Clinical characteristics of arteriovenous malformations in the cerebellopontine angle cistern

Kazuhiko Nishino, MD,¹ Hitoshi Hasegawa, MD,¹ Kenichi Morita, MD,¹ Masafumi Fukuda, MD,¹ Yasushi Ito, MD,¹ Yukihiro Fujii, MD,¹ and Mitsuya Sato, MD²

¹Department of Neurosurgery, Brain Research Institute, Niigata University, Niigata; and ²Gamma Knife Center, Kitanihon Neurosurgical Hospital, Gosen, Japan

OBJECTIVE Arteriovenous malformations (AVMs) in the cerebellopontine angle cistern (CPAC) are specific lesions that can cause neurovascular compression syndromes as well as intracranial hemorrhage. Although case reports describing the CPAC AVMs, especially those presenting with trigeminal neuralgia (TN), have been accumulating by degrees, the pathophysiology of CPAC AVMs remains obscure. The authors' purpose in the present study was to evaluate the clinical and radiographic features of CPAC AVMs as well as the treatment options.

METHODS This study defined a CPAC AVM as a small AVM predominantly located in the CPAC with minimal extension into the pial surface of the brainstem and closely associated with cranial nerves. All patients with CPAC AVMs treated in the authors' affiliated hospitals over a 16-year period were retrospectively identified. Clinical charts, imaging studies, and treatment options were evaluated.

RESULTS Ten patients (6 men and 4 women), ranging in age from 56 to 77 years (mean 65.6 years), were diagnosed with CPAC AVMs according to the authors' definition. Six patients presented with hemorrhage, 3 with TN, and the remaining patient developed a hemorrhage subsequent to TN. Seven AVMs were associated with the trigeminal nerve (Group V), and 3 with the facial-vestibulocochlear nerve complex (Group VII–VIII). All patients in Group VII–VIII presented with the hemorrhage instead of hemifacial spasm. Regarding angioarchitecture, the intrinsic pontine arteries provided the blood supply for all CPAC AVMs in Group V. In addition, 5 of 7 AVMs with hemorrhagic episodes accompanied flow-related aneurysms, although no aneurysm was detected in patients with TN alone. With respect to treatment, all patients with hemorrhagic presentation underwent Gamma Knife surgery (GKS), resulting in favorable outcomes except for 1 patient who experienced rebleeding after GKS, which was caused by the repeated rupture of a feeder aneurysm. The AVMs causing TN were managed with surgery, GKS, or a combination, according to the nidus-nerve relationship. All patients eventually obtained pain relief.

CONCLUSIONS Clinical symptoms caused by CPAC AVMs occur at an older age compared with AVMs in other locations; CPAC AVMs also have distinctive angioarchitectures according to their location in the CPAC. Although GKS is likely to be an effective treatment option for the CPAC AVMs with hemorrhagic presentations, it seems ideal to obliterate the flow-related aneurysms before performing GKS, although this is frequently challenging. For CPAC AVMs with TN, it is important to evaluate the nidus-nerve relationship before treatment, and GKS is especially useful for patients who do not require urgent pain relief.

<http://thejns.org/doi/abs/10.3171/2015.12.JNS152190>

KEY WORDS arteriovenous malformation; cerebellopontine angle; Gamma Knife surgery; vascular disorders

THE management of intracranial arteriovenous malformations (AVMs) continues to progress, due to the recent development and refinement of multiple therapeutic modalities.^{20,25,29} However, it remains challenging to treat AVMs in and around the brainstem because of

their vicinity to vital structures and arterial inflow from critical en passage brainstem-perforating vessels.^{12,17,21} In this context, there exists a distinct type of AVM that has a small nidus and sits extrinsically on and around the pial surface of the brainstem rather than intrinsically in the pa-

ABBREVIATIONS AICA = anterior inferior cerebellar artery; AVM = arteriovenous malformation; BVR = basal vein of Rosenthal; CISS = constructive interference in steady state; CPAC = cerebellopontine angle cistern; DSA = digital subtraction angiography; GKS = Gamma Knife surgery; ICH = intracerebral hemorrhage; IPA = intrinsic pontine artery; IPS = inferior petrosal sinus; MRA = MR angiography; mRS = modified Rankin Scale; SAH = subarachnoid hemorrhage; SCA = superior cerebellar artery; SPS = superior petrosal sinus; TN = trigeminal neuralgia; TOF = time of flight.

SUBMITTED September 18, 2015. **ACCEPTED** December 29, 2015.

INCLUDE WHEN CITING Published online April 1, 2016; DOI: 10.3171/2015.12.JNS152190.

renchyma. There is growing evidence that this unique feature allows this type of AVM to be treated by microsurgery or radiosurgery.^{1,5,30} In particular, cases in which the AVM is present in the cerebellopontine angle cistern (CPAC) have been reported in an anecdotal fashion because of their unique clinical symptoms, including trigeminal neuralgia (TN) and hemifacial spasm.^{7,13–15,27} However, there have been insufficient investigations of the pathophysiology of AVMs in this specific location.

We herein defined a CPAC AVM as a small AVM that predominantly sits in the CPAC with minimal extension into the pial surface of the brainstem and that closely associates with cranial nerves in the cistern. We evaluated the clinical and radiographic features as well as treatment options for this specific entity.

Methods

Between 1999 and 2014, we treated 249 patients with AVMs at Niigata University Hospital and an affiliated hospital. Among these patients, we identified 10 consecutive patients with CPAC AVMs. We strictly defined a CPAC AVM for this study as a lesion with a small nidus (less than 1 ml in volume or less than 1 cm in diameter) located predominantly in the CPAC, with minimal extension into the pial surface of the brainstem. Thus, lesions that were larger or that were located intrinsically in the parenchyma were excluded from this analysis. The medical charts of included patients were reviewed to evaluate patients' backgrounds, clinical symptoms, and treatment options. The exact location and the extent of the AVMs were identified by MRI obtained before treatment, including 2D time-of-flight (TOF) and constructive interference in steady state (CISS) imaging. The angioarchitecture of the nidus was evaluated using digital subtraction angiography (DSA). In patients who underwent Gamma Knife surgery (GKS), results of MRI, MR angiography (MRA; 3D TOF), and DSA obtained during the follow-up periods were reviewed to evaluate the obliteration of the AVM as well as radiation-related complications. The ethics committee at Niigata University School of Medicine approved this study, and informed consent was obtained from each patient.

Results

The 10 patients included 6 men and 4 women, with an average age of 65.6 years (range 56–77 years). Table 1 presents clinical, radiographic, treatment, and follow-up data.

Clinical Presentation

Six patients presented with intracranial hemorrhage—4 patients presented with subarachnoid hemorrhage (SAH) and 2 with intracerebral hemorrhage (ICH) with subarachnoid extension. Three patients presented with TN alone, and another patient presented with SAH subsequent to a facial pain due to TN.

Radiographic Characteristics

Localization of CPAC AVMs

The CPAC AVMs were divided into 2 distinct groups by their localization—a group in which the nidus was

closely associated with the trigeminal nerve (Group V, $n = 7$) (Fig. 1) and another group that had close association with the facial-vestibulocochlear nerve complex (Group VII–VIII, $n = 3$) (Fig. 2). The AVMs causing TN were naturally included in Group V, and all other patients with AVMs in Group V presented with hemorrhages. All patients in Group VII–VIII presented with hemorrhages, and no patient showed hemifacial spasm.

Arterial Supply

All AVMs in Group V received feeders from the superior cerebellar artery (SCA) and intrinsic pontine arteries (IPAs). In particular, IPAs functioned as the main feeders for the AVMs in 4 patients (Cases 4–7) who presented with TN during their clinical course (Fig. 3). Also, additional feeders from the anterior inferior cerebellar artery (AICA) were seen in 5 cases and from the middle meningeal artery and tentorial artery in 1 case. On the other hand, the AICA was the main feeder in all patients in Group VII–VIII (Fig. 2), and 1 case received an additional feeder from the SCA.

Venous Drainage

Generally, draining veins for CPAC AVMs emptied into petrosal veins and subsequently joined the venous sinuses. In Group V, 5 of 7 cases used the superior petrosal sinus (SPS) alone or in combination with multiple draining routes, and the other 2 cases had a single venous drainage route—one via the basal vein of Rosenthal (BVR) and the other via the inferior petrosal sinus (IPS). In Group VII–VIII, all cases had multiple draining routes—the SPS and BVR in 2, and the SPS and IPS in 1. Neither marked venous ectasia nor varix formation was detected among the draining veins.

Aneurysms Associated With the AVM

Of the 7 patients who experienced hemorrhage, aneurysm formation was detected in 5, including 3 feeder aneurysms and 2 intranidal aneurysms. The feeder aneurysms were localized adjacent to the nidus in 2 patients (Fig. 2) and remote from the nidus in 1 patient (Fig. 4). No AVM-associated aneurysms were detected in patients presenting with TN alone.

Treatment of AVMs

Based on the principles of treatment for AVMs presenting with hemorrhages, direct manipulation of the nidus was avoided during the acute phase to the extent possible. Therefore, 6 of 7 patients with hemorrhages were initially managed with conservative treatment and subsequently underwent GKS. One exception was Case 1, who presented with an SAH accompanying a feeder aneurysm on the AICA far from the nidus (Fig. 4). Because the aneurysm was considered to be a bleeding source, the feeder was clipped during the acute phase, resulting in ipsilateral deafness; GKS was subsequently performed for the remaining nidus. In patients with hemorrhagic presentation, the modified Rankin Scale (mRS) score immediately before GKS was 0 in 3 patients, 1 in 3, and 3 in 1. Of 3 patients presenting with TN alone, the chosen treatments

TABLE 1. Clinical and demographic data, angioarchitecture, treatments, and outcomes in 10 patients with CPAC AVMs

Case No.	Age (yrs)/ Sex	Mode of Onset	Assoc CN	Nidus Vol, ml	Feeders*	Draining Veins	Assoc Aneurysms	mRS Score	Symptoms	Treatments		Outcome	
										First	Second	FU	Final mRS Score
1	58/F	SAH	V	0.2	SCA, AICA, IPA (1)	SPS	Feeder (AICA)	1	Unilat deafness	Feeder clipping†	GKS (19 Gy)	6 yrs	1
2	65/M	SAH, ICH	V	0.3	SCA, AICA, IPA (2)	SPS, BVR	Intranidal	3	Hemiparesis, ataxia	GKS (18 Gy)	None	12 yrs	5‡
3	75/M	SAH, ICH	V	0.8	SCA, AICA, IPA (2)	SPS, BVR	None	1	Ataxia	GKS (20 Gy)	None	5 yrs	1
4	70/M	TN → SAH	V	0.6	SCA, MMA, TentA, IPA (2)	IPS	Feeder (IPA)	0	None	GKS (16 Gy)	None	16 mos	0
5	59/M	TN	V	0.1	SCA, AICA, IPA (2)	SPS	None	0	TN	Dissection of draining veins	GKS (18 Gy)	7 yrs	0
6	69/M	TN	V	0.4	SCA, AICA, IPA (1)	BVR	None	0	TN	GKS (18 Gy)	None	10 yrs	0
7	56/F	TN	V	0.6 cm§	SCA, IPA (1)	SPS, IPS	None	0	TN	Feeder clipping	None	3 yrs	0
8	77/M	SAH	VII–VIII	0.3	AICA	SPS, IPS	Intranidal	1	Unilat deafness	GKS (20 Gy)	None	8 yrs	6‡
9	58/F	SAH	VII–VIII	0.1	AICA	SPS, BVR	None	0	None	GKS (16 Gy)	None	5 yrs	0
10	69/F	SAH	VII–VIII	0.2	SCA, AICA	SPS, BVR	Feeder (AICA)	0	None	GKS (20 Gy)	Embolization (after rebleeding)	14 mos	4

Assoc = associated; AV = arteriovenous; CN = cranial nerve; FU = follow-up; MMA = middle meningeal artery; TentA = tentorial artery.

* An artery in boldface type indicates a main feeder, and a number in parentheses indicates the number of the IPA.

† The feeder was clipped during the acute phase in Case 1.

‡ The decrease of mRS score was caused by reasons unrelated to AVMs.

§ The volume could not be calculated exactly in Case 7.

FIG. 1. Case 7 (Group V), which presented with TN. **A:** Right vertebral angiogram, Townes view, demonstrating the CPAC AVM fed by the SCA (arrow) and the IPA (double arrows). **B:** MR 2D TOF image. **C:** MR CISS image. Note the abnormal vessels tangling in the right trigeminal nerve (white arrow).

were more variable. In Case 5, the initial surgery exposed the compression of the nerve root by draining veins; thus, the draining veins were dissected from the nerve, and GKS was later performed for the remaining nidus (Fig. 3). Case 7 was also managed surgically, resulting in feeder clipping because the nidus was embedded in the trigeminal nerve (Fig. 1). The remaining patient (Case 6) was treated with GKS alone.

Patient Follow-Up

In 7 patients with hemorrhagic presentation, 5 received periodic clinical and radiographic evaluation more than 3 years (5–13 years) after GKS. Although none of the 5 patients experienced rebleeding or neurological deterioration after GKS, the patient in Case 8 died of pneumonia 8 years after GKS, and the one in Case 2 became bedridden due to advancing age. Four of these 5 cases underwent

FIG. 2. Case 10 (Group VII–VIII), which presented with SAH. **A:** Right vertebral angiogram, straight anteroposterior view. Feeders originate from the AICA, and an aneurysm is seen on the feeders (arrow). **B:** MR 2D TOF image. **C:** MR CISS image. Abnormal vessels are seen around the VII–VIII nerve complex (white arrows in B and C).

FIG. 3. Case 5 (Group V), which presented with TN. **A:** Left vertebral angiogram, left oblique view, demonstrating the feeding from 2 IPAs (arrows). **B:** MR 2D TOF image. **C:** MR CISS image. Note the vessels crossing over the trigeminal nerve (white arrow).

follow-up DSA, resulting in complete obliteration in 2 patients (Cases 8 and 9) and residue of arteriovenous shunt in 2 patients (Cases 2 and 3). In Case 2, DSA obtained 5 years after GKS showed a disappearance of the nidus and the intranidal aneurysm but a residue of the fistulous component. Case 3 also exhibited a faint arteriovenous shunt on the DSA obtained 2 years after GKS; however, we abandoned further follow-up DSA because of the patient's advanced age. A remaining patient (Case 1) was followed using MRA because the bleeding point had been treated by the surgery, and final images demonstrated the disappearance of abnormal vessels (Fig. 4). Two patients with hemorrhagic presentation underwent GKS within 2 years. Therefore, follow-up MRA demonstrated residual nidus.

Regarding the 2 patients with hemorrhage, the one in Case 10 suffered from multiple rebleeding episodes 5 months after GKS, resulting in severe neurological deficits including disturbance of consciousness, ataxia, and hemiparesis. Angiograms obtained after the rebleeding demonstrated enlargement of the preexisting feeder aneurysm; thus, the aneurysm and some parts of the nidus were embolized with Onyx. However, the embolization was complicated by the brainstem infarction without further worsening of neurological symptoms (Figs. 2 and 5).

Of 3 patients with TN, Case 5 showed disappearance of pain immediately after vascular decompression, and the pain in Case 6, a patient who was managed with GKS alone, disappeared 6 months after GKS. In Case 7, the

FIG. 4. Case 1 (Group V), which presented with SAH. **A:** Left vertebral angiogram, straight anteroposterior view, demonstrating a feeder aneurysm on the AICA far from the nidus (arrow). **B:** Left vertebral angiogram, Townes view, obtained after the clip ligation of the AICA proximal to the aneurysm (arrowhead). **C:** MRA (3D TOF) obtained 3 years after GKS. The nidus is no longer depicted. L = left; R = right.

FIG. 5. Case 10. **A:** Right vertebral angiogram, Townes view, obtained after rebleeding. Note the enlargement of the feeder aneurysm (arrow) compared with that in Fig. 2. **B:** Right vertebral angiogram obtained after Onyx embolization, demonstrating preservation of the main trunk of the AICA. **C:** A digital radiograph demonstrating the Onyx cast. **D:** A diffusion-weighted MR image obtained after embolization. The ischemic lesion is seen in the brainstem.

patient also experienced pain relief after feeder clipping, resulting in a dose reduction for medications. Cases 5 and 6, both patients who underwent GKS, were followed by MRA, which revealed disappearance of the nidus. The prescribed dose of GKS ranged from 16 to 20 Gy, and no patient experienced complications associated with GKS, including cranial nerve palsies and brainstem dysfunction.

Discussion

Age at Onset

A noteworthy characteristic regarding the background of patients with CPAC AVMs is the age at onset. Among our cases, the mean age at presentation was 65.6 years (range 56–77 years). Gross and Du performed a meta-analysis of cerebral AVMs with data from 9 studies including 3923 patients, and described the mean age at onset as 33.7 years.⁸ In previous reports, we found 15 CPAC AVMs with morphological features that matched our definition of CPAC AVMs, and they also demonstrated an older age at onset (mean 55.5 years, range 35–76 years) (Table 2). It remains unknown why CPAC AVMs cause clinical symptoms in older individuals.

Hetts and colleagues investigated the angioarchitectural features of 833 patients with AVM and determined

a difference between children and adults.¹¹ According to their results, flow-related aneurysms, venous ectasia, and posterior fossa location were significantly more frequent among adult patients. It is reasonable to assume that flow-related aneurysms arise from chronic hemodynamic stress, and CPAC AVMs might require a longer period of time to develop aneurysms because they receive a smaller amount of the shunt flow. However, the age at onset for our cases with TN alone also exceeded 55 years. This finding may support a theory that brain AVMs are not static lesions; angioarchitectural features can change with time, resulting in the increase of somewhat adverse effects on the trigeminal nerves. Neither marked venous ectasia nor varix formation was observed in the present series. This finding might have been influenced by the small size of the nidus as well as the short length of the draining vessel due to the proximity to dural sinuses.

Arterial Supply

In the present study, the IPA participated in the feeding of all AVMs in Group V and played a role as the main feeder in cases with TN (Table 1). Similarly, the IPA provided the arterial supply to all of the previously reported AVMs presenting with TN, as shown in Table 2, although

TABLE 2. Published literature on AVMs in the CPAC

Authors & Year	Age (yrs)/Sex	Assoc CN	Symptom	Feeder(s)	Treatment	Complication
Kawano et al., 1984	48/M	V	TN	SCA, IPA	Resection	None
Edwards et al., 2002	38/M	V	TN	SCA, IPA	MVD + resection	V1 hypesthesia
	55/F	V	TN	AICA, IPA	Resection	V1–3 hypesthesia
	46/F	V	TN	IPA	Resection	V1–2 hypesthesia
	35/F	V	TN	IPA	Resection	None
	36/F	V	TN	IPA	Resection	V1–3 hypesthesia, facial palsy
Maher et al., 2003	76/M	V	ICH	SCA, IPA, ECA	Resection	None
Krischek et al., 2004	57/M	V	SAH	SCA, IPA	Resection	V1–2 hypesthesia
Karibe et al., 2004	55/M	V	TN	SCA, IPA	MVD + GKS	None
Anderson et al., 2006	39/Not given	V	TN	SCA, IPA, ECA	GKS	None
Feroli et al., 2010	52/F	V	TN	AICA, IPA	MVD + GKS	None
Sumioka et al., 2011	66/M	V	TN	SCA, IPA	MVD + GKS	None
Singh et al., 2010	45/M	V	TN	SCA, AICA, IPA	NA	NA
Kim et al., 1991	64/M	VII–VIII	HFS	AICA	MVD	None
Patel et al., 2011	50/M	VII–VIII	Vertigo	AICA	Embolization	None

ECA = external carotid artery; HFS = hemifacial spasm; MVD = microvascular decompression; NA = not applicable.

the authors of these studies used varying anatomical terms for the IPA. According to the previous anatomical studies, 2–6 trigeminal arteries that form a vascular network around the root provide the arterial supply for the trigeminal nerve root, and the trigeminal arteries usually originate from the SCA, AICA, and IPA.^{3,10} Above all, the superolateral pontine artery and the inferolateral pontine artery, which are branches of the IPA, are most often detected around the trigeminal nerve root.¹⁹ Although such anatomical findings indicate that CPAC AVMs fed by IPAs are likely to be intrinsic in the trigeminal nerve, they occasionally sit beside the trigeminal nerve, compressing the nerve by their feeders or draining veins as in Case 5.

Among previous reports, CPAC AVMs associated with the VII–VIII complex were less frequent than those associated with the trigeminal nerve, and the AICA was the main feeder in all reported cases as well as in our Group VII–VIII (Tables 1 and 2).^{15,22} It seems conceivable that this occurred because the roots of the facial and vestibulocochlear nerves receive their arterial supply from branches of the AICA, including the labyrinthine and recurrent perforating internervous arteries.^{2,31}

Treatment Options

Based on the principles of AVM management, CPAC AVMs with hemorrhagic presentation require more aggressive treatments than those with nonhemorrhagic symptoms. However, the ideal treatment option for hemorrhagic cases remains undetermined because the vast majority of CPAC AVMs in the literature presented with TN, as shown in Table 2. We selected GKS for the treatment of cases with hemorrhagic onset. We were hesitant with respect to surgery because the majority of patients had no or minimal neurological deficits during the chronic phase, which was probably due to the cisternal localization, resulting in SAH unaccompanied by ICH. Although our strategy fortunately achieved acceptable results, it

should be noted that the hemorrhagic cases in the present study were frequently accompanied by aneurysms associated with AVMs, which are considered to have high rebleeding risks.^{4,23,24,26} If GKS is chosen for the treatment of CPAC AVMs accompanied by such an aneurysm, it is ideal to obliterate the aneurysm before performing GKS. As shown in Case 1, it is feasible to manage the aneurysm with the surgery if the aneurysm is localized on the feeder far from the nidus (Fig. 4).

Embolization is another option for obliterating aneurysms. However, in cases of CPAC AVM, critical branches are likely to originate from the feeders. Actually, the patient in Case 10 developed a brainstem infarction after the embolization, even though the main trunk of the AICA was preserved (Fig. 5). Thus, if embolization is planned, it is essential to evaluate the detail of the angioarchitecture before embolization, and the pharmacological provocation test should be required in cases with minimal neurological deficits.

Compared with other treatment modalities, resection has the advantage of immediately removing the risk of rebleeding. Two patients with hemorrhagic presentation are described in Table 2. These patients underwent resection of the nidus. One patient presented with ICH causing facial hypesthesia and ataxia, and the resection resulted in no worsening of these symptoms.¹⁸ The other patient presented with SAH with no accompanying neurological deficits, but facial hypesthesia developed after the resection.¹⁶ Han et al. recently reported the results of microsurgery for brainstem AVMs, categorizing them into 6 types.⁹ The CPAC AVMs might be included in their lateral pontine type, which sits on the pial surface between the trigeminal nerve root medially and the cerebellopontine fissure laterally. The nidi were completely resected in all 7 patients with the lateral pontine type, without any neurological deterioration, although 4 of the 7 had preoperative neurological deficits with an mRS score > 3. Nozaki et al.

reported surgical results for CPA AVM.²¹ All 8 patients had hemorrhage on initial presentation, resulting in preoperative neurological deficits, including cranial nerve palsy and ataxia. Two AVMs were located completely epipially in the CPAC, and 6 had small subpial extensions. Total microsurgical resection was performed in all patients, resulting in worsening of preexisting deficits in 3 patients. Although these excellent results were achieved by experienced surgeons, surgery might be considered in cases with moderate to severe neurological deficits due to the initial hemorrhage. In addition, surgery might be indicated for AVMs with perinidal aneurysms unsuitable for embolization, causing repeat hemorrhages. On the other hand, elderly patients, who were frequently encountered in the present study, are likely to be candidates for GKS.

As shown in Table 2, CPAC AVMs presenting with neurovascular compression syndromes have been primarily managed by microsurgery; however, resection of the AVM frequently resulted in nerve root injury.^{5,16,18} On this subject, preoperative MRI (including TOF and CISS imaging) is likely to provide useful information for determining the nidus-nerve relationship. In cases in which symptoms are caused by feeders or draining vessels compressing the nerve root, vascular decompression followed by GKS seems to be a reasonable treatment.^{6,28} Although it remains unclear how to manage AVMs that induce symptoms by being embedded in the nerve root, GKS is considered a safe and effective treatment option for patients who do not require urgent relief of facial pain. Our patient treated by GKS alone showed pain disappearance 6 months later, and a patient in the previous reports no longer had painful episodes 13 months after treatment.¹

Study Limitations

Even considering the fact that CPAC AVMs are rare, the number of patients involved in the present study was small. Therefore, although the present study demonstrated that this specific entity tends to cause clinical symptoms at older ages, it was impossible to determine a statistically significant difference in the age at onset compared with that for AVMs in other locations. Also, although we primarily used GKS for the treatment of CPAC AVMs with hemorrhagic presentation, resulting in acceptable outcomes, the small number of cases means that we were unable to compare the results with those in patients who underwent resection. It is obvious that resection is superior to GKS regarding the prevention of rebleeding. To clarify the best treatment option for hemorrhagic cases, increased surgical experience seems to be required, especially with respect to complications.

Conclusions

Clinical symptoms caused by CPAC AVMs occurred in patients at an older age compared with the age of patients with AVMs in other locations. The CPAC AVMs were divided into 2 distinct groups according to associated cranial nerves, and each group had a specific pattern of arterial supply. In addition, AVMs with hemorrhagic onset frequently accompanied aneurysms in and around the nidus. Although GKS achieved acceptable results as

a treatment for AVMs with hemorrhagic presentation, it seems ideal to obliterate aneurysms associated with the AVM before performing GKS. In cases with TN, preoperative MRI might provide useful information regarding the nidus-nerve relationship, which is an important factor for selecting a treatment option. It appears that GKS is a safe and effective treatment for CPAC AVMs with TN if patients do not require urgent pain relief.

References

1. Anderson WS, Wang PP, Rigamonti D: Case of microarteriovenous malformation-induced trigeminal neuralgia treated with radiosurgery. **J Headache Pain** 7:217–221, 2006
2. Brunsteins DB, Ferreri AJ: Microsurgical anatomy of VII and VIII cranial nerves and related arteries in the cerebellopontine angle. **Surg Radiol Anat** 12:259–265, 1990
3. Cetković M, Antunović V, Marinković S, Todorović V, Vitošević Z, Milisavljević M: Vasculature and neurovascular relationships of the trigeminal nerve root. **Acta Neurochir (Wien)** 153:1051–1057, 2011
4. D'Aliberti G, Talamonti G, Cenzato M, La Camera A, Debernardi A, Valvassori L, et al: Arterial and venous aneurysms associated with arteriovenous malformations. **World Neurosurg** 83:188–196, 2015
5. Edwards RJ, Clarke Y, Renowden SA, Coakham HB: Trigeminal neuralgia caused by microarteriovenous malformations of the trigeminal nerve root entry zone: symptomatic relief following complete excision of the lesion with nerve root preservation. **J Neurosurg** 97:874–880, 2002
6. Ferrol P, Acerbi F, Broggi M, Broggi G: Arteriovenous micro-malformation of the trigeminal root: intraoperative diagnosis with indocyanine green videoangiography: case report. **Neurosurgery** 67 (3 Suppl Operative):onsE309–onsE310, 2010
7. Figueiredo PC, Brock M, Prill A: Arteriovenous malformation in the cerebellopontine angle presenting as trigeminal neuralgia. **Arq Neuropsiquiatr** 47:61–71, 1989
8. Gross BA, Du R: Natural history of cerebral arteriovenous malformations: a meta-analysis. **J Neurosurg** 118:437–443, 2013
9. Han SJ, Englot DJ, Kim H, Lawton MT: Brainstem arteriovenous malformations: anatomical subtypes, assessment of “occlusion in situ” technique, and microsurgical results. **J Neurosurg** 122:107–117, 2015
10. Hendrix P, Griessenauer CJ, Foreman P, Loukas M, Fisher WS III, Rizk E, et al: Arterial supply of the lower cranial nerves: a comprehensive review. **Clin Anat** 27:108–117, 2014
11. Hettis SW, Cooke DL, Nelson J, Gupta N, Fullerton H, Amans MR, et al: Influence of patient age on angioarchitecture of brain arteriovenous malformations. **AJNR Am J Neuroradiol** 35:1376–1380, 2014
12. Kano H, Kondziolka D, Flickinger JC, Yang HC, Flannery TJ, Niranjan A, et al: Stereotactic radiosurgery for arteriovenous malformations, Part 5: management of brainstem arteriovenous malformations. **J Neurosurg** 116:44–53, 2012
13. Karibe H, Shirane R, Jokura H, Yoshimoto T: Intrinsic arteriovenous malformation of the trigeminal nerve in a patient with trigeminal neuralgia: case report. **Neurosurgery** 55:1433, 2004
14. Kawano H, Kobayashi H, Hayashi M, Tsuji T, Kabuto M, Nozaki J: [Trigeminal neuralgia caused by arteriovenous malformation of the cerebellopontine angle—report of two cases.] **No To Shinkei** 36:1175–1179, 1984 (Jpn)
15. Kim Y, Tanaka A, Kimura M, Yoshinaga S, Tomonaga M: Arteriovenous malformation in the cerebellopontine angle presenting as hemifacial spasm—case report. **Neurol Med Chir (Tokyo)** 31:109–112, 1991

16. Kriscsek B, Yamaguchi S, Sure U, Benes L, Bien S, Bertalanffy H: Arteriovenous malformation surrounding the trigeminal nerve—case report. **Neurol Med Chir (Tokyo)** **44**:68–71, 2004
17. Lawton MT, Hamilton MG, Spetzler RF: Multimodality treatment of deep arteriovenous malformations: thalamus, basal ganglia, and brain stem. **Neurosurgery** **37**:29–36, 1995
18. Maher CO, Atkinson JL, Lane JI: Arteriovenous malformation in the trigeminal nerve. Case report. **J Neurosurg** **98**:908–912, 2003
19. Marinković SV, Gibo H: The blood supply of the trigeminal nerve root, with special reference to the trigeminocerebellar artery. **Neurosurgery** **37**:309–317, 1995
20. Natarajan SK, Ghodke B, Britz GW, Born DE, Sekhar LN: Multimodality treatment of brain arteriovenous malformations with microsurgery after embolization with Onyx: single-center experience and technical nuances. **Neurosurgery** **62**:1213–1226, 2008
21. Nozaki K, Hashimoto N, Kikuta K, Takagi Y, Kikuchi H: Surgical applications to arteriovenous malformations involving the brainstem. **Neurosurgery** **58** (4 Suppl 2):ONS-270–ONS-279, 2006
22. Patel PN, Connor S, Brew S, Gleeson MJ: An arteriovenous malformation within the internal acoustic meatus and cerebellopontine angle cistern. **J Laryngol Otol** **125**:1275–1278, 2011
23. Platz J, Berkefeld J, Singer OC, Wolff R, Seifert V, Konczalla J, et al: Frequency, risk of hemorrhage and treatment considerations for cerebral arteriovenous malformations with associated aneurysms. **Acta Neurochir (Wien)** **156**:2025–2034, 2014
24. Redekop G, terBrugge K, Montanera W, Willinsky R: Arterial aneurysms associated with cerebral arteriovenous malformations: classification, incidence, and risk of hemorrhage. **J Neurosurg** **89**:539–546, 1998
25. Rubin BA, Brunswick A, Riina H, Kondziolka D: Advances in radiosurgery for arteriovenous malformations of the brain. **Neurosurgery** **74** (Suppl 1):S50–S59, 2014
26. Signorelli F, Gory B, Pelissou-Guyotat I, Guyotat J, Riva R, Dailler F, et al: Ruptured brain arteriovenous malformations associated with aneurysms: safety and efficacy of selective embolization in the acute phase of hemorrhage. **Neuroradiology** **56**:763–769, 2014
27. Singh N, Bharatha A, O’Kelly C, Wallace MC, Goldstein W, Willinsky RA, et al: Intrinsic arteriovenous malformation of the trigeminal nerve. **Can J Neurol Sci** **37**:681–683, 2010
28. Sumioka S, Kondo A, Tanabe H, Yasuda S: Intrinsic arteriovenous malformation embedded in the trigeminal nerve of a patient with trigeminal neuralgia. **Neurol Med Chir (Tokyo)** **51**:639–641, 2011
29. Takagi Y, Sawamura K, Hashimoto N, Miyamoto S: Evaluation of serial intraoperative surgical microscope-integrated intraoperative near-infrared indocyanine green videoangiography in patients with cerebral arteriovenous malformations. **Neurosurgery** **70** (1 Suppl Operative):34–42, 2012
30. Tsubaki S, Fukushima T, Tamagawa T, Miyazaki S, Watanabe K, Kuwana N, et al: Parapontine trigeminal cryptic angiomas presenting as trigeminal neuralgia. **J Neurosurg** **71**:368–374, 1989
31. Woischneck D, Hussein S: The anterior inferior cerebellar artery (AICA): clinical and radiological significance. **Neurosurg Rev** **14**:293–295, 1991

Disclosures

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author Contributions

Conception and design: Nishino. Acquisition of data: Nishino. Analysis and interpretation of data: Nishino. Drafting the article: Nishino. Critically revising the article: Nishino. Reviewed submitted version of manuscript: Hasegawa, Fukuda. Administrative/technical/material support: Hasegawa, Morita, Sato. Study supervision: Ito, Fujii.

Correspondence

Kazuhiko Nishino, Department of Neurosurgery, Brain Research Institute, Niigata University, 1-757 Asahimachidori, Chuo-ku, Niigata 951-8585, Japan. email: nishino@bri.niigata-u.ac.jp.