

Case Report

Endovascular treatment for ruptured cervical anterior spinal artery aneurysm caused by occlusive disease of bilateral vertebral arteries: A case report and literature review

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ABSTRACT

A cervical anterior spinal artery (ASA) aneurysm not concomitant with vascular malformations is extremely rare, and is treated by conservative management or direct surgery in most cases. The endovascular treatment approach for these lesions is not well-documented. We present a case of a ruptured flow-related cervical ASA aneurysm due to the occlusive disease of the bilateral vertebral arteries (VA) treated by endovascular therapy. A 77-year-old woman with acute onset of headache and right hemiparesis was transferred to our hospital. Computed tomography revealed subarachnoid hemorrhage thick in the posterior fossa. A cerebral angiogram showed tandem stenosis of the left VA and the occlusion of the right VA at the V4 segment, with an enlarged C4 radiculomedullary artery and a subsequent aneurysm on the ASA. VA angiography revealed retrograde flow through the lesion and ASA axis to posterior circulation, suggesting a flow-related etiology of the aneurysm. We embolized the aneurysm using a flow-directed microcatheter and coils, preserving the ASA axis. Then, we dilated the VA stenosis using stents to prevent aneurysm recurrence by reducing hemodynamic stress on the ASA.

1. Introduction

Anterior spinal artery (ASA) aneurysms are rare lesions that usually presents with spinal cord compression or subarachnoid hemorrhage (SAH). The prevalence rate of SAH due to ASA aneurysm is less than 1%, and most are associated with a spinal vascular malformation [1]. Other etiologies including, connective tissue diseases, trauma, infection, vasculitis, and aortic coarctation, have been reported; however, a flow-related cervical ASA aneurysm without spinal arteriovenous malformation (AVM) or arteriovenous fistula (AVF) is extremely rare [2–5]. Conservative management or direct surgery is commonly chosen as treatment because of its morphological and anatomical characteristics [6,7]. Therefore, the treatment strategy via endovascular approach for this rare lesion is not well known. We report the case of a ruptured ASA aneurysm associated with occlusive lesions of the bilateral vertebral arteries that was treated with endovascular therapy. We also reviewed the literature regarding the anatomical features and treatment of cervical ASA aneurysms.

2. Case presentation

A 77-year-old woman with acute onset of severe headache, nausea, and progressive disturbance of consciousness was transferred to our hospital. She had a past medical history of ischemic stroke, valvular disease, hypertension, and diabetes mellitus. On arrival, her consciousness level was 12 on the Glasgow Coma Scale. Physical examination revealed right hemiparesis. Computed tomographic (CT) scan showed diffuse SAH in the basal cistern predominantly at the craniocervical junction with hydrocephalus (Fig. 1). CT angiography revealed a dilated ASA and a subsequent 3-mm saccular aneurysm at the level of the third cervical vertebrae (C3) (Fig. 1). Cerebral angiography showed hypoplastic bilateral posterior communicating arteries, suggesting poor collateral supply from the anterior circulation to the posterior. Vertebral artery (VA) angiography revealed severe tandem stenosis of the left VA at V4 segment and occlusion of the right VA at the craniocervical junction. The hypertrophied radiculomedullary artery originated from the C4 level of the right VA, leading to the ASA. Retrograde collateral flow to the posterior circulation through the ASA axis was confirmed, which was thought to cause the saccular aneurysm and SAH (Fig. 2).

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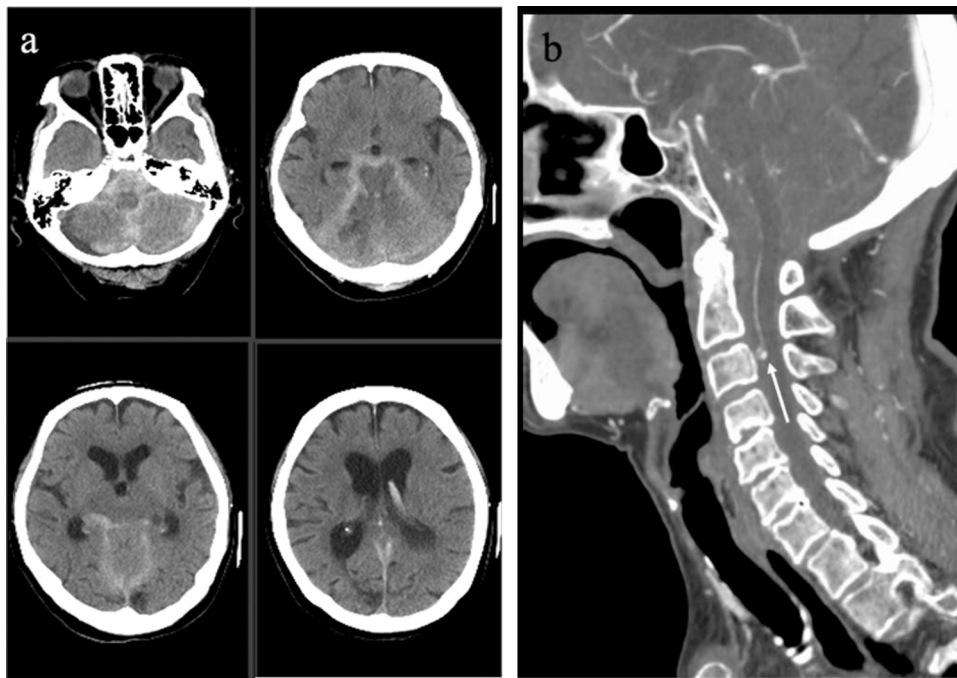


Fig. 1. Computed tomography (CT) shows thick subarachnoid hemorrhage in the posterior fossa (a) and sagittal view of CT angiography shows a saccular aneurysm (arrow) of the anterior spinal artery at C3 level (b).



Fig. 2. Anteroposterior (a) and lateral (b) view of the right vertebral artery (VA) angiography showing the right VA occlusion and an aneurysm (asterisks) on the anterior spinal artery (ASA). The aneurysm is fed by the C4 radiculomedullary artery (arrows). Retrograde collateral flow through the ASA axis to the basilar artery are observed (arrow heads).

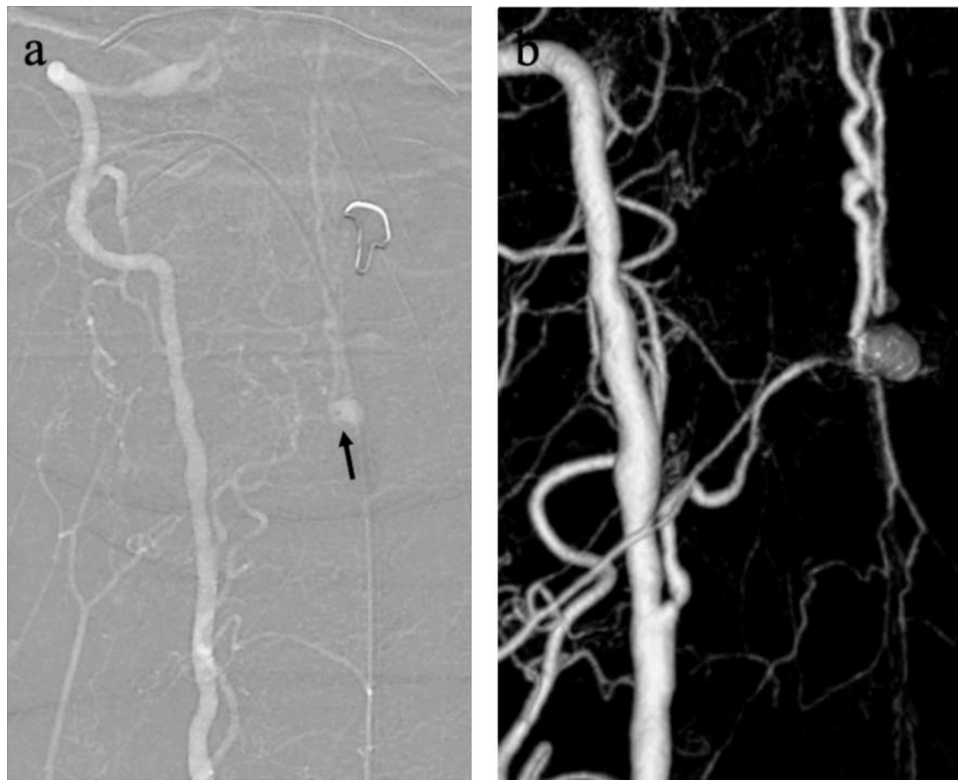


Fig. 3. Pre-coiling anteroposterior view of right VA angiography (a) showing a flow directed microcatheter placed in the aneurysm via C4 radiculomedullary artery (arrow). Post-coiling three-dimensional reconstructed image (b) show the complete obliteration of the aneurysm preserving the ASA axis.

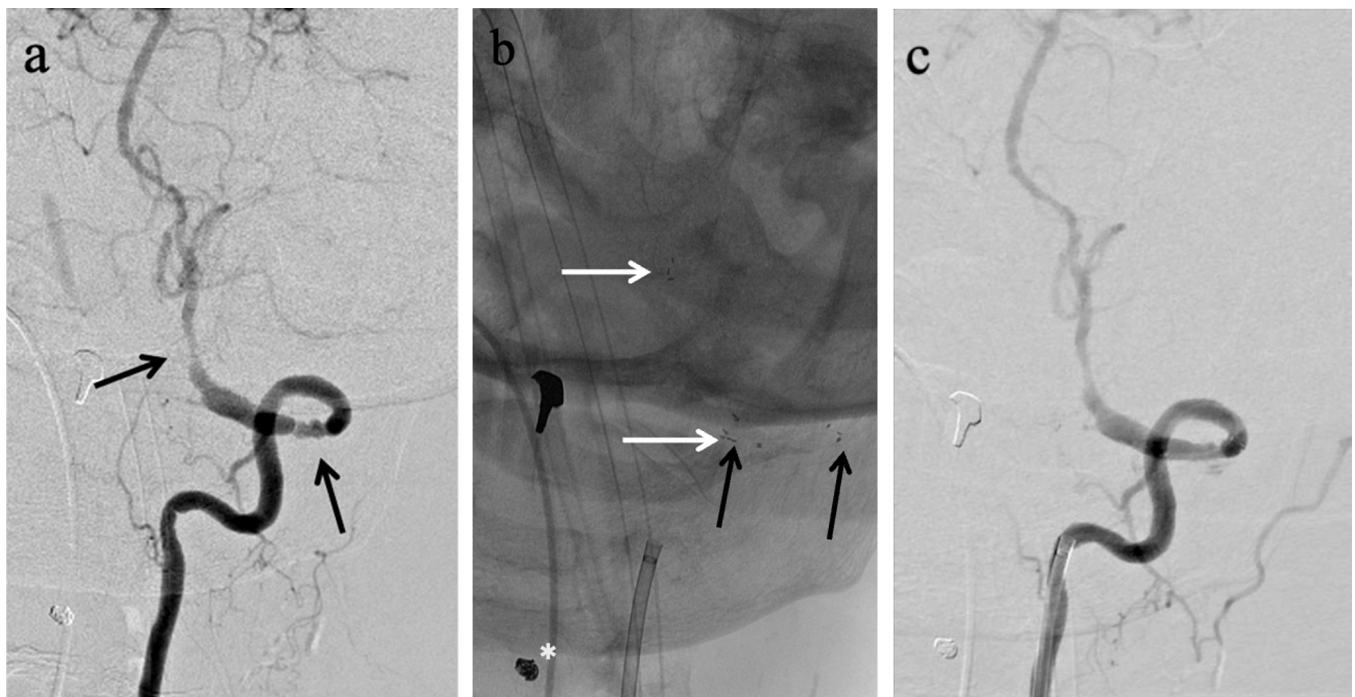


Fig. 4. Pre-treatment anteroposterior view of left VA angiography (a) showing the tandem stenosed VA lesions (arrows). Anteroposterior view of fluorography (b) shows the placed stents (white arrows; Neuroform Atlas 4.0 × 21 mm, black arrows; Wingspan 3.5 × 15 mm). The coil mass placed at the aneurysm is also observed (asterisk). Post-treatment anteroposterior view of left VA angiography (c) showing the dilated lesions.

Endovascular treatment was performed in two stages. First, we embolized the ASA aneurysm to prevent recurrent hemorrhage. A 5-Fr guiding catheter (Fubuki; TOKAI MEDICAL PRODUCTS, Aichi, Japan)

was placed in the right VA via 5-Fr femoral access. We then advanced a 1.5-Fr flow-directed catheter (Marathon; Medtronic, Minneapolis, MN, USA) over a microwire (CHIKAI 10; ASHI INTECC, Aichi, Japan) into the

Table 1
Clinical characteristics of the patients with cervical anterior spinal artery aneurysm without AVF or AVM.

Author & Published year	Age (yrs)	Sex	Location	Presentation	Neurologic finding	Size of aneurysm (mm)	Cause	Treatment	Clinical outcome
Yonas 1980	42	F	Vertebrobasilar junction	SAH	Stiff neck, LOC	4×5×6	Not stated	Conservative management	Died
Thomson 1980	66	F	C1	Compression	Quadriplegia, bladder and bowel dysfunction	Not stated	Isolated	Direct surgery (Clip)	Alive
Vincent 1981	30	F	C1–2	SAH		4	Not stated	Direct surgery (Clip)	Alive
Moore 1982	30	F	C1	SAH	Hemiparesis	Not stated	Isolated	Direct surgery (Clip)	Alive
Kawamura 1999	42	M	C1	SAH	Headache	4×7	Bilateral VA occlusion	Direct surgery (Clip)	Alive
Jiarakongmun 2002	39	M	Cervical	SAH	Quadriparesis and quadriparesis	12×13	Coarctation of aorta	Direct surgery (Clip)	Alive
Walz 2006	58	M	C4	SAH	Headache	11×11	Moyamoya disease	Coil embolization	Alive
Pollock 2009	55	F	C2	SAH	LOC	2	Chiari 1 malformation	Direct surgery (Trap & resection)	Alive
Sharma 2010	36	M	C6	Hematomyelia	Quadriplegia, urinary and fecal incontinence	Not stated	Coarctation of aorta	Direct surgery (Clip)	Alive
Karakama 2010	51	M	C1	SAH	Neck pain, numbness of extremities, consciousness disturbance	2.5×1.3	Dissection	Conservative management	Alive
Wilson 2011	86	M	C1–2	SAH	Neck pain, occipital neuralgia, occasional falls	Not stated	Trauma (odontoid screw placement)	Conservative management	Alive
Takashima 2012	84	M	C1	Hematomyelia	Quadriplegia	Not stated	Isolated	Conservative management	Died
Santana-Ramirez 2013	1	F	C3–5	Compression	Neck pain, quadriparesis	30×18×25	Congenital vessel abnormalities	C3–C6 laminectomy (decompression)	Alive
Yang 2013	47	M	C2	SAH	Neck pain, semicomatose state	0.7×0.5	Not stated	Conservative management	Died
Nagahata 2013	70	F	NA(cervical)	SAH	Not stated	Not stated	Carotid and vertebral rete mirabile	Embolization of parent A.	Alive
			NA(cervical)		Not stated	Not stated	Carotid and vertebral rete mirabile	Embolization of parent A.	
Pahl 2014	43	F	C1–2	SAH	Headache, vomiting, consciousness disturbance	2	Dissection	Conservative management	Alive
Aoun 2014	59	F	C7	SAH	Headache, neck pain	7	Coarctation of aorta	Coil embolization	Alive
Boeris 2015	22	M	C4	Subdural hematoma	Headache, neck pain	3	Isolated, Dissection	Conservative management	Alive
Ashour 2015	72	M	C 2	SAH	Headache, nausea, and somnolence	3	Bilateral VA occlusion	Direct surgery (Clip & wrap)	Alive
Erdi 2016	55	F	C6	IVH	Unconsciousness, tetraparesis	10	Coarctation of aorta	Coil embolization	Alive
Nakhla 2016	88	F	C 5–6	SAH	Headache, neck pain	Not stated	Herniated cervical disc	Conservative management, no treatment	Alive
Singh 2016	38	M	C4	SAH	Headache, neck pain, vomiting	3	Moya moya disease	Conservative management	Not stated
Singh 2017	18	M	C7	SAH	Neck pain, bilateral upper limb weakness	5.6×3.3	Coarctation of aorta	Conservative management, decompressive laminectomy	Alive
Simon-Gabriel 2018	65	M	At the level of the medulla oblongata	SAH	Neck stiffness, drowsiness	Not stated	Not stated	Flow diverter stent at parent artery	Alive
Ren 2018	57	F	CC junction	SAH	Headache	5	Not stated	Direct surgery (Clip & resection)	Alive
Dabus 2018	60 s	NA	Cervicomedullary junction	SAH	Headache, neck pain	Not stated	Dissection	Conservative management	Alive
Roka 2019	30	F	Posterior fossa	IVH	Headache, vomiting, vertigo, gait abnormality	2	Isolated	Conservative management	Alive
Turrini 2020	64	M	C1	SAH	Headache, vomiting, dizziness, numbness	Not stated	Dissection	Direct surgery (resection)	Alive
Abdalkader 2020	70 s	M	C2	SAH	Headache, vomiting	3×2.5	Bilateral VA occlusion	Direct surgery (Clip)	Alive
	50 s	M	C2–3	SAH	Headache, neck pain, vomiting	5×2.5	Not stated	Conservative management	Alive

Abbreviations: CC, craniocervical; SAH, subarachnoid hemorrhage; IVH, intraventricular hemorrhage; LOC, loss of consciousness; VA, vertebral artery

C4 radiculomedullary artery. The microcatheter was navigated and wedged into the aneurysm (Fig. 3), which was embolized with four platinum coils (ED coil; KANEKA MEDICS, Osaka, Japan). Control angiography showed complete obliteration of the aneurysm (Fig. 3).

The second endovascular treatment was performed 14 days after the initial treatment to prevent the aneurysm recurrence due to the increased flow through the VA, in which we performed balloon angioplasty and stent placement for the tandem VA stenosis. A 6-Fr guiding catheter (Fubuki; TOKAI MEDICAL PRODUCTS, Aichi, Japan) was placed in the left VA, and a microcatheter (Excelsior SL-10; Stryker Neurovascular, Fremont, CA, USA) was navigated over a 0.014-inch microwire (Traxcess14; MicroVenton, Inc., Tustin, CA, USA) into the left VA passing through the stenosis segment. A Gateway 3.5 × 20 (Stryker Neurovascular, Fremont, CA, USA) balloon catheter was used to perform percutaneous transluminal angioplasty, and stenting was performed using a Neuroform Atlas 4.0 × 21 mm stent (Stryker Neurovascular, Fremont, CA, USA) along with a Wingspan 3.5 × 15 mm stent (Stryker Neurovascular, Fremont, CA, USA) (Fig. 4). Post-treatment angiography showed an expanded VA with improved flow. We also confirmed persistent occlusion of the ASA aneurysm. All procedures were performed without complications. After surgery for placement of a ventriculoperitoneal shunt, the patient was transferred to a rehabilitation hospital at the neurologic baseline; her modified Rankin Scale score was 3. The patient's family declined radiographic follow-up for the patient owing to her poor condition; however, she had no recurrent hemorrhage two years after treatment.

3. Discussion

Spinal aneurysms are thought to be secondary to traumatic, hemodynamic, and inflammatory stress on the arterial wall and are classified as type 1, i.e., spinal aneurysms associated with AVMs, and type 2, i.e., isolated aneurysms [8]. Type 2 ASA aneurysms are exceedingly rare [9]. We reviewed 29 case reports and 30 patients with cervical ASA aneurysms that were not associated with AVM or AVF (Table 1) [2,4,6,7,9–33]. Fifteen cases had dissecting etiology or no underlying diseases. The aneurysm in most cases was at the upper cervical spine, and the two cases of unruptured aneurysm presented with spinal cord compression that caused quadriplegia. All cases of ruptured aneurysm presented with SAH, except for two cases with hematomyelia, two cases with intraventricular hemorrhage, and one case with subdural hematoma. The mean age was 49.9, with a slight male predominance ($n = 16, 55\%$). The aneurysms were mainly fusiform shape or pseudoaneurysms. In our case, flow-related etiology was assumed based on the angioarchitecture; chronic occlusive disease of the bilateral VAs seemed to demand the collateral flow through the ASA axis to the posterior circulation. The right C4 radiculomedullary artery and the ascending limb of the ASA were larger than descending limb of the ASA, suggesting compensatory enlargement. This etiology led to our treatment strategy.

The wait-and-see management or hematoma evacuation is usually chosen as treatment for a ruptured aneurysm, without treating the lesion. An ASA aneurysm is located on the ventral side of the spinal cord, which makes it technically challenging to access by surgery. In most cases, conservative management was performed because of compromised patient condition, surgical morbidity, and approach-related problems. The possibility of the spontaneous resolution of the ASA aneurysm without any intervention has been suggested; however, in some cases, acute enlargement of the aneurysm was observed within a few weeks after onset [6,19,34]. Moreover, the exact historical outcomes of untreated spinal artery aneurysms and the risk of rebleeding are unclear. Therefore, the feasibility of the endovascular treatment should be considered in any case of an ASA aneurysm.

There are few reports in the literature regarding endovascular treatment of ruptured ASA aneurysms. Cobb et al. treated a thoracic ASA aneurysm using the Onyx liquid embolic system, but it was after confirming the spinal cord infarction [35]. Embolization of the aneurysm

using liquid material cannot be validated for all solitary ASA aneurysms because of the high risk of ASA occlusion. Simon-Gabriel et al. treated an ASA aneurysm by placing a flow diverter stent in the vertebral artery across the origin of the radiculomedullary artery; however, further validation is required on the effectiveness and safety of the flow diverter stent in treating a distant lesion without sacrificing the ASA [30].

In the present case, the aneurysm was located at the junction between the radicular component of the radiculomedullary artery and the ASA axis. Thus, while there was increased hemodynamic stress that formed the aneurysm, this location made endovascular treatment feasible. The saccular shape of the aneurysm and the dilated feeding artery facilitated the catheter navigation and coil placement. We could treat the aneurysm with coils preserving the ASA axis, even though the aneurysm had a wide neck. Then, we added stents placement for the VA stenosis in the chronic phase of SAH. Considering the flow-related etiology and rupture of the aneurysm, we thought that decreasing the hemodynamic stress was necessary to prevent the recurrence or de novo formation of the aneurysm. This is a reasonable strategy based on previous literature reporting that the treatment of vascular malformations results in the resolution of the spinal artery aneurysms [34]. Given that spinal artery aneurysms are typically located on the parent artery rather than its branching sites, it is possible to accomplish selective endovascular embolization of ASA aneurysms.

4. Conclusions

We report a case of a ruptured solitary ASA aneurysm caused by a hemodynamic mechanism treated with endovascular approach. Endovascular treatment could be a treatment option for this rare disease when the morphological and anatomical features are favorable.

Ethics approval and consent to participate

The St. Luke's International Hospital Research Ethics Committee (Tokyo, Japan) approved the study protocol. (Research number: 21-R021).

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CRediT authorship contribution statement

Shogo Shima contributed to report design, acquisition and interpretation of the data, and drafting the article. **Shinsuke Sato** contributed to acquisition and interpretation of the data. **Tatsuki Motizuki** contributed to acquisition of the data. **Yasunari Niimi** contributed to interpretation of the data and supervision.

Data availability

The data and material that support the findings of this report are available from the corresponding author.

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None.

Conflicts of interest/Competing interests

The authors have no relevant financial or non-financial interests to disclose.

Consent to participate

Informed consent was obtained from all individual participants included in the study.

Consent for publication

The participant has consented to the submission of the case report to the journal.

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