined as an improved PTA of >15 dB in the affected ear without complete improvement. No improvement was defined as anything other than complete or partial improvement (including deterioration). A normal caloric response was defined as a response difference of 25% or less between the ears at the last follow-up, regardless of the extent of improvement from post-stroke baseline.

We compared the clinical and laboratory findings of patients with recovery (partial or complete) of hearing loss and those who showed no hearing recovery using the Student t-test for continuous variables, and Pearson's chi-square and Fisher's exact tests for categorical variables. A multivariable logistic regression analysis was performed to identify the independent contribution of factors for a poor outcome. Covariates whose p values were <0.2 in the comparisons of baseline characteristics were chosen for adjustments. The results were presented as odds ratio (OR) estimates of the relative risk with 95% confidence intervals (CIs). Statistical analyses were performed by using the SPSS program version 19. 0 and significance was established at a value of $p < 0.05$.

This study included all of the consecutive patients who met the above-listed inclusion criteria during the research period. Thus, 29 patients who were previously reported [2–7,9–11,16] were included for completeness of the data, but new information was added in this report.

All experiments followed the tenets of the Declaration of Helsinki and informed consents were obtained after the nature and possible consequences of the study had been explained to the participants. This study was approved by the Institutional Review Board of the School of Medicine at Keimyung University.

3. Results

3.1. Clinical and audiovestibular characteristics

We identified 62 patients (37 men and 25 were women) with AHL due to VBIS according to the above inclusion criteria. All but one were alert and oriented upon admission. The most common risk factors were hypertension (n = 45, 73%) and diabetes (n = 27, 44%). Current smoking, hyperlipidemia, and stroke were found in 24, 22, and 16 patients, respectively. Two or more vascular risk factors were identified in 47 (76%) patients, while no identifiable risk factor was presented in only 4 (6%) patients. Bithermal caloric test showed unilateral CP on the side of AHL in 56 (90%) patients and the remainder (n = 6) showed normal caloric responses. The CP was unilateral in all patients. The extent of hearing impairment at the initial assessment in the group with two or more risk factors for stroke was not significantly different compared to the group with no or a single risk factor (71.3 dB in 47 patients versus 67.6 dB in 15 patients, p = 0.60), but there was a trend of, though not significant, greater hearing improvement at the last follow-up evaluation in the group with no or a single risk factor for stroke (14.4 dB in 47 patients versus 23.9 dB in 15 patients, p = 0.11). In most patients (97%, 60/62), acute spontaneous prolonged vertigo (more than 24 h) was the presenting symptom.

The spontaneous nystagmus was predominantly horizontal and 49 (79%) patients showed spontaneous nystagmus beating away from (88% [43 of 49]) or toward (8% [4 of 49]) the side of the lesion. Asymmetrical bidirectional gaze-evoked nystagmus was found in 27 (44%) patients. Positive head impulse test result, limb dysmetria, ipsilateral facial sensory loss, skew deviation on bed side examination, and ipsilateral facial palsy were also found in, 56 (90%), 40 (65%), 20 (32%), 16 (26%), and 15 (24%) patients, respectively.

Pure tone audiogram detected unilateral (n = 60) or bilateral (n = 2) sensorineural hearing loss during the acute period. Hearing impairment was mild in 7 patients, moderate in 27, severe in 7, and profound in 23 (total numbers of hearing loss were 64 as two patients had bilateral hearing loss subsequently during admission). Among 34 patients with mild or moderate degree of hearing loss, 29 (85%) showed good speech discrimination, and normal auditory brainstem evoked response or delay in absolute latencies of all waves with no abnormalities in interpeak latencies, indicating a cochlear site of injury. Only 5 (15%) had test results indicating a retrocochlear site of lesion, which was characterized by severe impairment of speech discrimination compared to degree of hearing loss and absent auditory evoked response. Others had combined hearing loss of cochlear and retrocochlear type on affected