Neurol Med Chir (Tokyo) 51, 371～375, 2011

Trans-postpyramidal Fissure Approach for Ventral Vermian Cavernous Hemangioma

—Case Report—

Hiroshi NAKAGUCHI,[1] Katsumi HOYA,[1] Shoko YAMADA,[1] Mineko MURAKAMI,[1] Akira MATSUNO,[1] Kazuto YAMAZAKI,[2] and Yasuo ISHIDA[2]

Departments of [1]Neurosurgery and [2]Pathology, Teikyo University Chiba Medical Center, Ichihara, Chiba

Abstract

A 54-year-old man presented with a rare case of cavernous angioma located in the ventral vermis manifesting as sudden onset of vertigo, followed by cerebellar ataxia. T_1-weighted magnetic resonance imaging revealed a 1.5-cm homogeneously enhanced lesion in the ventral vermis. The postpyramidal fissure was opened widely, and upward exploration through the incision enabled piecemeal removal of the lesion without deterioration of the symptoms. The histological diagnosis was cavernous hemangioma with ruptured enlarged varices. The trans-postpyramidal fissure approach allows visualization of the entire fourth ventricle and avoids damaging the cerebellar nuclei and major postoperative cerebellar dysfunction.

Key words: cerebellar vermis, cavernous hemangioma, postpyramidal fissure, fastigial nucleus, fourth ventricle

Introduction

Surgical approaches for lesions located in the ventral cerebellar vermis are limited.[8,9,13] Vertical splitting of the vermis has occasionally been applied for such lesions, but truncal ataxia or cerebellar mutism was frequently observed after surgery.[1,2,4,6] The vermis and paravermian regions contain many cerebellar nuclei, including the fastigial nucleus, globosal nucleus, and emboliformic nucleus, as well as many cerebellar tracts, including the anterior and posterior spinocerebellar tracts and cuneocerebellar tract.[2] These parts of the nervous system are involved in maintaining postural stability against gravity by controlling the tonus of the skeletal muscles, especially while standing or walking. Paravermian regions include the globosal nucleus and the emboliformic nucleus, and fibers from the vermian cortex project to the fastigial nucleus. Fibers from these cerebellar nuclei project to the red nucleus via the upper cerebellar peduncle. The rubrospinal tract, rubroreticular tract, and reticulospinal tract regulate the function of the ipsilateral motor fibers. These tracts, which connect the caudate nucleus and the putamen, also influence extrapyramidal function. Injury to the pyramid and the uvula causes cerebellar mutism, and injury to the upper vermis results in truncal ataxia, because these regions are closely connected with the cerebellospinal tract via the upper cerebellar peduncle. Injury to the flocculus and the nodulus (the old cerebellum) leads to truncal ataxia. Therefore, splitting of the upper cerebellar surface or the pyramid and the uvular surface induces severe cerebellar dysfunction.[10] Consequently, surgical procedures for ventral vermian lesions must avoid injuries to these nuclei and nerve tracts.

Here we report a case of ventral vermian cavernous hemangioma which was treated using a surgical approach to the ventral vermis via the postpyramidal fissure, which is the secondary fissure of the vermis located between the pyramid and the uvula.

Received July 1, 2010; Accepted September 27, 2010

Case Presentation

A 54-year-old man experienced sudden onset of vertigo on June 5, 2006, when he was lying down at home. The vertigo continued for about 10 minutes, and afterwards he felt dizzy when walking. After several days, he visited the outpatient clinic of the department of internal medicine at our hospital. T_1-weighted magnetic resonance (MR) imaging revealed a 1.5-cm lesion in the ventral vermis, homogeneously enhanced by gadolinium-diethylenetriaminepenta-acetic acid (Gd-DTPA), and the patient was referred to the neurosurgical department. He had no preceding major medical conditions.

Neurological examination on admission revealed that the patient had clear consciousness, with good orientation and stable gait, despite impairment of tandem gait. No dysmetria in the bilateral finger to nose test or dysdiadochokinesis was noted. External ocular movement was full and smooth, and the patient had no complaint of

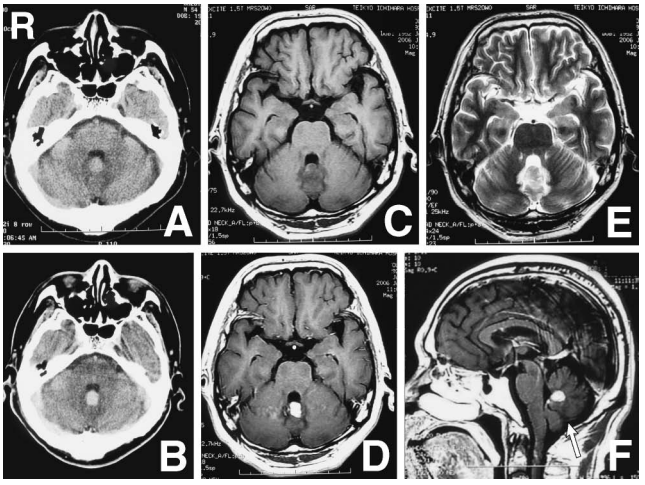

Fig. 1 A, B: Computed tomography scans revealing an isodense round mass lesion in the vermis (A) with homogeneous enhancement with contrast medium (B). C, D: Axial T_1-weighted magnetic resonance (MR) images revealing an iso-intense mass lesion measuring 1.5 × 1.3 × 2 cm in the ventral vermis (C), with clear enhancement by gadolinium (D). E: T_2-weighted MR image revealing prominent perifocal edema around the lesion. F: Sagittal T_1-weighted MR image showing the postpyramidal fissure (*arrow*).

double vision. No muscle weakness was found in the extremities, and Barre's test was negative bilaterally. No sensory disturbance was observed. Computed tomography (CT) disclosed an isodense round mass lesion in the vermis, homogeneously enhanced by contrast medium (Fig. 1A, B). T_1-weighted MR imaging revealed an iso-intense mass lesion measuring 1.5 × 1.3 × 2 cm in the ventral vermis, showing clear enhancement with Gd-DTPA (Fig. 1C, D). T_2-weighted MR imaging revealed prominent perifocal edema around the lesion (Fig. 1E). The lesion was isointense on diffusion-weighted imaging, and located at a depth of 1.7 cm from the vermian surface. Sagittal T_1-weighted MR imaging disclosed that the relatively well developed postpyramidal fissure, located 1 cm caudal to the lesion, extended to beneath the floor of the fourth ventricle (Fig. 1F). Cerebral digital subtraction angiography (DSA) revealed no tumor stain or tumor vessels.

We designed a surgical approach that involved opening of the postpyramidal fissure and subsequent cutting of the pyramid to reach the lesion. The patient was positioned prone with the head flexed slightly forward, and a midline occipital skin incision was made from over the 6th spinous process to 3 cm rostral from the inion. After a large suboccipital craniotomy using a high-speed drill, the occipital bone was rongeured off until the foramen magnum was opened to a width of 1.5 cm. The suboccipital dura was opened into a Y-shape. Cutting the arachnoid membrane over the cerebello-vermian cistern disclosed the pyramid-uvular point. The arachnoid membrane of the postpyramidal fissure was meticulously cut open between the cortical parts of the bilateral anterior inferior cerebellar arteries, and the fissure was opened widely to 2 cm beneath the cerebellar surface. The pyramid was then cut open to a width of 1 cm, and upward exploration through the incision enabled piecemeal removal of the lesion (Fig. 2).

Histological examination revealed vascular components of various calibers tightly arranged within the cerebellar tissue. Some of these vessels were partly thrombosed, and included both venous and arterial structures. Most of the vasculature had relatively thin walls, although the arterial components tended to have thickened walls. The final histological diagnosis was cavernous hemangioma with components of enlarged varices (Fig. 3).

The patient had no cerebellar symptoms such as nausea, nystagmus, ataxia, or mutism, except for one episode of vomiting after his body position was changed one day after surgery. One week after surgery, he had achieved stable gait and monopedal standing, and could go up and down stairs. Two weeks after the operation, he was discharged without any deterioration of his symptoms. Postoperative MR imaging revealed that the hemangioma in the vermis had been totally resected (Fig. 4).

Discussion

The present case of cavernous hemangioma located in the ventral vermis was treated using an approach through the postpyramidal fissure to avoid injury to the cerebellar nuclei and tracts. The postpyramidal fissure, located between the pyramid and the uvula, is the deepest of the lower vermian fissures. Sagittal T_1-weighted MR imaging detected this fissure in 12 normal adult subjects (age range 22–70 years, including 6 men and 6 women), and revealed that the distance from the deepest point of the fissure to the floor of the fourth ventricle was 15.0 ± 4.1 mm (mean ± standard deviation). Cutting of the pyramid at the deepest point of the postpyramidal fissure will not evoke severe cerebellar dysfunction, because no important cerebellar nuclei or tracts are located in the ventral pyramid. Furthermore, the floor of the fourth ventricle can be easily opened by cutting the deepest point of the postpyramidal fissure, and lesions in this location and the dorsal brainstem can be exposed by this approach without major cerebellar dysfunction (Fig. 5).

The two major alternatives for the surgical approach to the fourth ventricle are the transcerebellomedullary fissure approach[8] and the classical transvermian approach.[13] The former approach requires no cerebellar splitting but the rostral fourth ventricle cannot be observed. The latter approach allows observation of the area from the rostral to the dorsal part of the fourth ventricle, but risks postoperative cerebellar dysfunction evoked by splitting of the vermis and destroying the fastigial nucleus and the vestibulocerebellar tract (afferent fibers), and the vermian corticofugal axons (efferent fibers).

The present trans-postpyramidal fissure approach allows both visualization of the entire fourth ventricle and the caudal orifice of the aqueduct,[13] and avoids any major postoperative cerebellar dysfunction because no critical structures are located between the bottom of the postpyramidal fissure and the nearest part of the floor of the

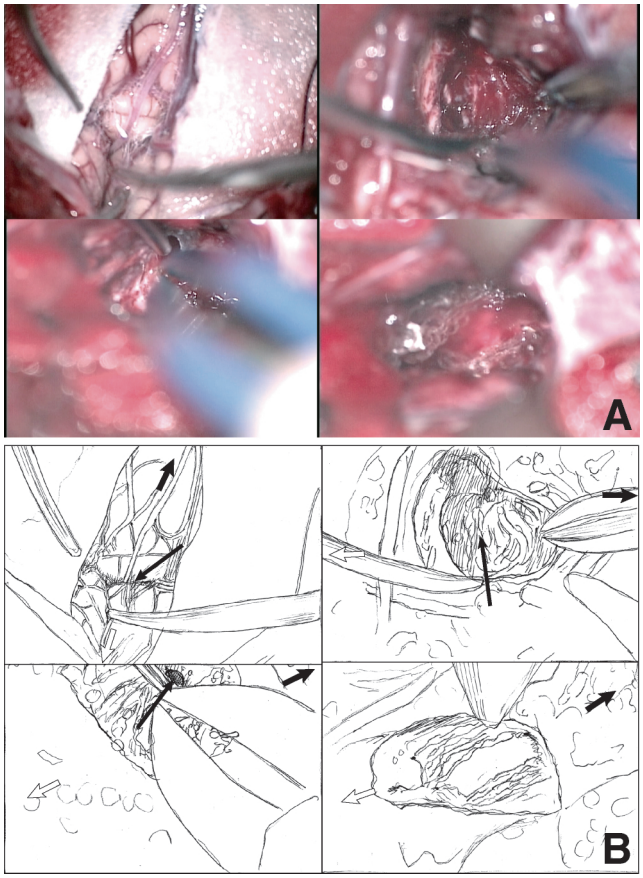

Fig. 2 Four representative intraoperative micrographs of the postpyramidal fissure approach for ventral vermian cavernous hemangioma (A) and explanatory schemas (B). *Thick black arrows* indicate the rostral direction and *thick white arrows* the caudal direction. *Upper left*: Arachnoid membranes above the postpyramidal fissure (*arrow*) have been cut using microscissors on the dorsal surface of the vermis. *Upper right*: The ventral vermian hemangioma is exposed widely by the postpyramidal fissure approach. No major axonal tracts or cerebellar nuclei would be destroyed by opening of the bottom of the postpyramidal fissure (*arrow*) and direct access to the floor of the fourth ventricle. *Lower left*: The ventral vermian cavernous hemangioma has been removed piecemeal via the postpyramidal fissure approach. Opening of the ependyma on the floor of the fourth ventricle (*arrow*) and leakage of cerebrospinal fluid are shown. *Lower right*: The final surgical stage. Surgical cellulose sheets have been spread on the surface of the cavity after resection of the vermian cavernous hemangioma.

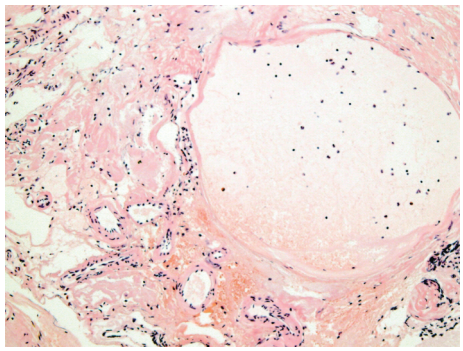

Fig. 3 Representative photomicrograph revealing cavernous hemangioma of the vermis with enlarged ruptured varices. Hematoxylin and eosin stain, original magnification ×10.

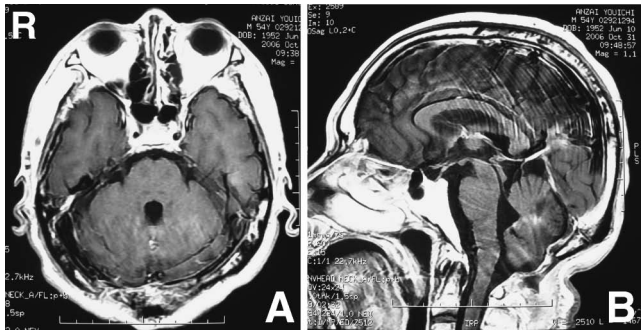

Fig. 4 Postoperative axial (A) and sagittal (B) T_1-weighted magnetic resonance images with contrast medium revealing that the hemangioma in the vermis was totally resected.

fourth ventricle (Fig. 5). The major shortcoming of this approach is difficulty with visualization of the caudal part of the fourth ventricle. If parts of the vermian lesion intrude into the caudal part of the fourth ventricle, the transcerebellomedullary fissure approach or a combination of these two approaches should be selected. The neighboring structures including the cerebellar nuclei might be destroyed by careless retraction of the cavity wall and disorientation of the microscopical axis, but might be avoided by gentle manipulation during the surgical procedures and utilization of an intraoperative navigation system. Sagittal T_1-weighted MR imaging with contrast medium is very useful for planning and carrying out this approach, because the relationship between the lesion and the postpyramidal fissure can be easily visualized.

Preoperatively, we considered high-grade glioma to be the most likely diagnosis, with metastatic tumor, hemangioblastoma (von Hippel-Lindau disease), and malignant lymphoma as differential diagnoses, because the lesion was enhanced homogeneously with prominent perifocal edema on MR imaging. We never considered the possibility of vascular malformation because CT revealed no calcification and DSA showed no tumor vessels, and cavernous hemangioma in the vermis has rarely been reported. The cerebellum is rarely affected by cavernous hemangioma. However, cerebellar cavernous hemangioma is known,[3] and most cavernous hemangiomas are angiographically occult. CT without enhancement commonly shows cavernous hemangiomas as calcified, but some are non-calcified. The key symptom that was diagnostically suggestive was the initial sudden onset of vertigo, and the intralesional hemorrhage evident on CT.

The present case of cavernous hemangioma in the ventral vermis shows that cavernous hemangioma should be considered as one of preoperative differential diagnoses of lesions in this region.[5,7,12] The trans-postpyramidal fissure

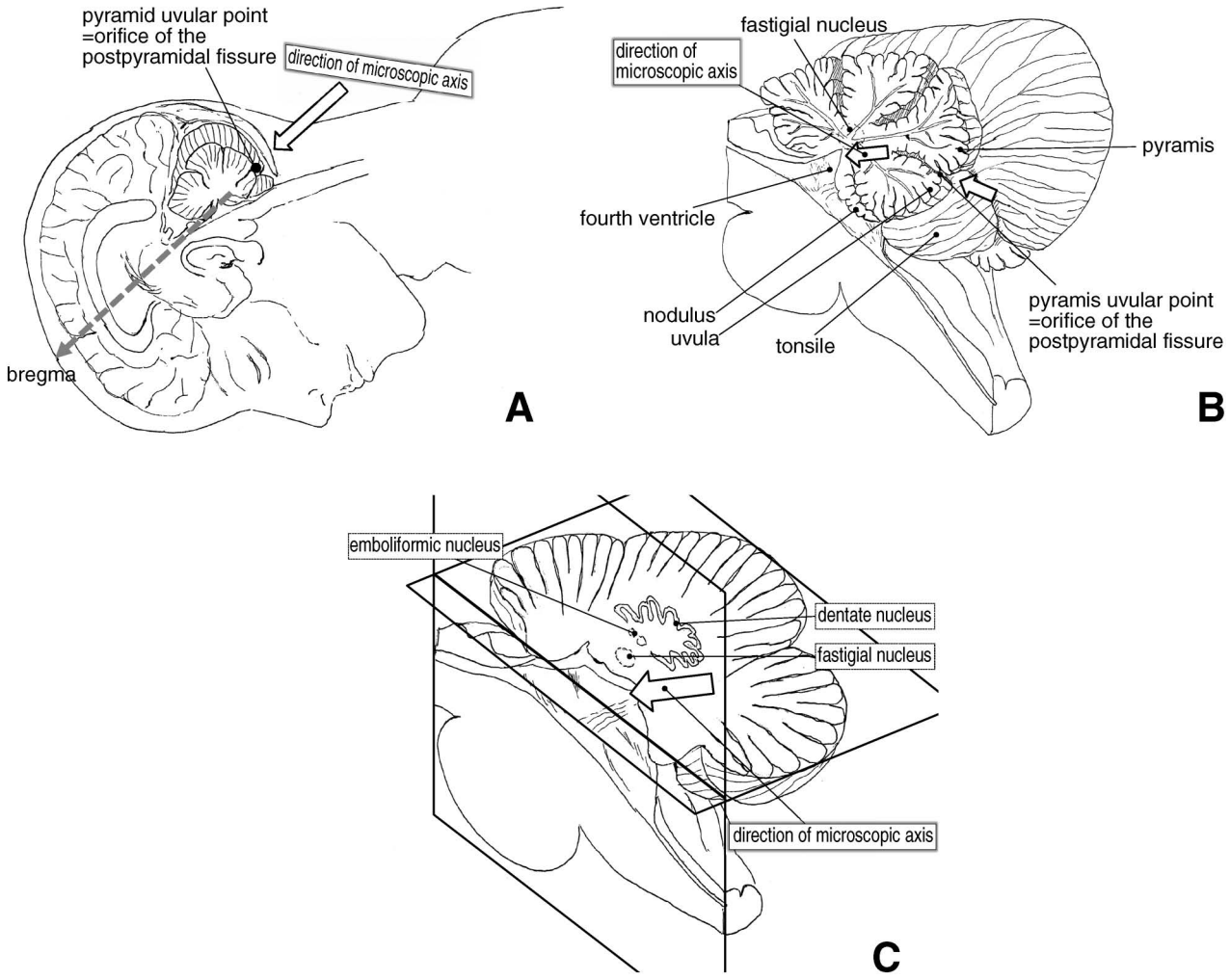

Fig. 5 Schematic demonstration of the postpyramidal fissure approach. The midline sagittal sections of the brainstem (A) and cerebellum (B) show the direction of the postpyramidal fissure approach. The relationship (C) between the postpyramidal fissure approach and the fastigial nucleus, the emboliformic nucleus, and the dentate nucleus (midline sagittal section and coronal section through the dentate nucleus of the cerebellum). There are no critical structures between the bottom of the postpyramidal fissure and the nearest part of the floor of the fourth ventricle.

approach should be considered for ventral vermian lesion associated with wide postpyramidal fissure, because of the advantages for visualization of the entire fourth ventricle and avoidance of major postoperative cerebellar dysfunction.

References

1) Bastian AJ, Mink JW Kaufman BA, Thach WT: Posterior vermal split syndrome. *Ann Neurol* 44: 601–610, 1998
2) Berry MM, Standring SM, Bannister LH: Regional organization of the central nervous system, rhombencephalon, cerebellum, *in* Williams PL, Bannister LH, Berry MM, Collins P, Dyson M, Dussek JE, Ferguson MWJ (*eds*): *Gray's Anatomy*. London, Churchill Livingstone, 1995, pp 1027–1065
3) Bruner JM, Tien RD, McLenodon RE: Vascular hamartomas, *in* Bigner DD, McLendon RE, Bruner JM (*eds*): *Russell & Rubinstein's Pathology of Tumours of the Nervous System, ed 6, vol 2*. London, Arnold, 1998, pp 260–264
4) Di Cataldo A, Dollo C, Astuto M, La Spina M, Ippolito S, Papotto M, Giuffrida S: Mutism after surgical removal of a cerebellar tumor: two case reports. *Pediatr Hematol Oncol* 18: 117–121, 2001
5) Hayashi T, Fukui M, Shyojima K, Utsunomiya H, Kawasaki K: Giant cerebellar angioma in an infant. *Childs Nerv Syst* 1: 230–233, 1985
6) Ildan F, Tuna M, Erman T, Gocer AI, Zeren M, Cetinalp E: The evaluation and comparison of cerebellar mutism in children and adults after posterior fossa surgery: report of two adult cases and review of the literature. *Acta Neurochir (Wien)* 144: 463–473, 2002
7) Kadota O, Sakaki S, Kumon Y, Ohta S, Kohno K: Large cystic cavernous angioma of the cerebellum—case report. *Neurol Med Chir (Tokyo)* 34: 768–772, 1994
8) Matsushima T, Inoue T, Inamura T, Natori Y, Ikezaki K, Fukui M: Transcerebellomedullary fissure approach with special reference to methods of dissecting the fissure. *J Neu-*

rosurg 94: 257–264, 2001
9) Mussi AC, Rhoton AL Jr: Telovelar approach to the fourth ventricle: microsurgical anatomy. *J Neurosurg* 92: 812–823, 2000
10) Pollack IF, Polinko P, Albright AL, Towbin R, Fitz C: Mutism and pseudobulbar symptoms after resection of posterior fossa tumors in children: incidence and pathophysiology. *Neurosurgery* 37: 885–893, 1995
11) Seeger W: *Microsurgery of the Brain: Anatomical and Technical Principles, vol 2*. Wien, Springer-Verlag, 1980, pp 680–693
12) Sung DI, Chang CH, Harisiadis L: Cerebellar hemangioblastomas. *Cancer* 49: 553–555, 1982
13) Tanriover N, Ulm AJ, Rhoton AL Jr, Yasuda A: Comparison of the transvermian and telovelar approaches to the fourth ventricle. *J Neurosurg* 101: 484–498, 2004

Address reprint requests to: Hiroshi Nakaguchi, MD, Department of Neurosurgery, Teikyo University Chiba Medical Center, 3426–3 Anesaki, Ichihara, Chiba 299–0111, Japan.
e-mail: hnakaguti@gmail.com