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반측 안면 연축 환자에서의 척추기저동맥 신연증과의 연관성에 관한 연구

The association between vertebrobasilar dolichoectasia and hemifacial spasm

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Abstract

The association between vertebrobasilar dolichoectasia and hemifacial spasm

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Objective: Hemifacial spasm (HFS) is frequently caused by vascular compression of the facial nerve. Dolichoectasia of vertebrobasilar arteries (VBDE) may cause vascular crowding in the limited space of the posterior fossa, increasing the chance of vascular contact to the facial nerve. We investigated the prevalence of VBDE in HFS.

Methods: We analyzed the presence of VBDE on MRI 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, history of ischemic heart disease, and/or stroke, and the presence of lacunes on brain MRI.

Results: A total of 620 subjects–310 HFS patients and 310 control subjects–were included. The prevalence of VBDE was higher in HFS patients (48/310, 15.5%) than in controls (10/310, 3.2%), with an odds ratio of 5.82 (95% confidence interval: 2.86–11.85, $p < 0.001$). The presence of facial nerve contacting vessels was more frequent in VBDE-positive HFS patients (81.3%) than VBDE-negative patients (54.2%), with an odds ratio of 3.48 (95% confidence interval: 1.60–7.57, $p = 0.002$). Among HFS patients, VBDE-positive patients showed to exhibit a higher mean age, as well as greater frequency of hypertension and history of ischemic heart disease than their VBDE-negative counterparts.

Conclusions: We found that VBDE is associated with HFS in a portion of HFS patients. Since vascular risk factors were more frequently observed in VBDE-positive patients, an investigation of VBDE and its risk factors in patients with HFS may be served to prevent vascular complications.

Keywords: Hemifacial spasm, Vertebrobasilar dolichoectasia.

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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, and secondary HFS may arise from peripheral facial palsy, demyelinating disorders, trauma, and tumor compression.[2,3] Facial nerve compression is thought to lead to ephaptic transmission and hyperactivity of the facial nucleus, resulting in HFS.[4] As for vascular compression, the branch vessels that originate from the vertebral and basilar arteries have been reported to cause HFS,[5] and there was an attempt to explain the influence of anatomical variation of vertebrobasilar arteries on the occurrence of HFS.[6] Dolichoectasia, defined as an increase in the diameter and/or length of the vessels, mainly affects the vertebral and basilar arteries.[7,8] Vertebrobasilar dolichoectasia (VBDE) may induce facial nerve compression via increased tortuosity and angulation in the branch vessels or direct contact with the nerve. However, such cases have been rarely reported, and VBDE can be observed even in healthy subjects.[8] In a series of HFS patients who underwent microvascular decompression surgery, only 0.7% showed direct compression of the facial nerve by VBDE.[9] In this study, we investigated the overall prevalence of VBDE in HFS, regardless of treatment modalities. Since VBDE is associated with vascular risk factors and higher prevalence in patients with stroke (12~17%) than in the general population (0.06~5.8%),[10-13] we compared HFS patients with age-, sex-, and hypertension-matched control subjects.

Methods

Standard protocol approvals, registrations, and patient consents.

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. For comparison, age-, sex-, and hypertension-matched healthy subjects with no signs of neurological disorders were included during the same period. All subjects underwent brain MRI at our hospital. Clinical information regarding diabetes mellitus, hyperlipidemia, history of ischemic heart disease, and/or stroke were obtained. Hypertension was defined as a history of treated hypertension or the presence of antihypertensive therapy. Diabetes mellitus was defined as documented abnormality of fasting and postprandial blood glucose or the presence of anti-diabetic medications. Hyperlipidemia was defined as elevated plasma cholesterol more than 240mg/dL or a history of taking lipid-lowering agent. History of ischemic heart disease and stroke was regarded as positive if there were documented records. The study protocol was approved by the institutional review board at our institution. Informed consent requirements were waived by the board.

Imaging protocol and analysis.

MRI was performed at 3 Tesla (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 msec, echo time (TE) 80 msec, field-of-view (FOV) 190 mm × 240 mm, acquisition matrix 400 mm × 320 mm, slice thickness 5 mm; FLAIR, TR 11000 msec, TE 125 msec, inversion time 2.5 sec, FOV 190 mm × 240 mm, acquisition matrix 370 mm × 260 mm, slice thickness 5 mm; 3D T_2-VISTA, TR 2000 msec, TE 290 msec, SENSE factor 2, FOV 160 mm × 160 mm, acquisition matrix 270 mm × 270 mm, slice thickness, 0.6 mm; TOF-MRA, TR 25 msec, TE 3.5 msec, flip angle 20 º, FOV 180 mm × 200 mm, acquisition matrix 700 mm × 360 mm, slice thickness, 1.2 mm, slab thickness, 70mm.

Two readers, who were blinded to the clinical information, independently assessed the presence of VBDE and lacunes. Lacunes were regarded as positive if there was a loss of the focal brain tissue surrounded by hyperintensity in FLAIR images. VBDE was defined as the presence of either dolichosis (elongation) or ectasia (dilatation) in the vertebrobasilar arteries.[14-16] Dolichosis was defined as an abnormal location of vertebrobasilar junction or as an abnormal elongation of arteries: the location of vertebrobasilar junction above the suprasellar cistern or lateral to the margin of clivus or dorsum sellae was considered as abnormal (figure 1A). 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 as 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 as abnormal. Ectasia was determined to be positive if the maximum diameter of the vertebrobasilar arteries is larger than 4.5 mm in any location along the course (figure 1B). Images were loaded into a database and presented in a blinded fashion for the readers. After making two independent readings, the two readers made a final consensus with regard to presence of VBDE. After coming to a decision, the vessel in contact with the facial nerve was identified. When there was a vascular structure that made contact with the facial nerve at the root entry zone, such vascular structure was regarded as an offending vessel.

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 and multiple logistic regression analyses were used for the estimation of 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 as statistically significant. Statistical analyses were performed using SPSS software (version 20.0; SPSS, Chicago, IL, USA).

Results

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. A resulting total of 310 HFS patients and 310 age-, sex-, and hypertension-matched control subjects were included for the final analysis (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. For treatment, 84 patients underwent microvascular decompression surgery, 70 received regular botulinum toxin injections, and remaining 156 were followed-up without treatment. Between HFS patients and control subjects, there was no significant difference in the vascular risk factors.

Out of the 620 subjects, 58 were found to have VBDE, including 48 HFS patients and 10 control subjects (Table 2). With regard to the evaluation of VBDE presence, the inter-observer agreement was 98.4% ($\kappa = 0.91$, $p < 0.001$). Between subjects with VBDE and those without VBDE, the maximum deviation from the reference line and maximum arterial diameter was larger, and the presence of facial nerve contacting vessels was more frequent in subjects with VBDE than those without VBDE.

The prevalence of VBDE in HFS patients (48/310, 15.5%) was higher than that of the control subjects (10/310, 3.2%), with an adjusted OR of 5.82 (Table 3). Dolichosis and dolichosis combined with ectasia was more frequent in HFS patients than in controls, but ectasia did not show significant difference. The maximum deviation from the reference line was larger in HFS patients than in control subjects, but the maximum arterial diameter did not differ. The presence of facial nerve contacting vessels was more frequent in HFS patients than in control subjects. Among HFS patients, the maximum deviation from the reference line and maximum arterial diameter was larger, and the facial nerve contacting vessels were more frequent in VBDE-positive patients than those without VBDE. Out of the 39 identified VBDE-positive HFS patients with facial nerve contacting vessels, 7 showed direct contact to the facial nerve by the dolichoectatic portion of the vertebral artery, and 32 showed contact by vessels branching from the vertebrobasilar artery (figure 1C and 1D). Among control subjects, the maximum deviation from the reference line was larger, and the facial nerve contacting vessels were more frequent in VBDE-positive controls than those without VBDE. However, the maximum arterial diameter did not differ. Among the 48 VBDE-positive HFS patients, 39 had VBDE on the same side as HFS (left: 28; right: 11), while 9 had VBDE on the opposite side from HFS (left HFS and right VBDE: 4; right HFS and left VBDE: 5). Among these 9 patients, the facial nerve contacting vessels were identified in 4 patients, who had the offending vessels on the same side as HFS. The left or right side of lateral deviation of VBDE were significantly related to the symptomatic side of HFS ($\chi^2 = 15.71$, $p < 0.001$, relative risk = 15.40). With regard to the treatment modalities, the prevalence of VBDE was 20.2% (17/84) in patients with microvascular decompression surgery, 15.7% (11/70) in patients with regular botulinum toxin injections, and 12.8% (20/156) in patients without treatment; there was no significant difference between the treatment modalities ($p = 0.14$).

To examine the association between VBDE and vascular risk factors, a comparative analysis was performed between subjects with VBDE and subjects without VBDE in each group (Table 4). The mean age and frequency of hypertension were both higher in subjects with VBDE than those without VBDE. Among HFS patients, VBDE-positive patients showed higher mean age, as well as greater frequency of hypertension and history of ischemic heart disease than VBDE-negative counterparts. Among control subjects, the hyperlipidemia and history of stroke were more frequent in VBDE-positive controls than those without VBDE.

Discussion

In this study, we found that the prevalence of VBDE was higher in patients with HFS (48/310, 15.5%) than the controls (10/310, 3.2%), with an OR of 5.82. And dolichosis showed more significant difference than ectasia, between HFS patients and controls. The maximum deviation of vertebrobasilar artery from the reference line was larger in HFS patients than control subjects, suggesting a contribution from dolichosis. The presence of facial nerve contacting vessels was more frequent in VBDE-positive HFS patients than VBDE-negative HFS patients. These results suggest an association between VBDE and HFS.

Higher prevalence of VBDE in HFS patients can be explained by the pathophysiology of HFS. The facial nerve compression is thought to lead to ephaptic transmission and hyperactivity of the facial nucleus, resulting in HFS; the main cause of facial nerve compression is mechanical irritation by adjacent vessels.[4] In the presence of VBDE, vertebrobasilar artery and its branching arteries could occupy relatively greater space in a limited area of the posterior fossa, thus increasing the chance of contact to the facial nerve. An association between HFS and a smaller area of the posterior fossa has been reported in Asian populations.[17,18] HFS was much more common in Asians, as this population was thought to have smaller posterior fossa compared with other populations.[17] The posterior fossa CSF volume was lower in HFS patients from Singapore compared with controls matched for age, sex, race, and hypertension.[18] These reports suggest that the chance of HFS occurrence increases as the posterior fossa area becomes more crowded. In our study, the prevalence of VBDE was higher in HFS patients than in control subjects, suggesting that VBDE could induce a crowded posterior fossa area via increased tortuosity and angulation in the vessels, resulting in vascular compression of the facial nerve. In addition, the direction of lateral deviation of the vertebral artery has been reported to be significantly related to the symptomatic side of HFS in a Chinese population: the likelihood that the vertebral artery deviated to the symptomatic side of HFS was 86.4%, while the likelihood of deviation to the asymptomatic side was 10.2%.[6] In our study, the direction of lateral deviation of VBDE were significantly related to the symptomatic side of HFS, suggesting that VBDE could induce a crowded posterior fossa space. Further studies in other populations are necessary to firmly establish the association between VBDE and HFS.

Pathogenesis of VBDE is thought to be associated with rarefaction of the elastic tissue in the tunica media with fragmentation of the internal elastic lamina.[19,20] The main causes of such process are vascular risk factors, like old age, male sex, and hypertension.[8,11] In our study, the prevalence of diabetes mellitus, hyperlipidemia, history of ischemic heart disease, and/or stroke, as well as lacunes on MRI did not differ between HFS patients and age-, sex-, and hypertension-matched control subjects. Between subjects with VBDE and those without VBDE, the mean age was higher and hypertension was more frequent in VBDE-positive subjects than those without VBDE, which is consistent with previous reports showing an association of VBDE with old age and hypertension.[8,11] Among HFS patients, higher age and greater frequency of hypertension and history of ischemic heart disease were observed in VBDE-positive patients compared with VBDE-negative patients, which is in accordance with the proposed pathogenesis of VBDE. Furthermore, the prevalence of VBDE in patients with HFS (15.5%) was similar to those of previous reports from patients with stroke (12~17%).[10,11] With regard to sex distribution, women were more common than men among the 310 patients with HFS, which is consistent with previous reports showing higher prevalence of HFS in women.[21,22] In VBDE-positive subjects, the proportion of men tended to be higher than VBDE-negative subjects, but without statistical significance. The vascular risk factors are worthy of investigation for VBDE-positive HFS patients, and measures to prevent vascular complications could be beneficial.

In our study, a small portion of HFS patients (15.5%) showed VBDE. In

HFS cases without VBDE, a smaller posterior fossa may be a contributing factor. A lateral deviation of the vertebrobasilar artery, which was not as prominent to be included in the criteria of VBDE, could also induce a crowded posterior fossa space. In our study, 3.2% of control subjects exhibited VBDE, and this prevalence of VBDE in asymptomatic controls is consistent with those of previous reports from the general populations (0.06~5.8%).[12,13] Among control subjects, 11.9% were identified as having the vessels in contact with the facial nerve, similar to a previous report showing innocent contacts to the facial nerve by blood vessels in 15% of asymptomatic side of HFS.[23]

Our study has some limitations. First, as this study was conducted by a cross sectional evaluation of MRI findings, long-term prognosis in accordance to the presence of VBDE was not sought. Second, as a small number of 10 control subjects were identified to have VBDE, the results from the VBDE-positive controls should be interpreted with caution. Future long-term longitudinal studies with larger samples of patients in various ethnic populations are needed.

In summary, we found that VBDE is associated with HFS in a portion of HFS patients. Since vascular risk factors were more frequently observed in subjects with VBDE than those without VBDE, patients with HFS may benefit from an investigation of the existence of VBDE and its risk factors to prevent the development of vascular complications.

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Table 1. Clinical finding of subjects.

Group - no. (%)	Hemifacial spasm (N = 310)	Control subjects (N = 310)	p Value
Age at diagnosis and imaging (years)	55.4±11.8	55.3±11.9	0.95
Onset age (years)	48.6±12.1	N/A	N/A
Sex (M:F)	93:217 (30.0%:70.0%)	93:217 (30.0%:70.0%)	1.00
Hypertension	89 (28.7%)	89 (28.7%)	1.00
Diabetes mellitus	15 (4.8%)	22 (7.1%)	0.31
Hyperlipidemia	48 (15.5%)	47 (15.2%)	1.00
History of ischemic heart disease	9 (2.9%)	12 (3.8%)	0.66
History of stroke	8 (2.6%)	6 (1.9%)	0.79
Lacunes on MRI	17 (5.5%)	21 (6.8%)	0.62

Abbreviations: N/A = not applicable.

Plus–minus values are means ± standard deviation.

Table 2. Characteristics of vertebrobasilar dolichoectasia.

Group (no.)	Subjects with VBDE (N =58)				Subjects without VBDE (N = 562)	p Value, OR (95% CI)
	Dolichosis only (37) [a]	Ectasia only (5)	Dolichosis and ectasia (16) [a]	Total		
Deviation (mm) [range]	10.4±2.3 [5.6–16.8]	5.2±3.6 [2.0–8.7]	8.9±2.9 [3.5–16.5]	9.6±2.9 [2.0–16.8]	4.8±2.0 [1.5–9.7]	p< 0.001
Diameter (mm) [range]	3.3±0.5 [2.2–4.1]	5.2±0.3 [5.0–5.7]	5.3±0.8 [4.5–7.2]	4.0±1.1 [2.2–7.2]	3.1±0.5 [1.5–4.4]	p< 0.001
Presence of contacting vessels no. (%)	43/58 (74.1%)				175/562 (31.1%)	6.57 (3.41–12.27), p< 0.001
Contacting vessels (no.)	AICA (19), PICA (11), VA (13), BA (0)				AICA (108), PICA (56), VA (10), BA (1)	

Abbreviations: VBDE = vertebrobasilar dolichoectasia; OR = odds ratio; CI = confidence interval; Deviation = Maximum deviation from the reference line; Diameter = Maximum arterial diameter; AICA= anterior inferior cerebellar artery; PICA= posterior inferior cerebellar artery; VA = vertebral artery; BA= basilar artery.

Results of maximum deviation from the reference line and maximum arterial diameter were obtained from a final consensus reading.

[a] Sixteen subjects (9 with dolichosis only, 7 with dolichosis and ectasia) were

judged to have dolichosis according to one of the criteria of VBDE, the vertebrobasilar junction located lateral to the clivus, although their maximum deviation from the reference line was less than 10 mm.

Table 3. Vertebrobasilar dolichoectasia in patients with hemifacial spasm and control subjects.

<table>
<tr><td>Group</td><td colspan="3">Hemifacial spasm (N = 310)</td><td colspan="2">Control subjects (N = 310)</td><td>p Value, OR (95% CI)</td></tr>
<tr><td>VBDE
no. (%)</td><td colspan="3">48/310 (15.5%)
Dolichosis only 30
Ectasia only 4
Dolichosis and ectasia 14</td><td colspan="2">10/310 (3.2%)
Dolichosis only 7
Ectasia only 1
Dolichosis and ectasia 2</td><td>5.82 (2.86–11.85), p< 0.001
4.91 (2.12–11.36), p< 0.001
4.58 (0.51–41.24), p= 0.175
8.02 (1.80–35.59), p= 0.006</td></tr>
<tr><td>Deviation (mm) [range]</td><td colspan="3">5.8±2.7 [1.6–16.8]</td><td colspan="2">4.6±2.0 [1.5–13.6]</td><td>p< 0.001</td></tr>
<tr><td>Diameter (mm) [range]</td><td colspan="3">3.2±0.8 [1.5–7.2]</td><td colspan="2">3.2±0.5 [1.6–6.0]</td><td>p= 0.60</td></tr>
<tr><td>Presence of contacting vessels no. (%)</td><td colspan="3">181/310 (58.4%)</td><td colspan="2">37/310 (11.9%)</td><td>10.35 (6.86–15.62), p< 0.001</td></tr>
<tr><td>Contacting vessels (no.)</td><td colspan="3">AICA (110), PICA (53), VA (17), BA (1)</td><td colspan="2">AICA (17), PICA (14), VA (6), BA (0)</td><td></td></tr>
<tr><td>Subgroup (no.)</td><td>HFS with VBDE [a] (48)</td><td>HFS without VBDE (262)</td><td>p Value, OR (95% CI)</td><td>Controls with VBDE [a] (10)</td><td>Controls without VBDE (300)</td><td>p Value, OR (95% CI)</td></tr>
<tr><td>Deviation (mm) [range]</td><td>9.7±2.7 [2.4–16.8]</td><td>5.1±2.1 [1.6–9.7]</td><td>p< 0.001</td><td>9.1±3.7 [2.0–13.6]</td><td>4.5±1.7 [1.5–9.4]</td><td>p= 0.003</td></tr>
<tr><td>Diameter (mm) [range]</td><td>4.1±1.1 [2.2–</td><td>3.1±0.6 [1.5–</td><td>p< 0.001</td><td>3.8±1.2 [2.3-6.0]</td><td>3.2±0.5 [1.6-4.4]</td><td>p= 0.10</td></tr>
</table>

	7.2]	4.4]				
Presence of contacting vessels no. (%)	39/48 (81.3%)	142/262 (54.2%)	3.48 (1.60–7.57), p= 0.002	4/10 (40%)	33/300 (11%)	6.39 (1.64–24.90), p= 0.007
Contacting vessels (no.)	AICA (19), PICA (10), VA (10) [b], BA (0)	AICA (91), PICA (43), VA (7), BA (1)		AICA (0), PICA (1), VA (3), BA (0)	AICA (17), PICA (13), VA (3), BA (0)	

Abbreviations: OR = odds ratio; CI = confidence interval; Deviation = Maximum deviation from the reference line; Diameter = Maximum arterial diameter; AICA= anterior inferior cerebellar artery; PICA= posterior inferior cerebellar artery; VA = vertebral artery; BA= basilar artery; HFS = hemifacial spasm; VBDE = vertebrobasilar dolichoectasia.

[a] Not significantly different for the maximum deviation from the reference line (p = 0.58) and the maximum arterial diameter (p = 0.59) between VBDE-positive HFS patients and VBDE-positive controls. Presence of contacting vessels was more frequent in the VBDE-positive HFS patients than VBDE-positive controls, with an OR of 7.69 (95% CI: 1.67– 35.32, p = 0.009).

[b] Seven out of 10 patients showed a direct contact to the facial nerve by the

dolichoectatic portion of vertebral artery.

Table 4. Characteristics of vascular risk factors.

Group (no. %)	Subjects with VBDE (58)	Subjects without VBDE (562)	p Value, OR (95% CI)	Hemifacial spasm (310)			Control subjects (310)		
				HFS with VBDE (48)	HFS without VBDE (262)	p Value, OR (95% CI)	Controls with VBDE (10)	Controls without VBDE (300)	p Value, OR (95% CI)
Age (years)	59.1±10.6	55.0±11.9	p= 0.01	58.8±11.0	54.8±11.8	p= 0.03	60.5±8.7	55.2±12.0	p= 0.16
Onset age	N/A	N/A	N/A	51.4±12.2	48.1±12.1	p= 0.09	N/A	N/A	N/A
Sex (M:F)	24:34	162:400	1.74 (1.00–3.03), p= 0.051	20:28	73:189	1.85 (0.98–3.48), p= 0.06	4:6	89:211	0.63 (0.44–5.75), p= 0.73
Hypertension	29 (50.0%)	149 (26.5%)	2.16 (1.17–3.99), p= 0.01	24 (50.0%)	65 (24.8%)	2.44 (1.20–4.95), p= 0.01	5 (50.0%)	84 (28.0%)	1.79 (0.45–7.13), p= 0.41
Diabetes mellitus	3 (5.2%)	34 (6.0%)	0.35 (0.16–1.92), p= 0.55	2 (4.2%)	13 (5.0%)	0.49 (0.10–2.40), p= 0.38	1 (10.0%)	21 (7.0%)	1.07 (0.12–9.33), p= 0.95
Hyperlipidemia	12 (20.7%)	83 (14.8%)	1.21 (0.60–2.44), p= 0.55	7 (14.6%) [a]	41 (15.6%)	0.70 (0.28–1.73), p= 0.44	5 (50.0%) [a]	42 (14.0%)	5.20 (1.40–19.38), p= 0.01

History of ischemic heart disease	5 (8.6%)	16 (2.8%)	2.12 (0.69–6.50), p= 0.19	5 (10.4%)	4 (1.5%)	5.05 (1.21–21.02), p= 0.03	0 (0%)	12 (4.0%)	N/A, p= 1.00 [b]
History of stroke	4 (6.9%)	10 (1.8%)	3.03 (0.89–10.36), p= 0.08	2 (4.2%)	6 (2.3%)	1.45 (0.27–7.84), p= 0.67	2 (20.0%)	4 (1.3%)	10.89 (1.32–90.17), p= 0.03
Lacunes on MRI	8 (13.8%)	30(5.3%)	2.03 (0.80–5.13), p= 0.14	6 (12.5%)	11 (4.2%)	2.36 (0.70–7.98), p= 0.17	2 (20.0%)	19 (6.3%)	2.95 (0.52–16.92), p= 0.22

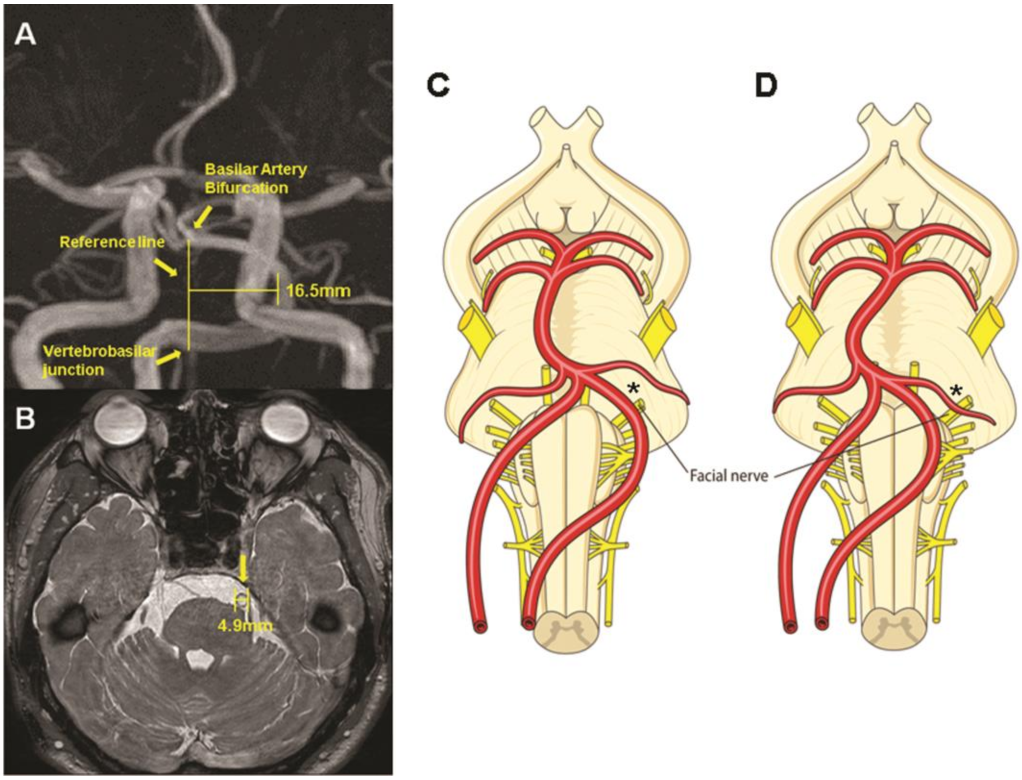
Abbreviations: HFS = hemifacial spasm; VBDE = vertebrobasilar dolichoectasia; OR = odds ratio; CI = confidence interval; N/A = not applicable.

Odds ratios of vascular risk factors, adjusted for age and sex, were obtained using binary logistic regression.

[a] Significantly different for hyperlipidemia between VBDE-positive patients and controls with an OR of 6.04 (95% CI: 1.31–27.90, p= 0.02).

[b] There was no case with positive history of ischemic heart disease in the controls with VBDE; Fisher's exact test was used.

Figure 1. Vertebrobasilar dolichoectasia in hemifacial spasm.



Legends for figure

(A) In a 51-year-old man with hemifacial spasm, vertebrobasilar junction was observed lateral to the margin of clivus, and a deviation of basilar artery from the reference line, joining the basilar artery origin to its bifurcation, was estimated to be 16.5 mm, showing dolichosis. (B) The maximum diameter of basilar artery was 4.9 mm, showing ectasia. (C, D) A schematic representation of vertebrobasilar dolichoectasia. Compression of the facial nerve by a direct contact with vertebrobasilar dolichoectasia (C), and by vessels branching from vertebrobasilar artery (D) (asterisks).

국문 초록

반측 안면 연축 환자에서의 척추기저동맥 신연증과의 연관성에 관한 연구

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목적: 반측 안면 연축은 주로 안면 신경이 뇌혈관의 압박을 받아 발생한다. 한편, 척추기저동맥 신연증은 뒤쪽 우묵 공간의 과밀화를 초래하여, 뇌혈관과 안면 신경이 접촉할 확률을 높일 수 있다. 본 연구에서는 척추기저동맥 신연증과, 반측 안면 연축의 연관성을 밝히는 것을 목적으로 한다.

방법: 반측 안면 연축으로 내원한 환자군과, 나이, 성별, 고혈압 유무를 맞춘 대조군을 후향적으로 설정하였다. 환자군과 대조군은 모두 MRI 검사를 받았다. 환자군 혹은 대조군 여부를 모르는 두 명의 연구자가 MRI를 확인하여 척추기저동맥 신연증 유무 및 혈관과 안면 신경의 접촉 여부, 그리고 열공성 경색 유무를 확인하였다.

척추기저동맥 신연증과 혈관 위험 인자의 연관성을 확인하기 위하여, 의무기록 참조를 통해, 환자군과 대조군 모두에서 고혈압, 당뇨, 고지혈증, 뇌졸중 및 허혈성 심장 질환 병력의 유무를 확인하였다.

결과: 총 620명(반측 안면 연축 환자군 310명, 대조군 310명)의 연구 대상이 포함되었다. 척추기저동맥 신연증의 유병률은 반측 안면 연축 환자군(48/310, 15.5%)에서 대조군(10/310, 3.2%)보다 높았으며, 승산비는 5.82였다 (95% 신뢰구간: 2.86-11.85, $p < 0.001$). 반측 안면 연축 환자에서 뇌혈관과 안면 신경의 접촉이 확인된 경우는 척추기저동맥 신연증이 있을 경우(81.3%), 척추기저동맥 신연증이 없을 경우(54.2%)보다 높았으며, 승산비는 3.48이었다(95% 신뢰구간: 1.60-7.57, $p = 0.002$). 반측 안면 연축 환자에서 척추기저동맥 신연증이 있을 경우, 척추기저동맥 신연증이 없는 경우에 비해 평균 나이, 고혈압 및 허혈성 심장 질환의 병력이 더 많았다.

결론: 척추기저동맥 신연증은 반측 안면 연축 발생의 일부를 설명할 수 있다. 또한, 혈관 위험 인자가 척추기저동맥 신연증과 연관되어 있으므로, 반측 안면 연축 환자에서 척추기저동맥 신연증 유무를 확인하는 것은, 향후 혈관 합병증을 예방하는 데 도움이 될

수 있다.

주요어: 반측 안면 연축, 척추기저동맥 신연증

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