

The Comparison of Clinical Findings and Treatment Between Unilateral and Bilateral Vertebral Artery Dissection

Masaki Takahara, MD,* Toshiyasu Ogata, MD, PhD,† Hiroshi Abe, MD, PhD,*
Toshio Higashi, MD, PhD,* Takashi Morishita, MD, PhD,*
Koichi Takano, MD, PhD,‡ and Tooru Inoue, MD, PhD*

Background: There are limited clinical studies of bilateral vertebral artery dissection (VAD). *Objective:* To compare the characteristics, imaging findings, and treatments between patients with bilateral and unilateral VAD. *Methods:* Between February 2007 and May 2017, 31 (mean age: 53.0 years; 23 men, 8 women) out of 171 VAD patients were hospitalized because of bilateral VAD. Onset type, dissection site, dominant side of the VA, imaging features, treatments, and outcomes were investigated based on medical records. The dominant side of the VA was determined by basi-parallel anatomical scanning. *Results:* Twenty (64.5%) of 31 patients exhibited bilateral VAD on both sides of V4. The dominant side of the VA was right in 16 patients and left in 15 patients. The pearl and string sign (an angiographical finding with both dilatation and stenosis) was frequently observed on the dominant VAD side, while a tapered occlusion and string sign were most common on the nondominant side. For clinical subtype of VAD, 6 (19.4%) patients had subarachnoid hemorrhage, 10 (32.3%) ischemic stroke, 3 (9.7%) infarction plus subarachnoid hemorrhage, and 12 (38.7%) only headache. The frequency of infarction was increased in bilateral VAD compared with unilateral ($P < .05$). Surgical intervention was performed in 3 cases, while 14 patients received endovascular intervention. *Conclusions:* Infarction occurred frequently in bilateral VAD patients, and 17 patients required an intervention (mainly endovascular) for VA. The treatment strategy varied depending on the clinical subtype, imaging findings of VAD, and morphology of the dominant VAD side.

Key Words: Vertebral artery dissections—clinical features—unilateral and bilateral—morphology—interventions

© 2019 Elsevier Inc. All rights reserved.

From the *Department of Neurosurgery, Fukuoka University, Fukuoka Japan; †Department of Neurology, Fukuoka University, Fukuoka Japan; and ‡Department of Radiology, Fukuoka University, Fukuoka Japan.

Received October 17, 2018; revision received December 13, 2018; accepted January 12, 2019.

Funding: This study was partially supported by a grant from the Clinical Research Foundation in Japan. Dr. Morishita has received grant support from the Japan Society for Promotion of Science, St. Luke Life Science Institute, Nakatomi Foundation, Takeda Science Foundation, the Uehara Memorial Foundation, and the Central Research Institute of Fukuoka University.

Conflict of interest: None.

Disclosure: Dr. Morishita has received an honoraria from Boston Scientific and Medtronic as a consultant within the past 12 months.

The information of our study: The Fukuoka Dissection Registry is a hospital-based study from our affiliated hospitals in which patients with cerebral artery dissections are prospectively enrolled. This study was approved by the human subject ethics committee at Fukuoka University hospital (IRB No.: 2016M062).

Contribution to this manuscript: M.T., drafting the manuscript for content, study concept, and analysis of data; T.O., drafting the manuscript for content, study design, statistical analysis, and study supervision; H.A., T.H., critical revision of the manuscript for intellectual content; T.M., acquisition of data and obtaining funding; K.T., critical revision of the manuscript for important intellectual content; and T.I., study supervision and obtaining funding.

Address correspondence to Toshiyasu Ogata, MD, Department of Neurology, Faculty of Medicine, Fukuoka University, 7-45-1, Namakuma, Jonan-ku, Fukuoka 814-0180, Japan. E-mail: toshiogata@fukuoka-u.ac.jp.

1052-3057/\$ - see front matter

© 2019 Elsevier Inc. All rights reserved.

<https://doi.org/10.1016/j.jstrokecerebrovasdis.2019.01.009>

Introduction

Vertebral artery dissection (VAD) has an estimated incidence of 1-1.5 per 100,000 individuals annually, and is frequently seen in the Japanese population, which contrasts with the Western population in which carotid dissection predominates.^{1,2} Although neck injury is a known cause of VAD, spontaneous VAD is increasingly reported.³ VAD is typically diagnosed based on both clinical symptoms (eg stroke and headache) and imaging findings, especially magnetic resonance imaging (MRI). We previously reported the importance of unilateral headache and neck pain for diagnosis of VAD,⁴ the use of new MRI methods to confirm VAD,⁵ and the comparison between intracranial and extracranial VADs.⁶

Bilateral VAD is rare. As a potential mechanism of bilateral VAD, it was recently suggested that VAD may continue via the VA union to the contralateral side of the VA. Alternatively, occlusion or severe stenosis on either side of the VA may increase perfusion of the contralateral side of the VA, and thus cause contralateral VAD.⁷ Several therapeutic options have been proposed for bilateral VAD to preserve flow in both VAs, and to prevent enlargement or rebleeding of the aneurysm.⁷⁻⁹

However, many of the reports concerning bilateral VAD are case reports,⁷⁻¹⁵ in which the risk factors and mechanisms of bilateral VAD were not reported. Thus, the frequency of the 2 potential mechanisms of bilateral VAD remains unclear, and the treatment strategies of bilateral VAD are not fully described. The aim of the present study was to examine the risk factors and morphological findings of bilateral VAD, and describe current treatment strategies and outcomes.

Methods

The patients who are diagnosed to have cerebral artery dissections are prospectively enrolled as previously reported.^{6,16} Among the patients in our study between February 2007 and May 2017, we used a dataset of patients with VAD in the present study. VAD was diagnosed as previously reported.^{4,6} In brief, if a patient who complained of sudden onset of headache with or without neurological deficit was suspected to have VAD, then MRI and/or digital subtraction angiography (DSA) was performed after written informed consent was obtained. This study was approved by the human subject ethics committee in our hospital. VAD was diagnosed according to the criteria from a multicenter observational study (SCADS-Japan).⁶

When we applied the recent diagnostic criteria of dissection to the patients with intracranial VAD,³ the numbers of each degree of certainty were 117 in definite, nine in probable, and 23 in possible dissection. Atherosclerotic risk factors were determined by medical records. The clinical subtype of the patients was categorized as subarachnoid hemorrhage (SAH), infarction, SAH and infarction,

and headache. Neurological severity at admission was estimated using the National Institute of Health and Stroke Scale score, while the clinical outcome was determined by the modified Rankin Scale (mRS) at discharge.

VAD was diagnosed by a high signal intensity on T1-weighted 3D turbo spin echo (VISTA) MRI within the VA wall, which indicates intramural hematoma,⁵ or by morphological findings of the pearl and string sign, string sign, pearl sign, or tapered occlusion on DSA. We obtained MRI around 7 days after the symptom onset for the evaluation of intramural hematoma. The morphology of VAD was determined and analyzed vessel-by-vessel. The site of VAD was defined as previously reported.¹⁷ For the mechanism of bilateral VAD, we reviewed whether the bilateral VAD was connected through the basilar artery union or existed separately. The dominant side of the VA was defined as the side with a diameter greater than or equal to 0.3 mm wider than the contralateral side when measured at the bilateral V4 portion within 3 cm from the union of the basilar artery. The dominant side was determined by basi-parallel anatomical scanning. When the difference was less than 0.3 mm, the VA side that was linearly connected to the BA was considered dominant. A similar method was recently reported for deciding the dominant side of the VA.¹⁸

We mainly used medical therapy such as antithrombotic and/or antihypertensive agents, while a surgical or endovascular intervention was used in some patients if required. Our policy was that medical therapy was first considered as a treatment option, although endovascular intervention was frequently performed in VAD patients with SAH. In cases with infarction in which it was difficult to continue only medical treatment, endovascular, or surgical interventions were considered. Endovascular intervention for SAH was usually conducted acutely, while intervention on patients with infarction was performed after a delay. The indications of superficial temporal artery-superior cerebellar artery bypass surgery (STA-SCA) were as follows: severe steno-occlusive lesions existed on bilateral VA; collateral formation of anterior circulation into the brain stem was insufficient; neurological symptoms were progressed regardless of the medications; and stent insertion was difficult technically. By contrast, the indication of surgery in patients with unilateral VAD was the presence of aneurysmal formation of VAD with a risk of rupture. If patients had a large dissecting VA aneurysm that involved the origin of the posterior inferior cerebellar artery, clipping or trapping of the VA aneurysm with or without occipital artery posterior inferior cerebellar artery bypass was considered. The treatment strategy was decided based on the age, clinical subtype, morphology, the degree of morphological changes, and personal choice of each patient.

Patients were divided into bilateral and unilateral VAD groups for statistical comparisons. The Fisher exact test was used to assess differences in clinical background treatment

and imaging data between the groups, the unpaired *t* test used to compare age, and the Mann–Whitney *U* test used to compare the National Institute of Health and Stroke Scale scores and mRS. Chi-square test was used for the statistical analyses of clinical subtype, morphology, and therapy. All statistical analyses were performed with statistical software (SPSS version 22.0; IBM, Armonk, New York).

Results

There were 140 patients admitted to our Neuro-center with unilateral VAD, and 31 patients with bilateral VAD. The background and characteristics of the patients are summarized in Table 1. There were no differences between the unilateral and bilateral VAD groups, except for the frequency of diabetes (unilateral 11.4% versus bilateral 25.8%). Infarction and/or SAH was observed in approximately 60% of bilateral VAD patients and 30% of unilateral VAD patients ($P < .001$). Three patients with bilateral VAD exhibited both SAH and infarction. No patients were treated with intravenous thrombolysis. Two patients with unilateral VAD had mild trauma as a potential trigger.¹⁹ The numbers of segments of the right VAD were 3 in V1, 2 in V2, 1 in V3, and 25 in V4, while the numbers of segments of the left VAD were 2 in V1, 3 in V2, 2 in V3, and 24 in V4 portion.

In bilateral VAD, 3 (9.7%) of 31 patients had the right and left sides connected via a basilar artery union, while the remaining patients had bilateral VAD that existed separately. V4 was the main site of dissection in the dominant and nondominant sides in bilateral VAD. The number of patients with unilateral and bilateral VAD was 140 and

31, respectively. Thus, a total of 202 vessels were analyzed. No patient had VAD pseudo-aneurysm formation. For imaging findings in the 62 vessels of 31 patients with bilateral VAD, the pearl and string sign was observed in 21.0% of patients, string sign in 25.8%, pearl sign in 27.4%, and tapered occlusion in 25.8%. However, the frequency of the VAD morphologies differed between VAD in the dominant and nondominant sides. The pearl sign (35.5%) occurred most frequently in the dominant side of VAD, while the string sign (32.3%) and tapered occlusion (32.3%) were mainly observed in the nondominant side. This pattern was consistently observed in the 202 vessels analyzed, indicating that the string sign and tapered occlusion were more frequent in the nondominant side of VAD, while the pearl sign was the main morphology in the dominant side of VAD (Table 2). Intramural hematoma was more frequent in the nondominant than the dominant side of the VA. The features of the bilateral VAD patients with ischemic stroke are summarized in Table 3. Seven of 13 patients with ischemic stroke showed a string sign or tapered occlusion on both sides of the VAD.

Endovascular intervention was frequently used in bilateral VAD patients, especially those with ischemic stroke (8 of 13 patients), while greater than 50% of unilateral VAD patients were treated medically (Table 4). STA-SCA was performed in the 2 bilateral VAD patients with stenosis and/or occlusion, while surgical intervention was conducted in 10% of patients with unilateral VAD. One patient with bilateral VAD died after STA-SCA. Although many patients with unilateral VAD exhibited a favorable mRS outcome of 0–1, bilateral VAD patients were significantly more severe.

Table 1. Comparison of background characteristics between patients with unilateral and bilateral VAD

	Unilateral (N = 140)	Bilateral (N = 31)	<i>P</i> value
Age	55.3 ± 13.6	53.0 ± 12.5	.39
Gender (male)	86 (61.4%)	23 (74.2%)	.22
Hypertension	87 (62.1%)	20 (64.5%)	.84
Diabetes	16 (11.4%)	8 (25.8%)	.047
Hyperlipidemia	41 (29.3%)	8 (25.8%)	.83
Smoking	53 (37.9%)	10 (32.3%)	.68
Drinking	95 (67.9%)	19 (61.3%)	.53
NIHSS score	0 (0–1)	0 (0–4)	.016
Clinical subtype			<.001
Nonstroke	98 (70%)	12 (38.7%)	
Infarction	23 (16.4%)	10 (32.3%)	
SAH	19 (13.6%)	6 (19.4%)	
Infarction + SAH	0 (0%)	Intracranial bilaterally: 6 3 (9.7%) Extra/intracranial: 2 Intracranial bilaterally: 1	

Abbreviations: NIHSS, National Institute of Health and Stroke Scale; SAH, subarachnoid hemorrhage; VAD, vertebral artery dissection. Extra/intracranial: intracranial on one side and extracranial on the other.

Table 2. Relationship of VA dominancy with VAD morphology

	Nondominant (N = 101)	Dominant (N = 101)	P value
Morphology			.004
Tapering occlusion	20 (19.8%)	11 (10.9%)	
Pearl sign	33 (32.7%)	58 (57.4%)	
Pearl and string sign	25 (24.8%)	20 (19.8%)	
String sign	23 (22.8%)	12 (11.9%)	
Intramural hematoma	56 (55.4%)	40 (39.6%)	.034
Double lumen sign	28 (27.7%)	35 (34.5%)	.36

Representative Case

A 53-year-old woman with a chief complaint of headache, dysphagia, and vertigo had ischemic stroke in the left lateral medullary and hospitalized immediately. Although the stenotic sites did not reveal high intensity on VISTA, the second VISTA acquisition indicated existence of intramural hematoma in the bilateral VA, which confirmed bilateral VAD (Fig 1). Right VA angiography revealed the string sign in the intracranial VA, while left VA angiography indicated the tapered occlusion in the intracranial VA (Fig 2). The patient complained of headache on the 16th day of hospitalization, and computed tomography showed SAH. A left VAD was the cause of SAH based on recanalization on a follow-up DSA study. Endovascular coil trapping was performed in the left VA during right VA stent placement. Her mRS at discharge was 4.

Discussion

The main finding of the present study was that bilateral VAD caused ischemic stroke more frequently than unilateral VAD. There was also a lower frequency of VAD in which both sides were connected via a union. Further, the morphology of VAD was significantly different between the dominant and nondominant sides of VAD, with the pearl sign the most common on the dominant side, and the string sign or tapered occlusion the most common on the nondominant side. There were no differences in the frequency of intracranial VAD between unilateral and bilateral VAD. However, medical therapy was used most frequently in unilateral VAD, while endovascular intervention was more common in bilateral VAD. Finally, the outcome of bilateral VAD was more severe at discharge.

In previous case reports of bilateral VAD, SAH was the main stroke subtype,^{8,9,20-23} while ischemic stroke caused by bilateral VAD was relatively uncommon.^{10,11} By contrast, in the present study we found higher rates of ischemic stroke than SAH, which is likely because many of the bilateral VAD patients with ischemic stroke had a stenocclusive lesion on either side. We also found that diabetes was a factor associated with bilateral VAD, which may have contributed to the increased rates of ischemic stroke.

There are 2 main hypotheses for the mechanism of bilateral VAD: type 1, where the dissection progresses from one side to the other through the basilar union,^{11,24} and type 2, where the dissection on one side of the VA causes hemodynamic stress on the other side, which leads to VA dissection.⁷ Type 1 is considered the main mechanism of bilateral VAD, which contrasts with the findings in the present study. The presence of separated dissection sites can be difficult to diagnose, although recent imaging technologies may provide more accurate assessment of bilateral VAD. The mechanism of bilateral VAD can be determined by examining the initial side of the dissection. We suggest that occlusion of one side of the VA causes an increase in contralateral VA flow. In turn, this may increase stress on the contralateral VA and cause dissection. Our data suggest that the initial symptoms of many patients with bilateral VAD were less severe, and that prevention of a second episode of SAH or ischemic stroke is critical. Our cohort included 3 patients with the right and left VA connected via a basilar artery union, all of whom developed severe ischemic stroke. The risks and prevention of type 1 VAD should be examined in future studies.

In the present study, patients with bilateral VAD received endovascular interventions more often than those with unilateral VAD. Endovascular intervention was frequently performed in VAD patients with SAH. However, in the present study, endovascular intervention was also used for treatment of bilateral VAD with ischemic stroke. Bilateral VAD often shows imaging findings of a string sign and/or tapered occlusion on either side. When one side requires treatment because of severe stenosis, then an endovascular intervention, particularly stent insertion, is used to prevent further ischemic stroke. However, we were concerned that using a stent to treat one side of a bilateral VAD with severe bilateral stenoses may cause occlusion of the treated side, and thus induce further ischemic stroke. To prevent this, we occasionally performed surgical interventions such as STA-SCA anastomosis in cases with severe bilateral stenoses, to maintain basilar arterial flow.^{25,26} Although STA-SCA anastomosis can preserve the perfusion of the brainstem in patients with bilateral severe VA stenoses, there are some reports of technical difficulties and potential safety concerns. Further studies are required to elucidate

Table 3. The features of bilateral VAD patients with ischemic stroke

Age	Gender	Risk factor	NIHSS	Site of lesions	Site of dissection		Plus SAH	Antithrombotic treatment on acute phase	Shape			Dominance		Intervention		sICH	mRS
					R	L			R	L	R	L	R	L			
64	M	HT, smoke, drink	1	R medulla	4	4	N	Antiplatelet	O	S	R	-	Stent	-	1		
58	M	HT, DM, HL, smoke, drink	4	L medulla, L cerebellar	4	1	N	Both	S	O	R	Stent	-	-	1		
62	M	HT, DM, HL, drink	2	Bil cerebellar	4	2	N	Antiplatelet	S	O	L	-	STA-SCA bypass	-	6 large brain stem infarction		
26	F	Drink	0	L cerebellar	2	1	N	Both	P	O	R	-	-	-	0		
68	M	HT, DM, HL, drink	4	L cerebellar	4	4	N	Antiplatelet	O	PS	L	STA-SCA bypass	-	+	4 ataxia due to left cerebellar infarction		
44	F	HT	40	L cerebellar	4	2	Y	Antiplatelet	P	S	L	Internal trapping	Stent	-	1		
53	F		2	L medulla	4	4	Y	Both	S	O	L	Stent	Internal trapping	-	4 ataxia due to Wallenberg syndrome		
49	M	HT, DM, drink	0	Bil cerebellar	2	4	N	Both	O	S	R	-	-	-	0		
61	M	HT, HL	0	Bil cerebellar	4	4	N	Antiplatelet	O	S	L	-	Stent	-	1		
66	M	HT, HL, drink	4	R medulla	1	4	N	Both	O	S	R	-	-	-	4 ataxia due to Wallenberg syndrome		
58	M	DM, smoke, drink	1	L cerebellar	4	4	N	Both	PS	O	L	-	Internal trapping	-	1		
35	M	HT, HL, Smoke	2	L medulla	4	4	N	Both	P	PS	R	Stent + coil	-	-	1		
34	M		11	R medulla, bil cerebellar	4	2	Y	Both	S	P	L	Internal trapping	Stent + coil	-	5 sever left hemiparesis due to medulla infarction		

Abbreviations: DM, diabetes mellitus; F, female; HL, hyperlipidemia; HT, hypertension; M, male; mRS, modified Rankin Scale at discharge; O, tapered occlusion; P, pearl sign; PS, pearl and string sign; S, string sign; SAH, subarachnoid hemorrhage; STA-SCA, superficial temporal artery-superior cerebellar artery.

Internal trapping: a treatment to insert coils into the dissecting aneurysm including proximal artery occlusion.

Stent + coil, stent assisted coil embolization.

Table 4. Treatment differences between patients with unilateral and bilateral VAD

	Bilateral (N = 31)	Unilateral (N = 140)	P value
Therapy			.069
Medical	14 (45.2%)	89 (63.6%)	
Antithrombosis			
Antiplatelet	13 (41.9%)	49 (35.0%)	
Anticoagulant	0 (0%)	9 (6.4%)	
Both	10 (32.5%)	14 (10.0%)	
None	8 (25.8%)	68 (48.6%)	
mRS	0 (0-2)	0 (0-0)	.92
Intervention	17 (54.8%)	51 (36.4%)	
Surgery (complications)	3 (9.7%) (1 death)	14 (10.0%) (2 cranial nerve palsy, 2 infarctions)	
mRS	4	1 (0-3)	N/A
Endovascular (complications)	14 (45.2%) (2 infarctions)	37 (26.4%) (3 infarctions, 1 infarction with sICH, 1 death)	
mRS	1 (1-1)	0 (0-2)	.21
mRS of all patients	1 (0-1)	0 (0-1)	.021

Abbreviations: N/A, not available; sICH, symptomatic intracerebral hemorrhage.

whether and which interventions should be chosen in cases with bilateral VAD with severe stenoses.

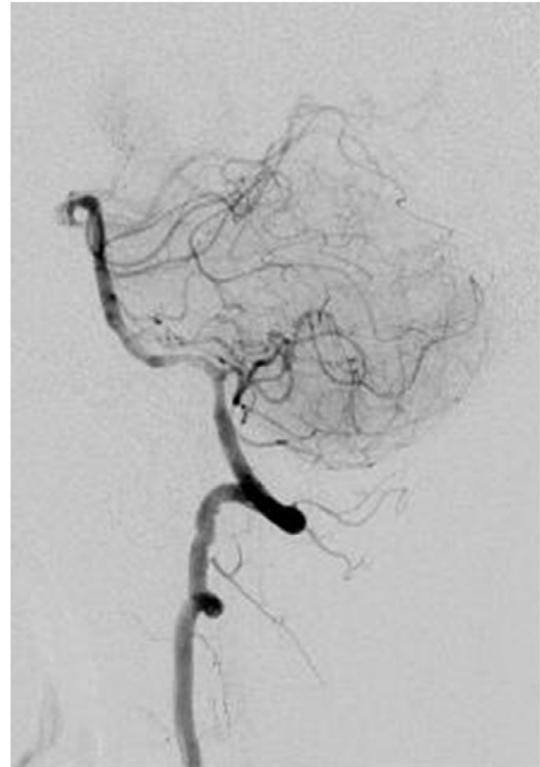
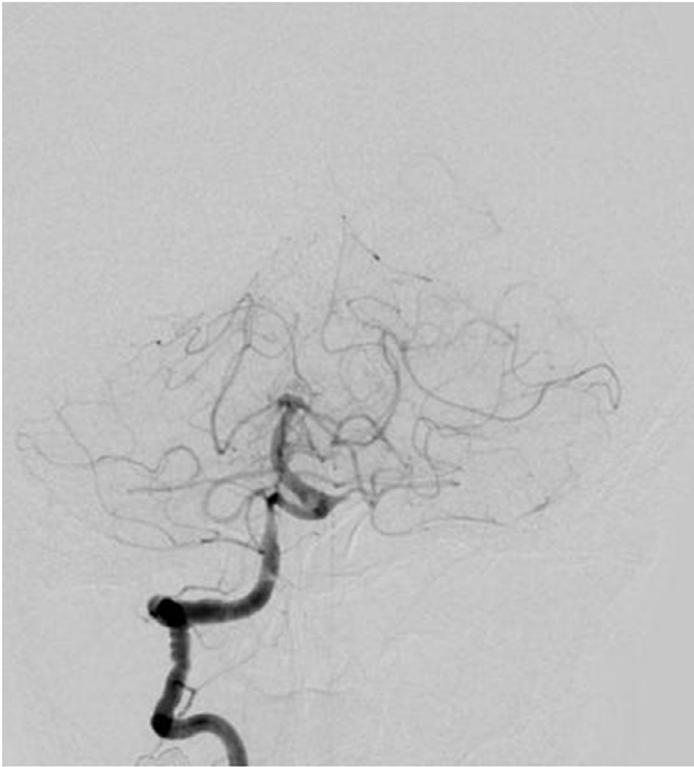
There are some potential limitations of our study. First, the number of patients participating was small. Second, this was a single center study, and thus various treatment

options were not attempted. Third, although our treatment strategy was not changed, advances in endovascular intervention equipment and techniques may have influenced our treatment decision. Fourth, antithrombotic agents were used in many cases with bilateral VAD, and



Figure 1. T1-weighted 3D turbo spin echo (VISTA) findings. Left: VISTA finding on the 5th day of hospitalization. High intensity signals in the bilateral VA were not visible. Right: VISTA finding on the 17th day. High intensity signals were detected in the bilateral VA, indicating the existence of VA dissection. VA, vertebral artery.

A



B

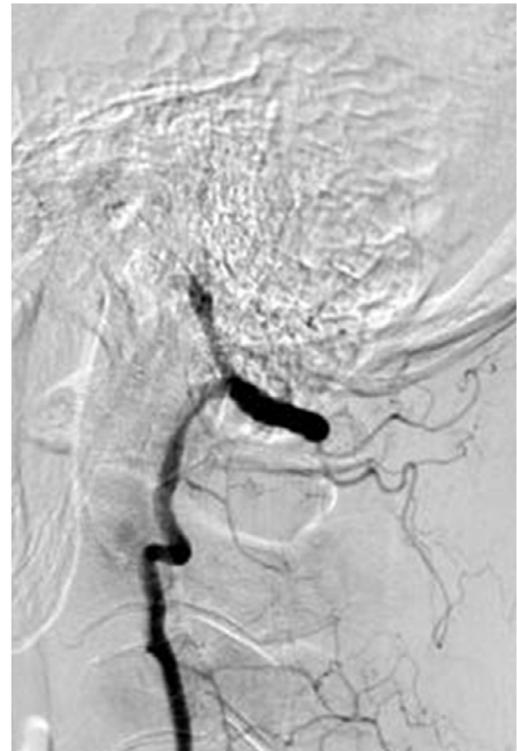
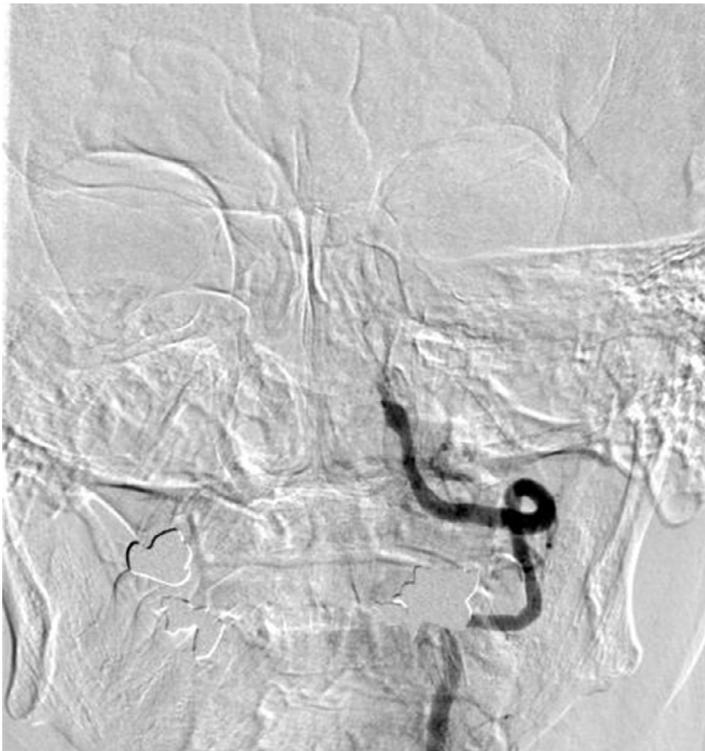


Figure 2. DSA findings in a representative case. (A) DSA on the right side of the patient, with evidence of a string sign. Left: anterior-posterior view, Right: lateral view. (B) DSA on the left side of the patient, with evidence of a tapered occlusion. Left: anterior-posterior view, Right: lateral view. DSA, digital subtraction angiographical.

it is difficult to prove the association of these medications with the incidence of SAH. Fifth, univariate analysis suggested that intramural hematoma was more frequent in the nondominant compared with the dominant side of the VA, which should be examined in future studies. Finally, we did not collect follow-up data from our patients.

Conclusion

We found that infarction was relatively frequent in bilateral VAD patients. Endovascular intervention was common in patients with bilateral VAD with SAH or infarction. The treatment strategy varied depending on the clinical subtype, imaging findings of VAD, and dominant or nondominant side of VAD.

Acknowledgments: We thank Ms. Asuka Ikezaki for support in data collection, and Edanz Group (www.edanzediting.com/ac) for editing a draft of this manuscript.

References

- Schievink WI. Spontaneous dissection of the carotid and vertebral arteries. *N Engl J Med* 2001;344:898-906.
- Redekop GJ. Extracranial carotid and vertebral artery dissection: a review. *Can J Neurol Sci* 2008;35:146-152.
- Debette S, Compter A, Labeyrie M-A, et al. Epidemiology, pathophysiology, diagnosis, and management of intracranial artery dissection. *Lancet Neurol* 2015;14:640-654.
- Fukuhara K, Ogata T, Ouma S, et al. Impact of initial symptom for accurate diagnosis of vertebral artery dissection. *Int J Stroke* 2015;10(Suppl A100):30-33.
- Takemoto K, Takano K, Abe H, et al. The new MRI modalities "BPAS and VISTA" for the diagnosis of VA dissection. *Acta Neurochir Suppl* 2011;112:59-65.
- Kobayashi H, Morishita T, Ogata T, et al. Extracranial and intracranial vertebral artery dissections: a comparison of clinical findings. *J Neurol Sci* 2016;362:244-250.
- Wilkinson DA, Wilson TJ, Stetler Jr. WR, et al. Subarachnoid haemorrhage with bilateral intracranial vertebral artery dissecting aneurysms treated by staged endovascular stenting. *BMJ Case Rep* 2013;2013:bcr0320126002.
- Ishikawa T, Yamaguchi K, Anami H, et al. Stent-assisted coil embolisation for bilateral vertebral artery dissecting aneurysms presenting with subarachnoid haemorrhage. *Neuroradiol J* 2016;29:473-478.
- Kono K, Shintani A, Fujimoto T, et al. Stent-assisted coil embolization and computational fluid dynamics simulations of bilateral vertebral artery dissecting aneurysms presenting with subarachnoid hemorrhage: case report. *Neurosurgery* 2012;71:E1192-E1200.
- Fukuda M, Aiba T, Takahashi S. Bilateral medial medullary infarction due to bilateral vertebral artery dissection. *Clinical Neurol. Neurosurg* 2004;106:132-135.
- Funaki T, Oshimoto T, Wataya T, et al. Bilateral vertebral artery dissection and its chronological changes detected by MR angiography: a case report. *No To Shinkei* 2004;56:247-250.
- Garcia-Monco JC, Fernandez Canton G, Gomez Beldarrain M. Bilateral vertebral artery dissection in a patient with afibrinogenemia. *Stroke* 1996;27:2325-2327.
- Kato Y, Nagoya H, Abe T, et al. Progressive bilateral vertebral artery dissection in a case of osteogenesis imperfecta. *J Stroke Cerebrovasc Dis* 2017;26:e43-e46.
- Frankowska E, Brzozowski K, Staszewski J, et al. Combined thrombolysis in posterior circulation stroke caused by bilateral vertebral artery dissection in squash player. *Neurol Neurochir Pol* 2014;48:299-304.
- Fukunaga A, Tabuse M, Naritaka H, et al. Spontaneous resolution of nontraumatic bilateral intracranial vertebral artery dissections. *Neurol Med Chir* 2002;42:491-495.
- Matsumoto J, Ogata T, Abe H, et al. Do characteristics of dissection differ between the posterior inferior cerebellar artery and the vertebral artery? *J Stroke Cerebrovasc Dis* 2014;23:2857-2861.
- Subclavian system of arteries. In: W PL, ed. *Gray's anatomy*, New York: Churchill Livingstone; 1995:1529-1534.
- Hong JM, Chung CS, Bang OY, et al. Vertebral artery dominance contributes to basilar artery curvature and peri-vertebrobasilar junctional infarcts. *J Neurol Neurosurg Psychiatry* 2009;80:1087-1092.
- Engelter ST, Grond-Ginsbach C, Metso TM, et al. Cervical artery dissection: trauma and other potential mechanical trigger events. *Neurology* 2013;80:1950-1957.
- Otawara Y, Ogasawara K, Ogawa A, Kogure T. Dissecting aneurysms of the bilateral vertebral arteries with subarachnoid hemorrhage: report of three cases. *Neurosurgery* 2002;50:1372-1374. discussion 4-5.
- Shin YS, Kim BM, Kim SH, et al. Endovascular treatment of bilateral intracranial vertebral artery dissecting aneurysms presenting with subarachnoid hemorrhage. *Neurosurgery* 2012;70:75-81. discussion.
- Yoon SM, Shim JJ, Kim SH, et al. Bilateral vertebral artery dissecting aneurysms presenting with subarachnoid hemorrhage treated by staged coil trapping and covered stents graft. *J Korean Neurosurg Soc* 2012;51:155-159.
- Zhao WY, Zhao KJ, Huang QH, et al. Single-stage endovascular treatment of subarachnoid hemorrhage related to bilateral vertebral artery dissecting aneurysms. *Interv Neuroradiol* 2016;22:138-142.
- Hosoya T, Adachi M, Yamaguchi K, et al. Clinical and neuroradiological features of intracranial vertebrobasilar artery dissection. *Stroke* 1999;30:1083-1090.
- Ota T, Usami K, Iijima A, et al. Staged surgical treatment for symptomatic vertebrobasilar artery stenosis: combined treatment with endovascular angioplasty and bypass surgery. *World Neurosurg* 2012;78:90-94.
- Ausman JI, Diaz FG, Vacca DF, et al. Superficial temporal and occipital artery bypass pedicles to superior, anterior inferior, and posterior inferior cerebellar arteries for vertebrobasilar insufficiency. *J Neurosurg* 1990;72:554-558.