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Evaluation of 40 hemifacial spasm patients with MR angiography of the brain and vertebral artery dominance in the etiology of hemifacial spasm

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Abstract

Objectives: Hemifacial spasm is characterised by involuntary, rhythmic, tonic or clonic contractions originating from the facial nerve and occurring intermittently in unilateral facial muscles. The most commonly reported cause of hemifacial spasm is the compression of the facial nerve at its root exit/entry zone by an ectatic or aberrant blood vessel, leading to local demyelination. In this study, we compared magnetic resonance angiography (MRA) findings from patients with hemifacial spasm and controls to determine whether vertebral artery dominance is part of the aetiology of this condition.
Methods: This retrospective case-control study was conducted with data from 40 patients with hemifacial spasm who received botulinum toxin injections and for whom MRA findings were accessible, and 42 patients of similar age and sex who presented with symptoms of nonspecific headache in the outpatient neurology clinic. Magnetic resonance imaging and unenhanced time-of-flight MRA examinations from the study and control groups were evaluated. *Results:* A dominant vertebral artery was present in 52.2% of patients in the hemifacial spasm group and 33.3% of those in the control group, but this difference was not significant ($p = 0.079$).
Conclusions: The lack of significant results supporting our hypothesis may be attributable to the small study population. Further studies with larger groups are warranted.

Keywords: Hemifacial spasm, vertebral artery dominance, magnetic resonance angiography, root exit zone, vascular compression

INTRODUCTION

Hemifacial spasm (HFS) is a disease characterised by involuntary, rhythmic, tonic or clonic contractions originating from the facial nerve and occurring intermittently in unilateral facial muscles.[1] Paroxysmal contractions generally start from the orbicularis oculi muscle and spread to the perinasal and perioral muscles, and even the platysma muscle over time.[2,3]

The root exit/entry zone of a cranial nerve is the junction between the central and peripheral nerve segments. In this area, a transition occurs from cells responsible for cranial nerve myelination (i.e. central oligodendroglial cells) to peripheral Schwann cells. In addition, the cranial nerves in this zone lack an epineurium, and are protected only by an arachnoid membrane. This segment of the nerve is thus highly susceptible to injury.[4,5]

The most commonly reported cause of HFS is the compression of the facial nerve at this root exit/entry zone by an ectatic or aberrant blood vessel, leading to local demyelination.[5,6] Although several theories have been proposed, various aspects of the aetiology of HFS are unknown. Control group studies have not demonstrated clearly which vessels are affected or whether vertebral artery (VA) dominance is related to vascular anomalies.

In this study, we compared magnetic resonance angiography (MRA) findings from patients with HFS and controls to determine whether VA dominance is involved in the aetiology of this condition.

METHODS

This retrospective case-control study was conducted with data from 40 patients with HFS who received botulinum toxin injections and for whom MRA results were accessible, and 42 patients of similar age and sex who presented with the symptoms of nonspecific headache in the

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outpatient neurology clinic. Those with normal neurological examination results were included in the control group.

Patients presenting with comorbid dystonia or neurological disease, and those with secondary HFS (due to a history of facial paralysis, demyelinating disease, intracranial space–occupying lesion, trauma or surgery) were excluded from the study. A radiologist blinded to patient status evaluated magnetic resonance imaging (MRI) and unenhanced time-of-flight MRA examinations from the study and control groups. All images were obtained with a Philips Ingenia 1.5-T system (Philips Healthcare, Eindhoven, The Netherlands) with SENSE XL Torso coil.

Various techniques have been used to determine VA dominance.[7-11] In this study, following Akgün *et al*.[7], we considered a difference in the diameter of the distal VA > 1 mm to reflect VA dominance and a difference ≤ 1 mm to reflect codominance (Figure 1).

Statistical analysis

The study data were analysed using the SPSS software (version 25.0 for Windows; IBM Corp., Armonk, NY, USA). Descriptive statistics were calculated using frequencies and percentages. The chi-squared test was used to determine the relationship between the two categorical variables.

RESULTS

In total, 40 (20 females, 20 males) and 42 (26 females, 16 males) patients were included in the HFS and control groups, respectively. No patient in either group had diabetes mellitus. Four patients in the study group and three in the control group had hypertension treated with oral medication. Age and sex did not differ between the groups. VA dominance was more common in the HFS group than in the control group, but the difference was not significant. The groups' demographic characteristics and MRA results are compared in Table 1.

The frequencies of anterior cerebral artery hypoplasia and left posterior communicant artery (L Pcom) differed significantly between the HFS and control groups. However, these differences were not clinically relevant. In patients with VA dominance, MRA findings did not differ according to the right- or left-sided presentation of HFS (Table 2).

When the patients with HFS were analyzed separately, no relation was identified between the sides with vertebral artery dominance and HFS. Moreover, there was no significant relation between the HFS side and other vascular structures identified in MRA.

DISCUSSION

HFS is characterised by involuntary contractions of unilateral muscles with facial nerve innervation. Although most HFS cases are considered to be idiopathic, neuropathological and electrophysiological studies of the facial nerve support compression as an aetiological cause. In recent studies, the structural causes of this compression have been investigated neuroradiologically.[12]

Although the anterior inferior cerebellar artery (AICA) and posterior inferior cerebellar artery (PICA) are considered to be involved in the aetiology of HFS, the vertebral and basilar arteries also play roles in the pathogenesis of this condition. HFS caused by the vertebrobasilar arteries is reported to be more common in elderly

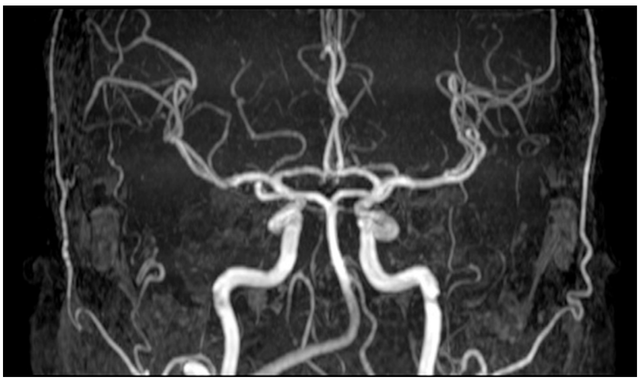

Figure 1: Right vertebral artery dominance: The differrence in the diameter of right and left vertebral arteries is more than 1 mm

Table 1: Comparison of demographic and other features between the HFS and control groups

	HFS Group (n=40)	Control Group (n=42)	P
Age (Mean+SD)	60.22±9.72	56.90±11.84	0.170
Sex	**Number (%)**	**Number (%)**	
Female	20 (50.0)	26 (61.9)	0.278
Male	20 (50.0)	16 (38.1)	
Dominant VA			0.079
Present	21 (52.5)	14 (33.3)	
Absent	19 (47,5)	28 (66.7)	
Fetal PCA			
Present	6 (15.0)	5 (11.9)	0.681
Absent	34 (85.0)	37 (88.1)	
ACA Hipoplasia			**0.029**
Present	14 (35.0)	6 (14.3)	
Absent	26 (65.0)	36 (85.7)	
AComA			
Present	30 (75.0)	36 (85.7)	0.221
Absent	10 (25.0)	6 (14.3)	
RPComA			
Present	13 (32.5)	12 (28.6)	0.699
Absent	27 (67.5)	30 (71.4)	
LPComA			
Present	13 (32.5)	5 (11.9)	**0.024**
Absent	27 (67.5)	37 (88.1)	

Verdom:Vertebral artery dominance, PCA: posterior cerebral artery, ACA: Anterior cerebral artery, Acom A: Anterior communicant artery, R Pcom A: Right posterior communicant artery, L Pcom A: Left posterior communicant artery

subjects and men, and presents more often on the left side. The vertebrobasilar system is considered to be more prone to atherosclerotic changes due to hypertension, diabetes and hyperlipidaemia. As the majority of the blood flow is provided by the left VA, haemodynamic changes affect primarily the left side.[13]

The vertebral or basilar artery was found to be the cause of HFS in 87 of 1,798 patients who underwent decompressive surgery because of HFS in a single centre. The researchers found that HFS due to the VA was more common in men and on the left side.[13]

Mathur *et al*.[14] and Şanal *et al*.[2] each reported a patient in whom left VA dolichoectasia caused a left HFS. Wang *et al*.[15] compared brain MRI and MRA findings from 341 patients with HFS who underwent microvascular decompression with those from 360 control patients. VA dominance was more common in the HFS group than in the control group ($p = 0.026$). The left and right forms of VA dominance were significantly more frequent in those with left and right HFS, respectively ($p < 0.001$). The rate of VA dominance in patients with HFS related to the VA was 94.3%.[15]

Park *et al*.[16] evaluated and compared MRI and MRA results from 120 patients who underwent microvascular decompression and a group of healthy controls, and found that VA dominance was more frequent in the group that underwent surgery than in the control group ($p = 0.03$). They detected no difference in laterality between the study and control groups ($p = 0.855$); however, the right and left forms of VA dominance were

Table 2: Comparison of MRA findings in terms of right and left HFS side in patients with vertebral artery dominance

	With HFS (n=21)		P
Dominant VA	**Right**	**Left**	
Right	1 (10.0)	1 (9.1)	0.945*
Left	9 (90.0)	10 (90.9)	
Fetal PCA			
Right	0 (0.0)	1 (9.1)	
Left	2 (20.0)	0 (0.0)	0.935*
Absent	8 (80.0)	10 (90.9)	
ACA Hypoplasia			
Righthypoplastic A1 segment	2 (20.0)	2 (18.2)	
Lefthypoplastic A1 segment	6 (60.0)	1 (9.1)	0.111*
Normal	2 (20.0)	8 (72.7)	
AComA			
Present	10 (55.6)	8 (44.4)	0.082*
Absent	0 (0.0)	3 (27.3)	
RPComA			
Present	2 (20.0)	4 (36.4)	0.418*
Absent	8 (80.0)	7 (63.6)	
LPComA			
Present	3 (30.0)	3 (27.3)	0.893*
Absent	7 (70.0)	8 (72.7)	

*Fisher's Exact Test
Verdom:Vertebral artery dominance, PCA: posterior cerebral artery, ACA: Anterior cerebral artery, Acom A: Anterior communicant artery, R Pcom A: Right posterior communicant artery, L Pcom A: Left posterior communicant artery

significantly more common in patients with right and left HFS, respectively, than in the control group.[16]

Similar to previous reports, the mean age of patients with HFS was 60.22 years, and VA dominance was prevalent on the left (n = 19) than on the right (n = 2) side, in this study. The larger number of patients with left than with right HFS is similar to the results reported by Kim *et al*.[13], but the numbers of male and female patients were the same. Wang *et al*.[15] identified more VA dominance in an HFS group than in a control group; our results were similar. They also identified significantly more left and right VA dominance in patients with left and right HFS, respectively; we found no such relationship in the present study, possibly due to the inclusion of insufficient numbers of patients overall and in subgroups.

Leong *et al*.[17] analysed data from 62 patients who underwent microvascular decompression to determine the relationship between hypertension and HFS, and found that hypertension was more prevalent in patients with than in those without HFS.

Peren *et al*.[18] performed colour doppler ultrasonography on nine patients with HFS and a control group, and found that the AICA and PICA flow rates were significantly higher in patients with HFS. They observed no difference in the vertebral and basilar arteries. They related these elevated flow rates to the AICA and PICA dolichoectasia rates (90% and 76%, respectively).[18]

Many researchers have studied the relationship between VA dominance and HFS, and obtained differing results. In a larger study population, Wang *et al*.[10] found that left and right HFS were significantly more common in patients with left and right VA dominance, respectively ($p < 0.001$). The pathophysiology underlying the relationship between VA dominance and HFS involves the elevated arterial blood flow rate on the side of the dominant VA; such haemodynamic changes cause compression in the facial nerve root exit zone. However, many other studies suggest that HFS is related to arterial hypertension.[18]

In conclusion, irregular haemodynamic force resulting from asymmetric VA flow may cause the veins in the vertebrobasilar system to expand, which could affect their tortuosity, and morphological deformations in the vascular structure may lead to compression in the root exit zone of the facial nerve. In this study, we investigated VA dominance in patients with HFS who received botulinum toxin injections. We did not obtain significant results supporting our hypothesis, possibly due to the relatively small size of the study population. Further studies with larger groups are warranted to assess whether a relationship between VA dominance and HFS exists and, if so, to determine its pathophysiology.

DISCLOSURE

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Conflicts of interest/: None

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