 doi: 10.2176/jns-nmc.2023-0125

NMC Case Report Journal 10, 253-257, 2023

Hemifacial Spasm Caused by Vascular Compression of the Anterior Inferior Cerebellar Artery-Posterior Inferior Cerebellar Artery Common Trunk Anomaly at the Cisternal Portion of the Facial Nerve: A Case Report

Mariya HOKAZONO,[1] Takafumi SHIMOGAWA,[1] Akira NAKAMIZO,[1] and Koji YOSHIMOTO[1]

[1]*Department of Neurosurgery, Graduate School of Medical Sciences, Kyushu University, Fukuoka city, Fukuoka, Japan*

Abstract

W report the first case of hemifacial spasm (HFS) caused by vascular compression of the anterior inferior cerebellar artery (AICA)-posterior inferior cerebellar artery (PICA) common trunk anomaly at the cisternal portion of cranial nerve VII (CN VII). A 71-year-old female with a typical right HFS was admitted to our hospital. As per her magnetic resonance (MR) imaging results, no offending arteries were noted around the CN VII root exit zone (REZ). Computed tomography angiography revealed an AICA-PICA common trunk anomaly with a dominant PICA, with the rostral branch of the AICA-PICA common trunk anomaly compressing the CN VII at the cisternal portion. The patient underwent microvascular decompression (MVD), and the HFS disappeared after surgery. The amplitude of the abnormal muscle responses (AMR) disappeared immediately after complete transposition of the offending artery. However, the patient experienced mild transient facial palsy 3 days after MVD which was eventually resolved with the administration of vitamin B12. No HFS recurrence was observed during the 1-year follow-up period. The AICA-PICA common trunk anomaly has been found to cause HFS as it compressed the CN VII at the cisternal portion, and not at the REZ. AMR monitoring might be helpful for cases where the unusual vessel particularly compresses the CN VII.

Keywords: hemifacial spasm, AICA-PICA, cisternal portion of the facial nerve, abnormal muscle response, delayed facial palsy

Introduction

Hemifacial spasm (HFS) has been commonly attributed to blood vessels compressing the cranial nerve VII (CN VII) at the root exit zone (REZ).[1)] However, HFS caused by vascular compression in the distal, cisternal portions of the CN VII has also been reported in a few cases.[2-13)] Microvascular decompression (MVD) has been noted to be a promising treatment for neurovascular compression syndromes including HFS and trigeminal neuralgia.[5-7,11-20)] Common offending vessels in HFS are the anterior inferior cerebellar artery (AICA), posterior inferior cerebellar artery (PICA), and vertebral artery. Only two cases of HFS associated with an AICA-PICA common trunk anomaly with a dominant PICA have been reported to date, and the vessels compressed the REZ in both cases.[21,22)]. Here, we report the first case of HFS caused by vascular compression of the AICA-PICA common trunk anomaly with a dominant PICA at the cisternal portion of CN VII. Ethics approval was obtained from the Institutional Review Board of the Kyushu University Hospital (no. 23003-00). Written informed consent was obtained from the patient for the publication of this case report and any accompanying images.

Case Report

A 71-year-old female with an 8-year history of right HFS was admitted to our hospital. The spasm was initially confined to her right superior orbicularis oculi muscle and

Received May 29, 2023; Accepted July 24, 2023

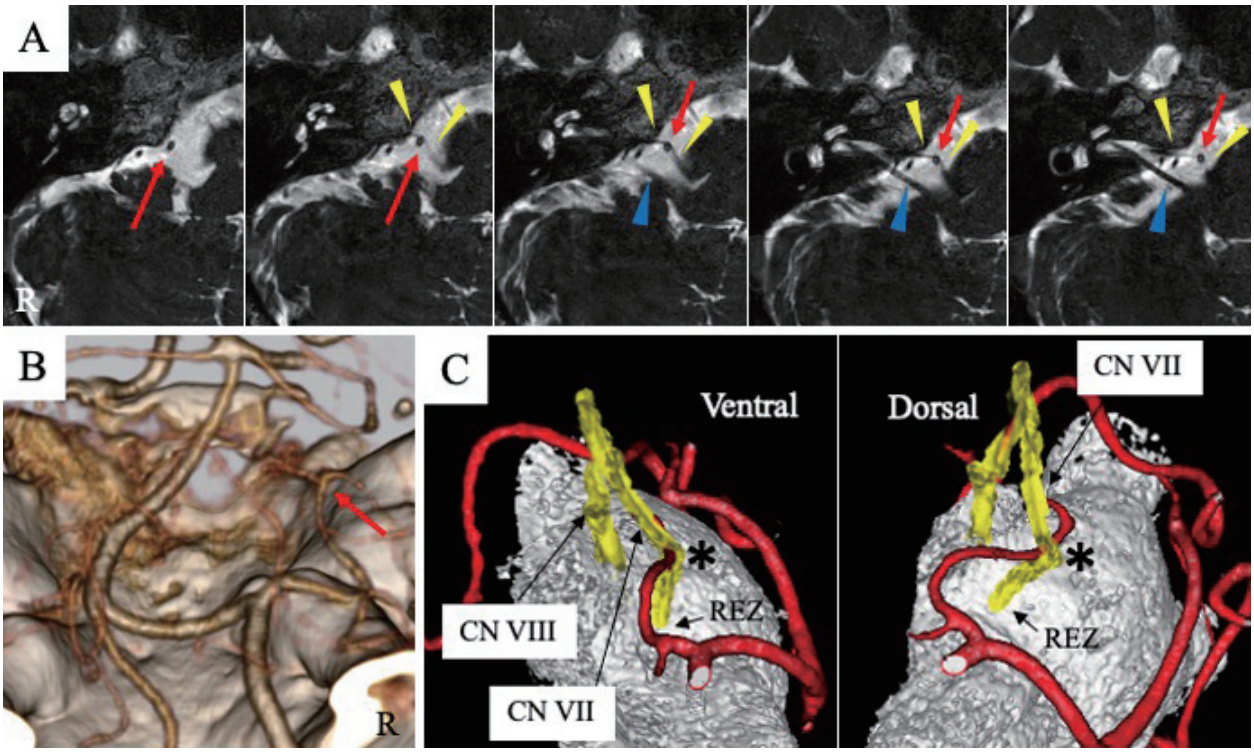

Fig. 1 Preoperative Images.

A) Preoperative magnetic resonance imaging with 3D heavily T2-weighted image (3D-hT2WI) reveals that there is no offending artery around the cranial nerve VII (CN VII) (yellow arrowheads) at the root exit zone (REZ). The cisternal segment of CN VII is bent by the offending artery (red arrow). CN VII arises >5 mm away from the cranial nerve VIII (CN VIII) (blue arrowhead) in the brainstem.

B) Computed tomography angiography shows the anterior inferior cerebellar artery (AICA) -the posterior inferior cerebellar artery (PICA) common trunk with a dominant PICA, and this artery is suspected to be the offending artery.

C) The preoperative schema of vascular compression of the rostral branch of AICA-PICA common trunk anomaly with a dominant PICA at cisternal portion of CN VII (asterisk).

gradually affected the entire right side. The platysma was not involved. Medical treatment with carbamazepine was deemed ineffective. She was also treated with repeated botulinum toxin injections before admission; however, the effect was only temporary. Her physical and neurological examinations, including hearing tests, were normal. No tinnitus or discernible noise was observed in the right ear. Typical clonic hemifacial spasms were evident.

Preoperative magnetic resonance (MR) imaging revealed no offending artery around the REZ of CN VII (Fig. 1A). CN VII arose >5 mm from the REZ of cranial nerve VIII (CN VIII) in the brainstem. Both nerves traveled apart toward the internal acoustic meatus in the cerebellopontine cistern. Computed tomography angiography then revealed an AICA-PICA common trunk anomaly with a dominant PICA (Fig. 1B). The rostral branch of the AICA-PICA common trunk anomaly compressed the CN VII at the cisternal portion (Fig. 1A, C). This artery passes between CV VII and VIII and was suspected to be the offending artery.

The patient then underwent MVD for the right CN VII via the lateral suboccipital approach under general anesthesia. Auditory brainstem evoked responses were monitored throughout the surgery. The abnormal muscle response (AMR), which can be evoked by stimulating one facial nerve branch in the muscles innervated by other branches of the facial nerve and is specific to patients with HFS, was also monitored. The entire courses of CN VII, CN VIII, and the arteries running near the nerves were exposed (Fig. 2A, F). CN VII arose >5 mm from the REZ of CN VIII in the brainstem. As expected, there was no offending vessel around the REZ of CN VII (Fig. 2B, C, D, F). The AICA-PICA common trunk anomaly ran through CN VII and CN VIII, and the rostral branch of the AICA-PICA common trunk anomaly compressed CN VII at the cisternal portion; as a result, CNVII was bent (Fig. 2B, C, D, F). The perforators from the AICA-PICA common trunk anomaly were running to the brainstem, not to the CN VII. The offending artery was then carefully detached from the CN VII and transposed by fixing the petrous bone dura mater with Teflon felt and fibrin glue (Fig. 2E, F). The transposition of the offending artery was difficult due to a perforator. When the compression of the offending artery

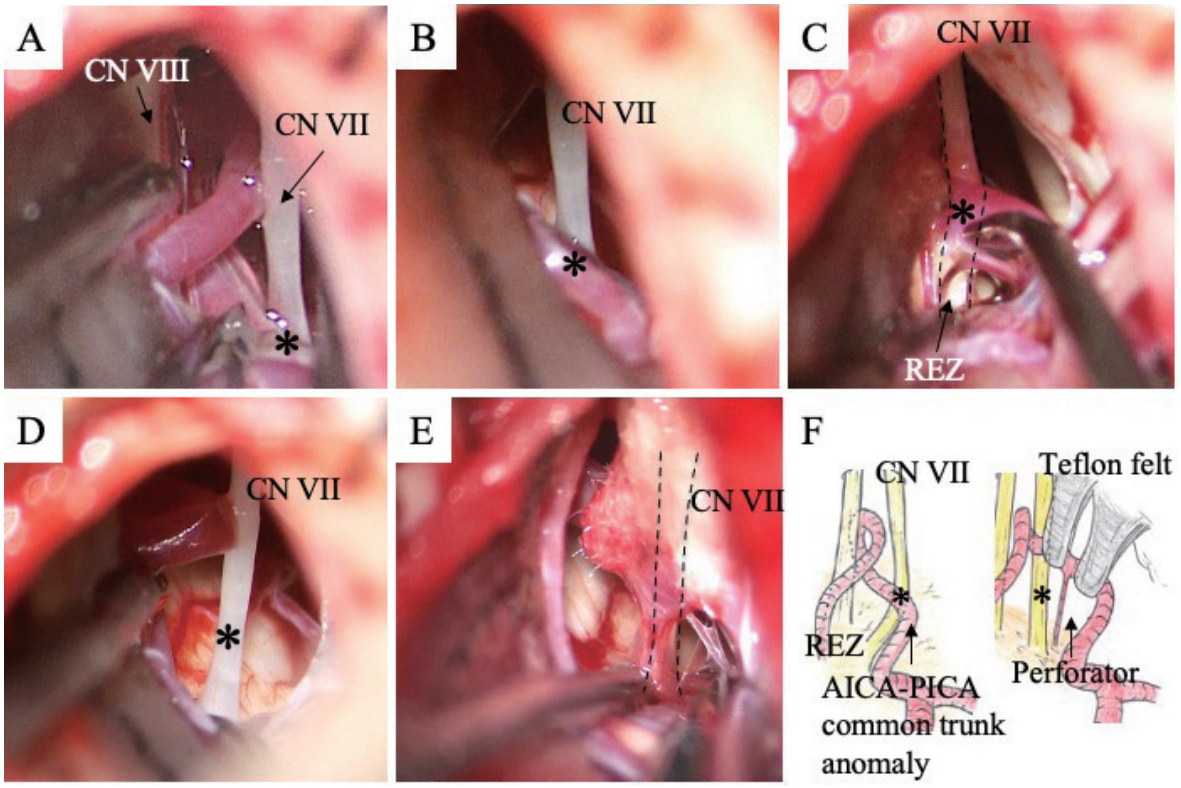

Fig. 2 Intraoperative photograph of microvascular decompression (MVD).
A) The rostral branch of AICA-PICA common trunk anomaly runs near CN VII and CN VIII.
B) C) D) The rostral branch of AICA-PICA common trunk compresses CN VII at the cisternal portion (asterisk). There is no offending vessel in the CN VII at the REZ. The cisternal segment of CN VII is bent by the offending artery (black dotted lines).
E) The offending artery is carefully detached from CN VII (dotted lines) and then transposed and fixed to the nearby dura of the petrous bone with Teflon felt and fibrin glue.
F) The intraoperative schema of vascular compression of the rostral branch of AICA-PICA common trunk anomaly with a dominant PICA at the cisternal portion of CN VII (asterisk) (left) and vascular decompression (right).

on CN VII was partially released, the AMR amplitude decreased (Fig. 3A). Finally, the AMR disappeared after complete transposition of the offending artery from the CN VII (Fig. 3B).

The HFS was completely resolved immediately after surgery. However, the patient developed mild House-Brackmann grade 2 facial nerve palsy 3 days after MVD, and was administered vitamin B12. Her facial nerve palsy completely resolved 6 weeks later. No HFS recurrence was observed during the 1-year follow-up period.

Discussion

CN VII consists of a short central nervous system (CNS) segment with a central oligodendrocyte-derived myelination length at the proximal end and long peripheral nervous system (PNS) segment with peripheral Schwann cell-derived myelination at the distal end.[7,23-25] Depending on the type of myelin sheath, tolerance to external forces may differ, with the CNS segment myelin sheath being considered more vulnerable than the PNS.[26] The CNS segment is replaced by the PNS segment at the REZ, which is located within from 0.8 to 2.5 mm of the site where CN VII emerges from the brainstem.[24,25,27] Due to the structural difference between the CNS and PNS segments, numerous researchers have supported the hypothesis that the cause of HFS is vascular compression, not at the PNS segment but at the CNS segment of CN VII.[26,28,29] However, HFS caused by vascular compression at the cisternal portions of the CN VII has also been reported in a few cases.[2-13] Some researchers have reported in their case series that the rate of HFS caused by vascular compression at the cisternal portions of the CN VII is 0.7-2.1%.[5,7,30] The underlying mechanism remains to be unclear; however, as with proximal compression, structural changes, such as partial demyelination and axonal degeneration of the PNS segment of CN VII, and physiological changes may be involved in the pathogenesis of this entity.[5,7,31] In contrast, Nomura et al.[32] reported that the length of the CNS segment of CN VII was greater than that previously reported in pathological studies.[24,25,27] These reports suggest that vascular compression of the CNS segment at the cisternal portion of the CN

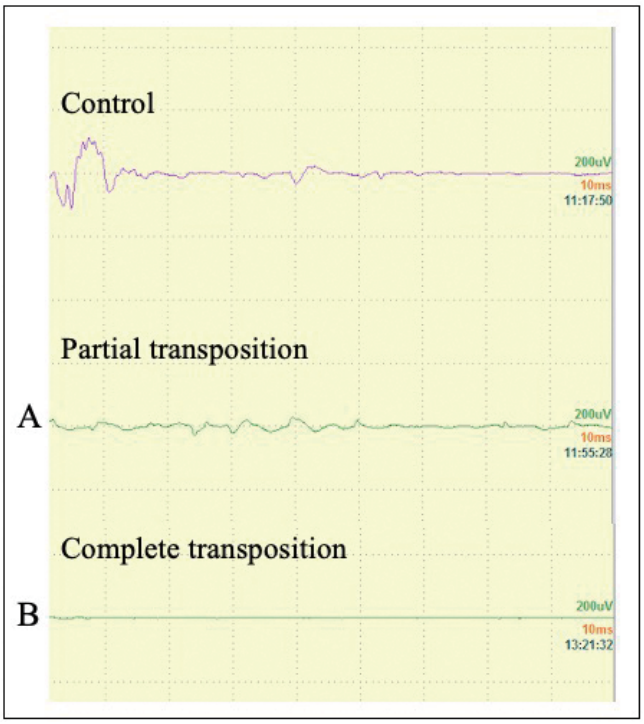

Fig. 3 Abnormal muscle responses (AMRs).
A) When the AICA is partly transposed from CN VII, the amplitude of the AMRs was decreased.
B) The AMRs disappear completely after complete transposition of the offending artery.

VII may also cause HFS. In this case, the distance from the brainstem to the vascular compression point was 5 mm.

Previous reports have demonstrated that the clinical outcomes of patients with HFS and distal compression may be worse than those of patients with proximal compression.[7,13] Chang et al.[7] reported that excellent surgical results (complete resolution of spasm) were achieved in 87.7% (1773/2022) of patients in the proximal compression group, 86.1% (87/101) in the mixed compression group, and 71.4% (10/14) in the cisternal compression group from a total of 2127 cases. In our case, although the compression point was at the cisternal portion of CN VII, the facial spasms disappeared immediately after surgery.

In cases where the compression site was at the cisternal portion of CN VII, the offending artery was the AICA in 84.3% and the PICA in 10.9% of patients.[13] In this present case, the offending vessel was determined to be the rostral branch of the AICA-PICA common trunk anomaly with a dominant PICA. The AICA-PICA common trunk anomaly has been identified as one of the most common vessel variants in the posterior circulation;[33,34] however, this anomaly rarely causes HFS.[21,22,34] In a study examining 159 HFS cases, the AICA-PICA common trunk anomaly with a dominant AICA or a dominant PICA compressed the REZ of the CN VII in 14 and 2 cases, respectively.[22] No case has been previously reported in which the AICA-PICA common trunk anomaly with a dominant PICA caused HFS by compressing the cisternal portion of the CN VII.

The disappearance of AMR during MVD apparently guarantees a high probability of relief from HFS; however, its role in HFS caused by vascular compression at the cisternal portion of CN VII has rarely been demonstrated.[6,10,35,36] In the present case, when the offending artery was partially transposed from CN VII, the amplitude of the AMR decreased and disappeared completely after complete transposition of the artery. We demonstrated that AMR was helpful in treating HFS caused by vascular compression, even at the cisternal portion of CN VII.

In our case, the patient experienced transient facial nerve palsy 3 days after MVD. Postoperative transient facial nerve palsy is commonly reported in 2.8-8.3% of patients after MVD;[34] however, there are no reports of postoperative delayed transient facial nerve palsy associated with vascular compression at the cisternal portion of the CN VII. While many factors may have contributed to postoperative facial nerve palsy, direct manipulation of the nerves and impairment of blood supply should be taken into consideration.[34]

Conclusions

We report the first case of HFS caused by vascular compression of the AICA-PICA common trunk anomaly with a dominant PICA at the cisternal portion of CN VII. AMR monitoring may be helpful for HFS caused by vascular compression at the cisternal portions of CN VII. Postoperative delayed transient facial nerve palsy can occur even in patients with HFS caused by vascular compression at the cisternal portion of the CN VII.

Acknowledgments

We would like to thank Editage (www.editage.com) for the English language editing.

Funding

This study was supported by a Grant-in-Aid for Scientific Research from the Japan Society for the Promotion of Science (JSPS) (JP21K17456 to T. S.).

Conflicts of Interest Disclosure

The authors declare no conflict of interest.

References

1) Jannetta PJ: Microsurgical exploration and decompression of the facial nerve in hemifacial spasm. *Curr Top Surg Res* 2: 217-220, 1970
2) Yeh HS, Tew JM Jr, Ramirez RM: Microsurgical treatment of in-

tractable hemifacial spasm. *Neurosurgery* 9: 383-386, 1981
3) Nagahiro S, Takada A, Matsukado Y, Ushio Y: Microvascular decompression for hemifacial spasm. Patterns of vascular compression in unsuccessfully operated patients. *J Neurosurg* 75: 388-392, 1991
4) Fukuda M, Kameyama S, Honda Y, Tanaka R: Hemifacial spasm resulting from facial nerve compression near the internal acoustic meatus - case report. *Neurol Med Chir (Tokyo)* 37: 771-774, 1997
5) Ryu H, Yamamoto S, Sugiyama K, Uemura K, Miyamoto T: Hemifacial spasm caused by vascular compression of the distal portion of the facial nerve: report of seven cases. *J Neurosurg* 88: 605-609, 1998
6) Kawashima M, Yamada M, Sato S, Oka H, Fujii K, Matsushima T: Hemifacial spasm caused by vascular compression of the distal portion of the facial nerve associated with configuration variation of the facial and vestibulocochlear nerve complex. *Turk Neurosurg* 19: 269-275, 2009
7) Chang WS, Kim HY, Chung SS, Chang JW: Microneurovascular decompression in patients with hemifacial spasm caused by vascular compression of facial nerve at cisternal portion. *Acta Neurochir (Wien)* 152: 2105-2111, 2010
8) Zhong J, Li ST, Zhu J, Guan HX: Is entire nerve root decompression necessary for hemifacial spasm? *Int J Surg* 9: 254-257, 2011
9) Li S, Hong W, Tang Y, et al.: Re-operation for persistent hemifacial spasm after microvascular decompression with the aid of intraoperative monitoring of abnormal muscle response. *Acta Neurochir (Wien)* 152: 2113-2118, 2010
10) Zheng X, Feng B, Zhang W, Ying T, Li S: Hemifacial spasm caused by cross type vascular compression. *Neurol Res* 33: 965-969, 2011
11) Park YS, Chang JH, Cho J, Park YG, Chung SS, Chang JW: Reoperation for persistent or recurrent hemifacial spasm after microvascular decompression. *Neurosurgery* 58: 1162-1167; discussion 1162, 2006
12) Hatayama T, Kono T, Harada Y, et al.: Indications and timings of reoperation for residual or recurrent hemifacial spasm after microvascular decompression: personal experience and literature review. *Neurol Med Chir (Tokyo)* 55: 663-668, 2015
13) Son BC, Ko HC, Choi JG: Hemifacial spasm caused by vascular compression in the cisternal portion of the facial nerve: report of two cases with review of the literature. *Case Rep Neurol Med* 2019: 8526157, 2019
14) Barker FG 2nd, Jannetta PJ, Bissonette DJ, Shields PT, Larkins MV, Jho HD: Microvascular decompression for hemifacial spasm. *J Neurosurg* 82: 201-210, 1995
15) Choi HJ, Choi SK, Rhee BA: Microvascular decompression for hemifacial spasm due to four offending vessels: a case report. *Turk Neurosurg* 23: 241-244, 2013
16) Jannetta PJ: Arterial compression of the trigeminal nerve at the pons in patients with trigeminal neuralgia. *J Neurosurg* 26: Suppl: 159-Suppl:162, 1967
17) Jannetta PJ: Microvascular management of trigeminal neuralgia. *Arch Neurol* 42: 800, 1985
18) Kyoshima K, Watanabe A, Toba Y, Nitta J, Muraoka S, Kobayashi S: Anchoring method for hemifacial spasm associated with vertebral artery: Technical note. *Neurosurgery* 45: 1487-1491, 1999
19) Chung SS, Chang JH, Choi JY, Chang JW, Park YG: Microvascular decompression for hemifacial spasm: a long-term follow-up of 1169 consecutive cases. *Stereotact Funct Neurosurg* 77: 190-193, 2001
20) Yuan Y, Wang Y, Zhang S, Zhang LR, Guo J: Microvascular decompression in patients with hemifacial spasm: report of 1200 cases. *Chin Med J (Engl)* 2118: 833-836, 2005
21) Carlos R, Fukui M, Hasuo K, et al.: Radiological analysis of hemifacial spasm with special reference to angiographic manifestations. *Neuroradiology* 28: 288-295, 1986
22) Shimano H, Kondo A, Yasuda S, et al.: Significance of anomalous anterior inferior cerebellar artery-posterior inferior cerebellar artery common trunk compression in microvascular decompression for hemifacial spasm. *World Neurosurg* 92: 15-22, 2016
23) Lang J: Uber Bau, Länge und Gefässbeziehungen der "zentralen" und "peripheren" Strecken der intrazisternalen Hirnnerven [Anatomy, length and blood vessel relations of "central" and "peripheral" paths of intracisternal cranial nerves]. *Zentralbl Neurochir* 43: 217-258, 1982
24) Skinner HA: Some histologic features of the cranial nerves. *Arch Neurol Psychiatry* 25: 356-372, 1931
25) Tarlov IM: Structure of the nerve root: I. Nature of the junction between the central and the peripheral nervous system. *Arch Neurol Psychiatry* 37: 555-583, 1937
26) De Ridder D, Møller A, Verlooy J, Cornelissen M, De Ridder L: Is the root entry/exit zone important in microvascular compression syndromes? *Neurosurgery* 51: 427-434, 2002
27) Lang J, Reiter U: The intracisternal length of the VII to XII cranial nerves. *Neurochirurgia (Stuttg)* 28: 153-157, 1985
28) Campos-Benitez M, Kaufmann AM: Neurovascular compression findings in hemifacial spasm. *J Neurosurg* 109: 416-420, 2008
29) Jannetta PJ, Abbasy M, Maroon JC, Ramos FM, Albin MS: Etiology and definitive microsurgical treatment of hemifacial spasm. *J Neurosurg* 47: 321-328, 1977
30) Han IB, Chang JH, Chang JW, Huh R, Chung SS: Unusual causes and presentations of hemifacial spasm. *Neurosurgery* 65: 130-137, 2009
31) Ruby JR, Jannetta PJ: Hemifacial spasm: ultrastructural changes in the facial nerve induced by neurovascular compression. *Surg Neurol* 4: 369-370, 1975
32) Nomura K, Ryu H, Ohno K, Sato K: Wide distribution of central myelin segment along the facial nerve might explain hemifacial spasm with distal nerve compression. *Clin Anat* 34: 405-410, 2021
33) Takahashi M: The anterior inferior cerebellar artery, in Newton TH, Potts DG (eds): Radiology of the Skull and Brain: Angiography, Book 2. St. Louis, Mosby, 1974, pp 1796-1808
34) Goto Y, Inoue T: Common trunk anomaly of the anterior and posterior inferior cerebellar artery in hemifacial spasm. *Acta Neurochir (Wien)* 164: 2945-2951, 2022
35) Li Y, Zheng X, Hua X, et al.: Surgical treatment of hemifacial spasm with zone-4 offending vessel. *Acta Neurochir (Wien)* 155: 849-853, 2013
36) Amano Y, Asayama B, Noro S, et al.: Significant correlation between delayed relief after microvascular decompression and morphology of the abnormal muscle response in patients with hemifacial spasm. *Neurol Med Chir (Tokyo)* 62: 513-520, 2022

Corresponding author: Takafumi Shimogawa, MD, PhD
Department of Neurosurgery, Graduate School of Medical Sciences, Kyushu University, 3-1-1 Maidashi, Higashi-ku, Fukuoka, 812-8582, Japan.
e-mail: shimogawa.takafumi.338@m.kyushu-u.ac.jp