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The association between vertebrobasilar dolichoectasia and hemifacial spasm

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ABSTRACT

Background: Hemifacial spasm (HFS) is frequently caused by vascular compression of the facial nerve. Vertebrobasilar dolichoectasia (VBDE) may cause vascular crowding in the limited space of the posterior fossa, increasing the chance of vascular compression of the facial nerve. We investigated the prevalence of VBDE in HFS.
Methods: We analyzed the presence of VBDE on 3.0 T magnetic resonance images in patients with HFS and control subjects; age, sex and hypertension were matched. Two blinded readers independently assessed the images. We evaluated the vascular risk factors, including diabetes mellitus, hyperlipidemia, ischemic heart disease, stroke, and presence of lacunes.
Results: A total of 310 patients with HFS and 310 control subjects were included. The prevalence of VBDE was higher in patients with HFS (48/310, 15.5%) than in controls (10/310, 3.2%), with an odds ratio (OR) of 5.82 ($P < 0.001$). Among patients with HFS, the presence of facial nerve compressing vessels was more frequent in dolichoectasia-positive patients (87.5%) than in dolichoectasia-negative patients (58.4%) (OR: 4.99, $P < 0.001$). Dolichoectasia-positive patients had a higher mean age (58.8 versus 54.8 years, $P = 0.03$), as well as greater frequency of hypertension (OR: 2.44, $P = 0.01$) and history of ischemic heart disease (OR: 5.05, $P = 0.03$) than their dolichoectasia-negative counterparts.
Conclusions: We found that VBDE is associated with HFS in a portion of patients. Since vascular risk factors were more prevalent in dolichoectasia-positive patients, an investigation of VBDE and its risk factors may serve to prevent vascular complications.

1. Introduction

Hemifacial spasm (HFS) is a peripherally induced movement disorder characterized by involuntary and unilateral contractions involving the facial muscles [1]. Primary HFS appears to be caused most frequently by vascular compression of the facial nerve—at its exit zone from the brainstem [2]. Secondary HFS seems to be caused from peripheral facial palsy, demyelinating disorders, trauma, and tumor compression. Facial nerve compression is thought to lead to ephaptic transmission and/or hyperactivity of the facial nucleus, resulting in HFS [3]. As for vascular compression, the branch vessels that originate from the vertebral and basilar arteries have been reported to cause HFS [4], and there was an attempt to explain the influence of anatomical variation of vertebrobasilar arteries on the occurrence of HFS [5,6]. Several studies have shown a higher prevalence of hypertension in patients with HFS than in control subjects [7–12]. It has previously been asserted that hypertension may predispose to ectatic vessels and contribute to facial nerve compression [13]. An association between HFS and a smaller area of the posterior cranial fossa has been reported in Asian populations, suggesting that the chance of facial nerve

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compression increases as the posterior fossa space becomes more crowded [14,15].

Dolichoectasia, defined as an increase in the length and/or diameter of the vessels, mainly affects the vertebral and basilar arteries [16]. Vertebrobasilar dolichoectasia (VBDE) may induce facial nerve compression via increased tortuosity and angulation in the branch vessels of vertebrobasilar arteries, or via direct compression of the nerve by the dolichoectatic portion itself. However, such cases of direct compression of the facial nerve by VBDE have sporadically been reported (references in eSupp file), and VBDE can be observed even in healthy subjects [16]. In a series of HFS patients who underwent microvascular decompression surgery, only 0.7% showed direct compression of the facial nerve by VBDE [17]. In this study, we investigated the overall prevalence of VBDE in HFS, regardless of treatment modalities, and compared HFS patients with age-, sex-, and hypertension-matched control subjects.

2. Subjects and methods

2.1. Subjects

Between January 2007 and May 2015, patients who were diagnosed with primary HFS were consecutively recruited in our movement disorders unit at Seoul National University Bundang Hospital, which is a tertiary referral-based hospital. Secondary HFS cases were excluded. All patients underwent brain magnetic resonance imaging (MRI) at our hospital. Among the 320 HFS patients who visited our hospital during the study period, 10 patients—8 with peripheral facial palsy and 2 with facial nerve schwannoma—were excluded from this study. For comparison, 310 age-, sex-, and hypertension-matched subjects with no signs of neurological disorders were randomly selected from 9423 health-check examinees, who underwent brain MRI as a part of health-check examination at our hospital during the same period. Thus, 310 HFS patients and 310 control subjects were included for the final analysis. Clinical information regarding hypertension, diabetes mellitus, hyperlipidemia, history of ischemic heart disease, and/or stroke was obtained from medical records. Hypertension was diagnosed using the criteria and methodology set by the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (a consistent average of two or more blood pressure measurements on two or more visits, systolic blood pressure $\geq$140 mmHg and/or diastolic blood pressure $\geq$90 mmHg), history of treated hypertension, or current antihypertensive therapy [18]. Diabetes mellitus was diagnosed using the standard criteria (hemoglobin A1c $\geq$6.5%, fasting plasma glucose $\geq$126 mg/dL, or postprandial 2-h plasma glucose $\geq$200 mg/dL), history of treated diabetes mellitus, or current anti-diabetic medications. Hyperlipidemia was defined as elevated plasma cholesterol $\geq$240 mg/dL, history of treated hyperlipidemia, or current lipid-lowering therapy. Prior history of ischemic heart disease and stroke was obtained. The study protocol was approved by the institutional review board at Seoul National University Bundang Hospital (August 26, 2015, protocol number: B-1508/312-105). Informed consent requirements were waived by the board due to retrospective nature of this study.

2.2. Imaging protocol

MRI was performed at 3.0 T (Achieva and Ingenia, Philips Healthcare, Best, the Netherland) using a 32-channel sensitivity encoding (SENSE) head coil. Patients underwent axial T_2-weighted imaging (T_2-WI), axial fluid-attenuated inversion recovery (FLAIR) imaging, 3D T_2-weighted volume isotropic turbo spin echo imaging (3D T_2-VISTA), and time-of-flight (TOF) MR angiography (MRA). The parameters for MRI were as follows: T_2-WI, repetition time (TR) 3000 ms, echo time (TE) 80 ms, field-of-view (FOV) 190 mm $\times$ 240 mm, acquisition matrix 400 mm $\times$ 320 mm, slice thickness 5 mm; FLAIR, TR 11000 ms, TE 125 ms, inversion time 2.5 s, FOV 190 mm $\times$ 240 mm, acquisition matrix 370 mm $\times$ 260 mm, slice thickness 5 mm; 3D T_2-VISTA, TR 2000 m, TE 290 m, SENSE factor 2, FOV 160 mm $\times$ 160 mm, acquisition matrix 270 mm $\times$ 270 mm, slice thickness, 0.6 mm; TOF-MRA, TR 25 m, TE 3.5 m, flip angle 20°, FOV 180 mm $\times$ 200 mm, acquisition matrix 700 mm $\times$ 360 mm, slice thickness, 1.2 mm, slab thickness, 70 mm.

2.3. Criteria of VBDE

Two readers, who were blinded to the clinical information, independently assessed the presence of VBDE. VBDE was defined, by applying the previously adopted criteria, as the presence of either dolichosis (elongation) or ectasia (dilatation) in the vertebrobasilar arteries [19–21]. Dolichosis was defined as an abnormal location of vertebrobasilar junction or as an abnormal elongation of arteries (Fig. 1A). Vertebrobasilar junction located above the suprasellar cistern or lateral to the margin of the clivus or dorsum sellae was considered abnormal. As for the elongation of basilar artery, a deviation of >10 mm from the reference line (a straight line joining the basilar artery origin to its bifurcation) was considered abnormal. For vertebral arteries, a deviation of >10 mm from the reference line (a straight line joining its intracranial entry point to the basilar artery origin) was considered abnormal. Ectasia was determined to be positive if the maximum diameter of the vertebrobasilar arteries was larger than 4.5 mm in any location along the course (Fig. 1B). Images were loaded into the database and presented to the readers in a blinded manner. After making two independent readings, a final consensus was made with regard to the presence of VBDE. After coming to a decision, the vessel responsible for facial nerve compression was identified. If a vascular structure was in close contact with the facial nerve at the root exit zone and/or caused indentation or deviation of the facial nerve, then such a structure was regarded as a compressing vessel. Lacunes were considered to be positive if there was a loss of focal brain tissue surrounded by hyperintensity in FLAIR images.

2.4. Statistical analysis

We used the Kolmogorov-Smirnov test to assess the normality of distribution of variables. If the variables reached a significance level ($P > 0.05$), parametric statistics were used. Clinical findings were compared using Student's *t*-test and chi-squared test for parametric data, and Mann-Whitney *U* test and Fisher's exact test for non-parametric data. Binary logistic regression was used to estimate the odds ratios (OR) adjusted for age, sex and hypertension. The inter-observer agreement was tested using Cohen's κ statistics. *P* values of less than 0.05 were considered statistically significant. Statistical analyses were performed using SPSS software (version 20.0; SPSS, Chicago, IL, USA).

3. Results

3.1. Clinical characteristics of the subjects

A total of 310 HFS patients and 310 age-, sex-, and hypertension-matched control subjects were included in this study (Table 1). Among the 310 patients with HFS, there were 2.3 times more women than men; 175 showed left HFS and 135 showed right HFS. One case of coexisting HFS and ocular neuromyotonia was observed

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