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Original Article

Clinical Course of Abducens Nerve Palsy in Patients with Ruptured Vertebral Artery Dissecting Aneurysms

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Objective: There are few detailed reports on abducens nerve palsy due to a ruptured vertebral artery dissecting aneurysm (VADA). We investigated the clinical characteristics and long-term course of abducens nerve palsy in ruptured VADA patients treated by endovascular surgery.

Methods: Of the 51 cases of ruptured VADA treated by endovascular intervention from 2011 to 2019, 31 with a good/fair outcome, in which ocular motility disorder was able to be followed, were included and investigated.

Results: In all, 11 patients (35.5%) had abducens nerve palsy, and the World Federation of Neurological Surgeons (WFNS) grade and Hunt & Hess (H&H) grade at the time of arrival of patients with abducens nerve palsy were significantly higher than those of patients without abducens nerve palsy. Of the 10 patients who were able to be followed, abducens nerve palsy in 3 completely recovered in 7–180 days. Abducens nerve palsy improved in five patients and remained in two patients.

Conclusion: More severe neurological findings on admission reflect a higher rate of abducens nerve palsy. Diplopia induced by abducens nerve palsy is one of the most important sequelae of ruptured VADA, which impairs the daily activities of the patients. Some cases of abducens nerve palsy improve over a long period. Therefore, appropriate diagnosis and follow-up should be concerned.

Keywords ▶ abducens nerve palsy, vertebral artery dissecting aneurysm, subarachnoid hemorrhage

Introduction

Abducens nerve palsy can occur as a complication of several diseases, with the age- and sex-adjusted annual incidence of all abducens nerve palsies being 11.3 per 100000 people. Of all abducens nerve palsies, 4% are considered to be caused by cerebral aneurysms[1)] and 0.63–5.90% were reported as complications of subarachnoid hemorrhage (SAH).[2–4)] In particular, it complicates vertebral artery dissecting aneurysm (VADA) with SAH at a rate of 26.9–50.0%.[2,5)] VADA is often treated with endovascular surgery, which is considered to be less invasive.[6)] Despite this advantage, abducens nerve palsy persists in many patients after endovascular surgery, and there have been few reports about its frequency or long-term outcome. In patients with severe SAH, the examination and follow-up of abducens nerve palsy are difficult.[2,5)] In this study, we evaluated the incidence, risk factors, and clinical course of abducens nerve palsy that developed in patients who had VADA with hemorrhagic onset and were treated with endovascular surgery.

Materials and Methods

This study was carried out retrospectively at two institutions after approval by their institutional review boards and in compliance with the Declaration of Helsinki.

Of the 51 patients who had VADA with SAH and were admitted to the two study centers and received endovascular surgery between January 1, 2011, and December 31, 2019, 31 in whom detailed examination of ocular motility was possible and the modified Rankin Scale (mRS) score at the time of discharge was 0–3 were enrolled in this study.

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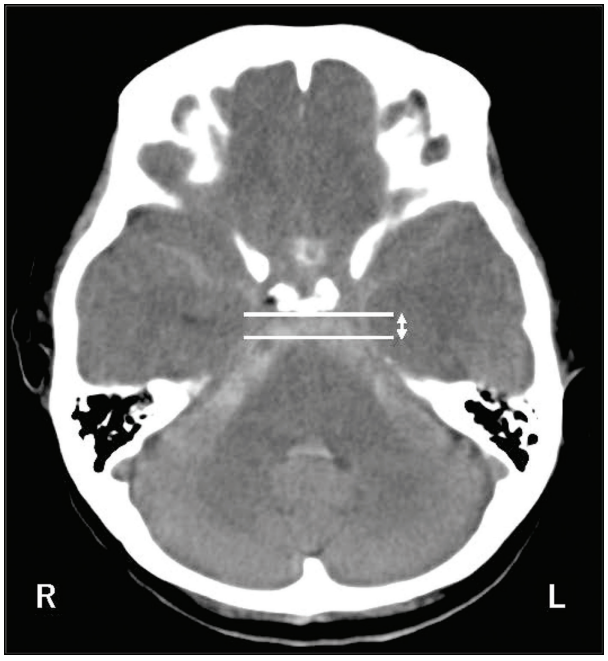

Fig. 1 Measurement of hematoma in the prepontine cistern. Hematoma size in the prepontine cistern was defined as the distance between the posterior clinoid process and the front of the pons (white double-headed arrow).

Patients with the mRS of 4–5 were excluded because most of them had disturbance of consciousness or cognitive impairment, such as aphasia, which made it difficult to accurately evaluate neurological symptoms. Abducens nerve palsy was diagnosed by the physician in charge by face-to-face testing. During the follow-up period, the patient status of abducens nerve palsy was defined as follows: "recovered" is complete recovery from abducens nerve palsy, "improved" is an improvement but not complete recovery, and "remained" is the persistence of symptoms. The clinical data included the age, sex, World Federation of Neurological Surgeons (WFNS) grade, Hunt & Hess (H&H) grade, Fisher group, presence of acute hydrocephalus, whether ventricular drainage was performed, SAH hematoma thickness, location of the aneurysm, the maximum diameter of the aneurysm, length of dissecting lesion, occluded length of artery, presence of symptomatic cerebral vasospasm, and mRS score. The SAH hematoma thickness was measured at the prepontine cistern of the posterior clinoid process level on axial plain CT (**Fig. 1**).[5] Symptomatic cerebral vasospasm was defined as cerebral arterial narrowing diagnosed by CTA or DSA in patients with new neurological deteriorations during acute management of SAH. According to these findings, the patients were divided into those with abducens nerve palsy (palsy [+] group) and those without abducens nerve palsy (palsy [−] group), and differences in the clinical features and detailed clinical characteristics in the palsy (+) group were retrospectively evaluated. In the palsy (+) group, the group with complete recovery during the observation period and the group without were also compared. Statistical comparisons between the two groups were made by Pearson's chi-square test and Mann–Whitney's U-test at the $P < 0.05$ level of significance using the R statistical package (version3.6.3; R Foundation for Statistical Computing, https://www.r-project.org/).

Results

Of the 51 VADA patients, 31 who presented with SAH and mRS ≤3 at the time of discharge were evaluated. Accurate evaluation of ocular motility and follow-up were possible in all patients. The subjects were aged 52.0 ± 10.2 years (mean ± standard deviation), there were 24 males (77.4%), and the mRS score at the time of discharge was 0 in 12 (38.7%), 1 in 9 (29.0%), 2 in 4 (12.9%), and 3 in 6 (19.4%). The mean duration of hospitalization was 59.3 days.

The WFNS grade at the time of arrival was IV or V in 17 (54.8%), the H&H grade was IV or V in 15 (48.4%). Acute hydrocephalus was observed in 12 (38.7%), 10 (32.3%) of them had ventricular drainage within 48 hours after arrival and 2 (6.1%) of them had no surgery. The median thickness of hematoma was 5.9 mm (interquartile range [IQR], 4.5–8.9 mm). The aneurysm was located on the right side in 22 (70.1%), on the distal side of the posterior inferior cerebellar artery (PICA) in 13 (41.9%), on the proximal side of the PICA in 10 (32.3%), and involved the origin of the PICA in 8 (25.8%). Endovascular surgery was performed within 48 hours after the onset in all patients; parent artery occlusion was performed in 26 and intra-aneurysmal coil embolization was performed in 5. The median maximum aneurysm diameter was 5.4 mm (IQR, 4.9–6.7 mm), median length of dissecting artery was 9.2 mm (IQR, 6.8–13.9 mm), and median occluded length of artery was 14.3 mm (IQR, 8.4–18.3 mm).

Unilateral or bilateral abducens nerve palsy was observed in 11 (35.5%) of the 31 patients. The clinical characteristics were compared between the palsy (+) and palsy (−) groups (**Table 1**). The WFNS grade at the time of arrival was IV or V in 9 (81.8%) in the palsy (+) group and 8 (40%) in the palsy (−) group, being significantly more frequent in the palsy (+) group ($p = 0.025$). The H&H grade was IV or V in 8 (72.7%) in the palsy (+) group and 7 (35%) in the palsy

Table 1 Characteristics of ruptured vertebral artery dissection aneurysm cases with or without abducens nerve palsy

Variable	Abducens nerve palsy		p value
	(+)	(–)	
Number of patients	11	20	
Age (mean ± SD)	53 ± 6.5	51.5 ± 11.7	0.66
Male	9 (81.8%)	15 (75.0%)	0.62
WFNS grade: IV, V	9 (81.8%)	8 (40.0%)	0.025
Hunt & Hess grade: IV, V	8 (72.7%)	7 (35.0%)	0.044
Fisher group: III	11 (100%)	18 (90.0%)	0.28
Acute hydrocephalus	4 (36.4%)	8 (40.0%)	0.84
EVD installation	3 (27.3%)	7 (35.0%)	0.66
SAH thickness of prepontine cistern (median, IQR) (mm)	7.0, 5.7-10.3	5.05, 3.6-7.9	0.19
Location			
Right side	8 (72.7%)	14 (70.0%)	0.87
Distal to PICA	6 (54.5%)	7 (35.0%)	0.29
PICA involved	1 (9.10%)	7 (35.0%)	0.11
Proximal to PICA	4 (36.3%)	6 (30.0%)	0.72
Treatment modality			
Coil embolization for cerebral aneurysm	1 (9.10%)	4 (20%)	0.46
Parent artery occlusion	10 (90.1%)	16 (80%)	
Maximum diameter of aneurysm (median, IQR) (mm)	5.6 (4.6–6.0)	6.0 (5.1–6.8)	0.17
Lesion length (median, IQR) (mm)	9.7 (7.0–12)	10.2 (6.3–13.8)	0.59
Occulusion length (median, IQR) (mm)	12.5 (8.4–16.8)	14.2 (9.4–18.6)	0.51
Symptomatic vasospasm	1 (9.10%)	3 (15.0%)	0.64
mRS			
0	0	12 (60.0%)	
1	6 (54.5%)	3 (15.0%)	
2	2 (18.2%)	2 (10.0%)	
3	3 (27.3%)	3 (15.0%)	

EVD: external ventricular drainage; IQR: interquartile range; mRS: modified Rankin Scale; NA: not available; PICA: posterior inferior cerebellar artery; SAH: subarachnoid hemorrhage; SD: xxxxx xxxxx; WFNS: World Federation of Neurological Surgeons

(−) group, being significantly more frequent in the palsy (+) group (p = 0.044). All patients in the palsy (+) group had diffuse SAH and the median thickness of the hematoma was 7.0 mm (IQR, 5.7–10.3 mm). Acute hydrocephalus was observed at the time of arrival in 4 (36.4%), 3 (27.3%) of whom underwent ventricular drainage. Symptomatic vasospasm was seen in one patient (9.1%). No significant difference was observed in the frequency of these conditions compared with the palsy (−) group. Although all patients underwent endovascular surgery, no significant difference was observed in the location of the aneurysm, maximum diameter, length of dissecting lesion, occluded length of artery, or the presence of hemorrhagic or ischemic complications during treatment between the two groups.

The details of the 11 patients in the palsy (+) group, including changes in symptoms, are shown in **Table 2**. The WFNS grade at the time of admission was II in 2 (18.2%), IV in 2 (18.2%), and V in 7 (63.6%), and the H&H grade was II in 3 (27.3%), IV in 6 (54.5%), and V in 2 (18.2%). Abducens nerve palsy was unilateral in 3 (27.3%) and bilateral in 7 (63.6%), and palsy was ipsilateral to rupture in all 3 patients with unilateral palsy. In 1 patient, the side of palsy was not mentioned in the chart, and follow-up after discharge was not possible. The median time of confirmation of abducens nerve palsy was 3 days (IQR, 2–5.5 days) after treatment. In the palsy (+) group, the mRS score was 1 in 6 (54.5%), 2 in 2 (18.2%), and 3 in 3 (27.3%). The median follow-up period was 24 months (IQR 5.5–51 months). Recovery from abducens nerve palsy was observed in 3 (27.3%), improvements were observed in 5 (45.5%), and symptoms remained in 2 (18.2%). In the 3 patients with recovery, the time until recovery was 7–180 days after the onset of palsy. Severe diplopia was observed in 3 (27.3%) of the 11 patients, and they were followed up by an ophthalmologist using the Hess screen test and 1 (9.1%) patient underwent strabismus correction surgery 50 days after the onset. None of the patients were treated medically for abducens nerve palsy. In consideration of the aneurysm morphology and hematoma volume at the time of endovascular surgery, the maximum aneurysm diameter, length of dissecting lesion, occluded length of artery, and SAH hematoma volume were compared between the 3 (27.3%) with

Table 2 Characteristics of patients with abducens nerve palsy

Case No.	Age/sex	Location	Side	WFNS grade	H&H grade	Fisher group	Acute hydrocephalus	Site of paresis	Outcome of paresis	Time of recovery (day)	Follow-up period (month)	mRS
1	50/F	Distal to PICA	Right	V	V	III	None	Bilateral	Recovered	60	31	1
2	51/M	Distal to PICA	Right	V	IV	III	Yes	Bilateral	Improve	NA	94	1
3	46/M	Distal to PICA	Right	IV	IV	III	None	Bilateral	Improve	NA	72	1
4	55/M	Proximal to PICA	Right	V	IV	III	None	Bilateral	Improve	NA	2	2
5	55/M	Distal to PICA	Right	V	IV	III	None	Bilateral	Remain	NA	0	2
6	40/M	Proximal to PICA	Right	V	IV	III	Yes	Bilateral	Recovered	180	66	3
7	50/M	Distal to PICA	Left	V	V	III	None	Left	Improve	NA	24	1
8	53/M	Proximal to PICA	Right	II	II	III	None	Right	Recovered	7	36	3
9	62/F	Proximal to PICA	Left	V	IV	III	None	Bilateral	Improve	NA	24	1
10	64/M	Distal to PICA	Right	II	II	III	Yes	Right	Remain	NA	9	3
11	57/M	PICA involved	Left	IV	II	III	Yes	NA	NA	NA	2	1

F: female; H&H: Hunt & Hess; M: male; mRS: modified Rankin Scale; NA: not available; PICA: posterior inferior cerebellar artery; WFNS: World Federation of Neurological Surgeons

complete recovery and the 7 (63.6%) without, but there were no significant differences. In consideration of the method for perioperative intracranial pressure control, the presence of ventricular drainage, period of ventricular drainage, whether lumbar spinal fluid drainage was performed, and period of lumbar spinal drainage were reviewed. In the palsy (+) group, ventricular drainage was performed in 3 (27.3%) for a median of 9 days (IQR, 7–12 days) and lumbar spinal drainage was performed in 4 (36.4%) for a median of 5 days (IQR, 3.8–11.5 days). One patient (9.1%) was managed by ventricular drainage alone, 2 (18.2%) were managed by lumbar spinal fluid drainage alone, and 2 (18.2%) were first managed by ventricular drainage and then by lumbar spinal fluid drainage. None of the 5 (45.5%) patients who underwent ventricular or lumbar spinal drainage had a complete recovery from abducens nerve palsy (**Table 3**). As cranial nerve symptoms other than abducens nerve palsy, 1 patient (9.1%) exhibited facial nerve palsy, and 1 (9.1%) developed hoarseness and dysphagia due to Wallenberg syndrome.

Discussion

Of the 31 VADA patients with mRS ≤3 at the time of discharge evaluated in this study, 11 (35.5%) developed abducens nerve palsy. In the palsy (+) group, the WFNS grade and H&H grade at the time of arrival were significantly higher than those in the palsy (−) group. This suggests that patients with a more severe condition at the time of onset are more likely to develop abducens nerve palsy. No significant difference was observed in the prepontine cistern hematoma thickness, complication by acute hydrocephalus, or implementation of ventricular drainage, and they were not related to the presence of local hematoma or acute hydrocephalus. Recovery from abducens nerve palsy was observed in 3 patients (27.3%) in the palsy (+) group; however, the palsy persisted in 7 (63.6%), and abducens nerve palsy was considered to be one of the sequelae requiring long-term follow-up even in patients with a favorable outcome. No significant difference was observed in any of the factors examined between those with recovery and those in whom paresis persisted.

The incidence of abducens nerve palsy that complicates VADA causing SAH was reported to be 26.9–50.0%,[2,5] but the abducens nerve palsy cannot be accurately evaluated in many patients because patients with a poor outcome cannot respond to verbal commands and examination of ocular motility is difficult.[2,5] In addition, patients in whom abducens nerve palsy poses problems in daily living after treatment are usually mild cases in which walking is possible,

Table 3 Lesion properties and treatment modality in abducens nerve palsy cases

Case No.	Maximum diameter (mm)	Lesion length (mm)	Occlusion length (mm)	SAH thickness (mm)	Treatment modality	EVD installation	Duration of EVD (day)	SPD installation	Duration of SPD (day)	Intra-procedural rupture	Intra-procedural thrombosis
1	4.5	6.7	5.9	6	PAO	Not performed	NA	Not performed	NA	None	None
2	7.1	9.1	5.8	5.9	PAO	Performed	9	Performed	4	None	None
3	4.7	6.8	13.6	8.8	PAO	Not performed	NA	Performed	6	None	None
4	5.4	7.5	8.5	10.3	PAO	Not performed	NA	Not performed	NA	None	None
5	5.2	7.2	9.3	10.4	PAO	Not performed	NA	Performed	28	None	None
6	4.1	3.5	8.3	10.3	PAO	Not performed	NA	Not performed	NA	None	None
7	10.7	15	20	1.1	PAO	Not performed	NA	Not performed	NA	None	None
8	5.1	8.4	23.7	12.7	PAO	Not performed	NA	Not performed	NA	None	None
9	5	19.4	17	4.5	CE	Not performed	NA	Not performed	NA	None	None
10	6.6	8.5	9.2	7	PAO	Performed	15	Not performed	NA	None	None
11	3.2	15	16.5	5.5	PAO	Performed	5	Performed	3	None	None

CE: coil embolization; EVD: external ventricular drainage; NA: not available; PAO: parent artery occlusion; PICA: posterior inferior cerebellar artery; SAH: subarachnoid hemorrhage; SPD: spinal drainage

and accurate evaluation is possible in such cases. For that reason, patients with mRS ≤3 were thus evaluated as the subjects in this study. The incidence of abducens nerve palsy was 35.5%, which was consistent with previously reported figures, but the incidence may increase further if patients in a more severe condition are included.

The intracranial pressure often increases with a higher WFNS grade and H&H grade at the time of arrival,[7] and abducens nerve palsy was reported to be more likely in patients with a higher intracranial pressure.[8,9] Moreover, Zhang et al. hypothesized that the abducens nerve is likely to receive mechanical pressure with changes in the intracranial pressure by his own autopsy findings, it is because the nerve has a long course and is surrounded by a lot of structures such as the cerebellar tentorium, ligaments, and microvessels.[10] Mechanical compression of hematoma in the prepontine cistern[11] and false localizing signs due to acute hydrocephalus were reported to be involved in abducens nerve palsy,[2] but in this study, no relationship was observed between the SAH thickness in the prepontine cistern and the presence of abducens nerve palsy. As the prepontine cistern has wide individual variation, and comparison between before and after bleeding is impossible due to the lack of information before the onset, measurement of the cistern after the onset alone may not reflect the condition. In this study, a significant difference was observed only in the WFNS grade at the time of arrival, and abducens nerve palsy was more likely with a higher posterior cranial fossa or intracranial pressure. Abducens nerve palsy being bilateral in 7 of the 11 patients also suggests that the pressure on the entire posterior cranial fossa rather than direct compression by hematoma exerted a greater effect. There have been reports of the occurrence of abducens nerve palsy despite the absence of hematoma in the prepontine cistern or acute hydrocephalus.[10] Therefore, the intracranial pressure may increase at the onset of SAH through the involvement of brain edema and dysfunction of cerebral perfusion, in addition to hematoma and acute hydrocephalus in a complicated manner,[12] leading to abducens nerve palsy.

Regarding the course of abducens nerve palsy, complete recovery was observed in 3 (27.3%) and required 7–180 days. Improvements were observed in 5 (45.5%) during a mean observation period of 43.2 months and exacerbation was not observed during the observation period. The time of the diagnosis of abducens nerve palsy and the time needed until complete recovery were comparable with those in previous reports.[2,5] On the other hand, although

complete recovery from abducens nerve palsy might be expected in 71–89% of patients,[2,3] complete recovery was observed in only 3 (27.3%) in this study. Although the comparison is difficult because of the absence of reports of the long-term outcome of abducens nerve palsy exclusively in VADA patients with a favorable outcome, the small number of patients with complete recovery in this study might be partly explained by the possibility of detailed questioning about diplopia in all patients, which enabled us to evaluate the presence of abducens nerve palsy more carefully. In addition, symptoms often persisted for years in patients who demonstrated improvement. Abducens nerve palsy is often regarded as a relatively mild complication of SAH, and 8 (72.7%) of the 11 patients in this study were followed up by neurosurgeons. As suggested by this study, abducens nerve palsy is a relatively common complication and severe cases can be indications for strabismus surgery. Indeed, in this study, one patient (9.1%) exhibited no improvement in diplopia and underwent strabismus surgery by an ophthalmologist. As their gait recovered and they were able to resume work, consultation with an ophthalmologist is recommended if severe abducens nerve palsy persists with little improvement. Diplopia may cause difficulties in rehabilitation and daily life after discharge; therefore, it is also recommended to design nursing and rehabilitation including ophthalmological intervention in consideration of diplopia.

There have been no reports of the incidence or improvement rate of abducens nerve palsy according to the treatment, and no reports on abducens nerve palsy as a complication of craniotomy were available. Although craniotomy is considered to involve the risk of nerve injury associated with brain traction, there is the possibility of removal of hematoma during the procedure and relatively early accomplishment of decompression of the posterior cranial fossa. However, Moteki et al. evaluated risk factors of eye movement disturbance as a complication of SAH by multivariate analysis and reported that VADA itself is an independent risk factor even though the selection of endovascular surgery is not.[13] Toyama et al. considered it difficult to expose the prepontine cistern by craniotomy, and a combination of ventricular drainage and lumbar spinal drainage is more effective for controlling the posterior cranial fossa pressure.[14] In this study, ventricular drainage or lumbar spinal drainage was not considered to have led to the improvements in abducens nerve palsy, but further studies about related matters, including their timing, are considered necessary.

Regarding limitations of this study, this was a retrospective study conducted at two institutions. Moreover, no early ophthalmological examination or intervention was performed for any patient to make an accurate evaluation. The further accumulation of cases is considered necessary for comparison of groups with and without complete recovery of abducens nerve palsy.

Conclusion

Of the VADA patients with hemorrhagic onset who received endovascular treatment in whom the mRS score at the time of discharge was 0–3, abducens nerve palsy was observed in 11 (35.5%) and paresis persisted in 7 (63.6%). A characteristic of patients who developed abducens nerve palsy as a complication was a poor neurological condition at the time of arrival. Although the therapeutic results of SAH are improving, abducens nerve palsy is a sequela affecting daily activities, and it requires appropriate diagnosis and follow-up.

Disclosure Statement

All authors submitted the self-certification form of conflicts of interest to the Japanese Society for Neuroendovascular Therapy. They have no conflicts of interest to declare concerning this paper.

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