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Distal Parent Vessel Occlusion of 2 Superior Cerebellar Artery Fusiform Aneurysms: Report of 2 Cases and Literature Review

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Key words
- Aneurysm
- Cerebrovascular
- Fusiform
- Subarachnoid hemorrhage
- Superior cerebellar artery

Abbreviations and Acronyms
CT: Computed tomography
OSH: Outside hospital
S2: Lateral pontomesencephalic segment
SAH: Subarachnoid hemorrhage
SCA: Superior cerebellar artery

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BACKGROUND: **Fusiform superior cerebellar artery (SCA) aneurysms are rare, and their management represents a technical challenge. In complex aneurysms, endovascular parent vessel occlusion of the SCA may be a treatment option. Here, we present 2 cases of fusiform SCA aneurysms, 1 ruptured and 1 unruptured, as well as our institution's management with parent vessel occlusion. We also provide a review of the literature.**

CASES DESCRIPTION: **Case 1: A 42-year-old male was transferred from an outside hospital with subarachnoid hemorrhage. On admission, the patient had a Glasgow Coma Scale score of 8, a Hunt and Hess grade 4, and a Fisher grade 4. A diagnostic angiogram demonstrated a right SCA fusiform lesion with proximal and distal dilatations of 1.45 mm and 5.35 mm long, respectively, likely representing a single dissecting pseudoaneurysm. The distal dilatation was coiled, resulting in parent vessel occlusion. The patient recovered clinically and was discharged in stable condition.**
Case 2: A 27-year-old female was transferred from an outside hospital due to a brainstem stroke. A diagnostic angiogram revealed an S2/S3 segment left SCA fusiform lesion, likely representing a dissecting aneurysm. The patient was neurologically intact at admission and managed conservatively. At the 2-month follow-up angiogram, the dissection had extended along the length of the SCA. Consequently, the patient underwent coil embolization of the distal left SCA. At the 6-month follow-up, the vessel remained obliterated and the patient's neurologic status had improved.

CONCLUSIONS: **Endovascular coil embolization of fusiform SCA aneurysms offers a reasonable and safe treatment approach.**

INTRODUCTION

Superior cerebellar artery (SCA) aneurysms of fusiform morphology are uncommonly encountered by neurosurgeons and neurointerventionalists.[1-3] These can be treated with either open cerebrovascular surgery or endovascular techniques, but their management is more challenging than saccular aneurysms.[1,4-6] Often, these aneurysms rupture, leading to subarachnoid hemorrhage (SAH). Endovascular parent vessel occlusion of the SCA can be a reasonable treatment option if adequate collateral circulation is present, if the aneurysm is located distally, and if the lesion is located within the nondominant circulation.[7,8] Few studies have described the use of coil embolization for the purpose of parent vessel sacrifice. Here, we describe the endovascular management of 2 patients with fusiform SCA aneurysms as a result of probable dissections. In both cases, the lesions were treated with distal parent vessel occlusion. We also provide a review of the literature of all previously published studies on SCA fusiform aneurysms.[5-22]

Case 1

Initial Presentation. A 42-year-old male nonsmoker with a medical history significant for paraplegia secondary to a spinal gunshot wound, osteomyelitis secondary to a chronic coccygeal wound, and a longstanding ostomy presented to an outside hospital (OSH) with a severe headache. A head computed tomography (CT) scan was performed and read as being normal; the patient was subsequently discharged home. Several hours later, the patient became unresponsive and returned to the OSH. The patient was intubated and underwent a second head CT that revealed SAH. Consequently, he was transferred to our institution for definitive management. The Glasgow Coma Scale score was 8, and the Hunt and Hess grade was 4. The SAH was a Fisher grade of 4, and the World Federation of Neurological Surgeons grade was 4. Imaging (CT head/CT angiography of the head and neck) performed at our institution revealed extensive SAH with intraventricular extension (Figure 1A). Multiple dissections throughout the extracranial carotid and vertebral arteries

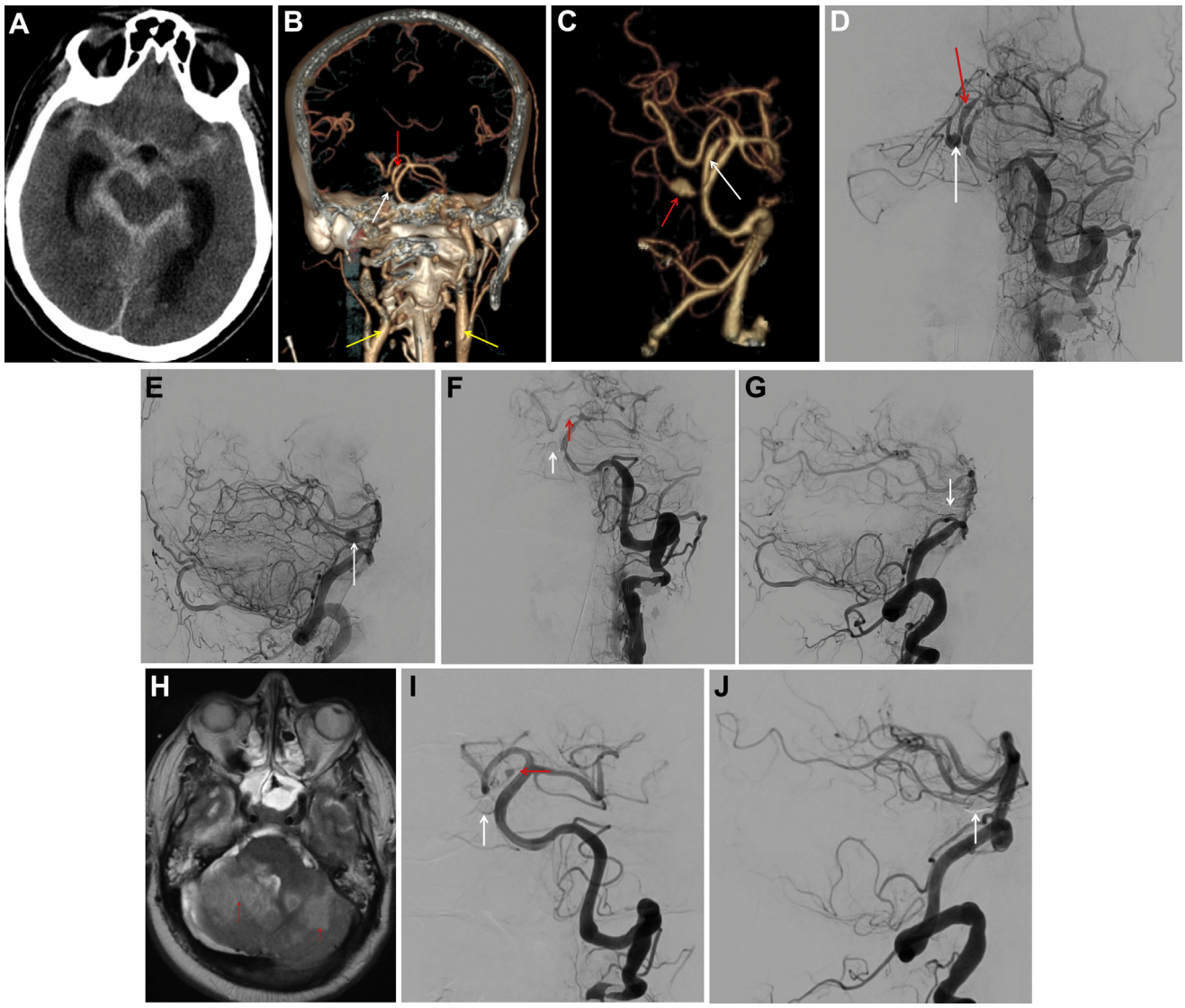

Figure 1. Admission computed tomography (CT) shows subarachnoid hemorrhage around the basilar cisterns (**A**). Coronal (**B**) projection of head and neck CT angiography showing bilateral extracranial carotid dissection (*yellow arrows*) and the distal (*white arrow*) and proximal (*red arrow*) portion of a fusiform right superior cerebellar artery aneurysm. A 3-dimensional reconstruction of basilar artery CT angiography (**C**) shows the distal (*white arrow*) and proximal (*red arrow*) portion of the pseudoaneurysm. Anteroposterior (AP) (**D**) and lateral (**E**) projections from the preprocedure angiogram shows the distal (*white arrow*) and proximal (*red arrow*) portions of the aneurysm. AP (**F**) and lateral (**G**) projections from the postprocedure angiogram shows coil embolization and occlusion of the distal portion of the aneurysm (*white arrow*) and blood flow reduction to the proximal portion of the aneurysm (*red arrow*). Bilateral superior cerebellar infarcts (*red arrows*) observed on brain magnetic resonance imaging 8 days after treatment (**H**). A 2-months' follow-up, angiogram revealed complete obliteration of the distal portion of the aneurysm on AP (**I**) and lateral (**J**) projections (*white arrow*), as well as the parent vessel with persistent flow to the proximal portion of the aneurysm (*red arrow*).

were present along with apparent aneurysmal dilatations of the right SCA, suggesting the presence of pseudoaneurysms (Figure 1B and C). A right external ventricular drain was inserted in the emergency department.

A diagnostic angiogram confirmed the presence of multiple pseudoaneurysms in the cervical segments of the internal carotid arteries, bilaterally. In the posterior circulation, multiple dissection points with several pseudoaneurysms were present in the left V2 and V3 segments of the vertebral artery. The basilar artery, similarly, had a beaded appearance. Two dilatations were present within the right SCA. The proximal dilatation measured 1.45 mm long and was located in the anterior pontomesencephalic segment, and the distal dilatation measured 5.35 mm long and was located in the lateral pontomesencephalic segment (S2) (Figure 1D and E). This entire lesion, we believe, likely represented a single

dissecting pseudoaneurysm. Because the dissection was the most likely reason for SAH development, there was a high risk of rupture, so the decision was to coil embolize its distal portion.

Treatment. Informed consent for the procedure was obtained. The right SCA was cannulated with an Excelsior SL-10 microcatheter (Stryker Neurovascular, Freemont, California, USA) over a Transend EX Soft Tip Microwire (Stryker Neurovascular, Freemont, California, USA). The microcatheter was advanced beyond the proximal dilatation. The distal dilatation was coiled with 5 Target Helical Ultra coils (Stryker Neurovascular), leading to obliteration of the distal SCA, flow reduction within the proximal SCA, and decreased size of the pseudoaneurysm (Figure 1F and G) on posttreatment angiograms. There were no perioperative complications. An immediate posttreatment head CT was performed, and no new infarctions were observed.

Hospital Course and Follow-Up. A head CT/CT angiography was performed 2 days after treatment, which identified vasospasm, managed with milrinone and permissive hypertension, and new hypodensities suggestive of acute infarction on both cerebellar hemispheres (larger on the left side) and brainstem. This was also seen on a brain magnetic resonance imaging 5 days after treatment (Figure 1H). He also developed aspiration pneumonia and communicating hydrocephalus for which he underwent ventriculoperitoneal shunt placement. The patient was discharged in stable condition and was able to move the upper extremities. At the 2-month follow-up, a diagnostic angiogram revealed complete obliteration of the distal portion of the SCA and a significant reduction in the size of the pseudoaneurysm (Figure 1I and J). Clinically, the patient's strength in the upper extremities improved and he was able to follow simple commands.

Case 2

Initial Presentation. A 27-year-old female with no significant medical history presented to an OSH with a headache for the past 2 months, as well as acute onset of dizziness and nausea that improved while sitting and worsened while standing. She underwent a head CT scan, which was read as unremarkable, and was soon discharged home. Several hours later, she presented to the OSH with numbness in the right arm, leg, and face. The previous head CT scan was reread, and a posterior fossa hyperdensity involving the left ventrolateral pons and left prepontine cistern was identified. She was transferred to our institution for definitive treatment (Figure 2A and B).

On admission, the Glasgow Coma Scale was 15 and the rest of the physical examination was nonfocal. A diagnostic angiogram revealed a left SCA fusiform lesion, likely secondary to dissection along the S2 and the cerebellomesoencephalic segments, which measured 2.5 cm long (see Figure 2C–E). The hospital course was uneventful apart from an episode of supraventricular tachycardia that was managed medically. Besides the patient's favorable clinical status, definitive treatment options were widely discussed with our cerebrovascular group and other cerebrovascular groups from other institutions. Because there was no acute hemorrhage, the patient was neurologically stable, and it was determined that the risks of treatment were higher than the benefits, we decided to pursue conservative management. She was discharged in stable condition on 325 mg aspirin, and close follow-up was indicated. At 2-month radiographic follow-up angiography, an extension of the left SCA dissection to the anterior pontomesencephalic segment and cortical segments of the SCA was observed (see Figure 2F and G). While the patient remained neurologically intact, the decision to admit for endovascular treatment was made.

Treatment. Informed consent for the procedure was obtained. The proximal left SCA was cannulated with an Excelsior SL-10 microcatheter (Stryker Neurovascular) over a Synchro2 soft microwire (Stryker Neurovascular). The Synchro2 soft microwire was removed, and an Asahi 0.010 microwire (Asahi Intecc USA, Burlington, Massachusetts, USA) was used to cannulate the distal aspect of the dissecting pseudoaneurysm. Six Target 360 ultracoils (Stryker Neurovascular) were deployed in a distal-to-proximal sequential fashion. At the end of the procedure, the proximal left SCA was patent and complete obliteration of the distal left SCA was observed. There were no periprocedural complications (see Figure 2H and I).

Hospital Course and Follow-Up. The patient's hospital course was uneventful, apart from a transient episode of numbness on the right side of the body 4 days after the procedure. Magnetic resonance imaging/angiography revealed areas of small acute infarcts involving the left pons and the left superior cerebellum (see Figure 2J). The symptoms subsided, and the patient was neurologically intact at the time of discharge. At the 3-week clinical follow-up, the patient complained of a mild left-sided headache and numbness on the right side of the body. The neurological examination, however, was nonfocal. At 6-month angiographic follow-up, persistent occlusion of the distal SCA was observed (see Figure 2K and L).

DISCUSSION

Intracranial fusiform aneurysms are rare, representing 3%–13% of all intracranial aneurysms.[2] Fusiform SCA aneurysms, however, are even less frequently encountered. Currently, in the neurosurgical literature, there are 26 published cases of fusiform SCA aneurysms (Table 1). Most reports describe the use of either traditional microsurgical techniques or endovascular approaches in the management of these lesions.[1,18,23] These reports have demonstrated that clinical outcomes are comparable between different microsurgical modalities, but aneurysm recurrence and complication rates were higher in comparison with endovascular-based approaches.[1,3] Thus endovascular treatment has become the standard approach to these aneurysms over the past decade.[1,3,5,24]

Coil embolization has proven to be safe and effective in treating ruptured saccular aneurysms in the posterior circulation.[3] This modality is less effective in the treatment of wide-necked aneurysms and fusiform aneurysms. In such cases, coil embolization with parent vessel occlusion can be performed.[1,3,4,7,8,23-27] Parent vessel occlusion is typically considered on the basis of select angiographic criteria, such as a broad aneurysmal neck, the presence

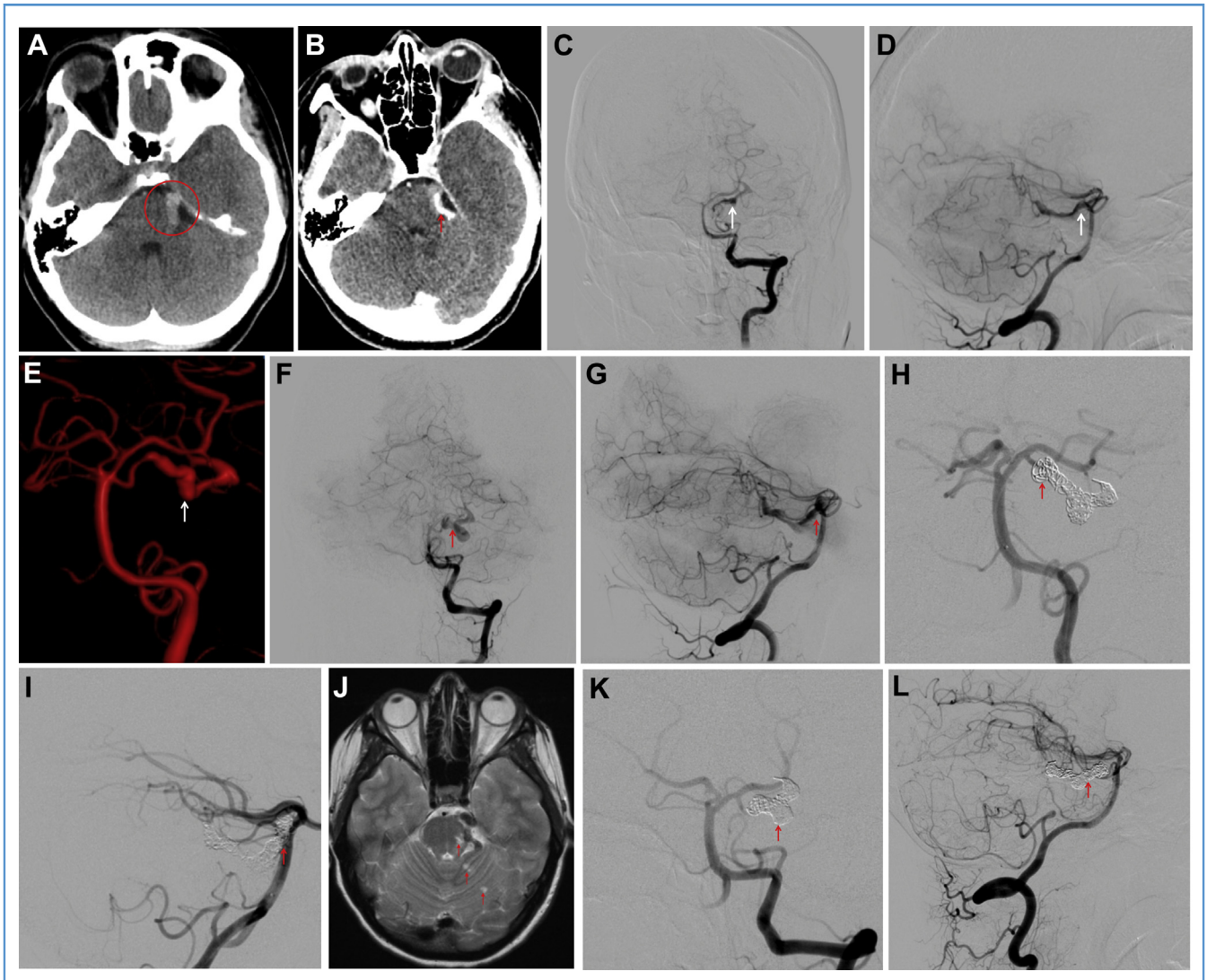

Figure 2. (**A**) Head computed tomography (CT) from an outside hospital revealing a hyperintensity in the left parapontine region of the brainstem (*red circle*). (**B**) Head CT angiography shows ectasia of the perimesencephalic portion of the left superior cerebellar artery (SCA) (*red arrow*). Admission angiogram revealed a dissection along the S2 and cerebellomesoencephalic segment segments (*white arrow*) of the left SCA in anteroposterior (AP) (**C**), lateral (**D**), and virtual reconstruction (**E**). A 2 months' follow-up, an angiogram showed extension of the previously identified dissection to the S1 and cortical segment segments on AP (**F,** *red arrow*) and lateral projections (**G,** *red arrow*). The patient underwent elective endovascular treatment by coiling the distal left SCA and leaving patent the proximal left SCA (**H** and **I,** *red arrow*). A 5 days' posttreatment brain T2 magnetic resonance image shows several punctate areas of diffusion abnormality in the left midbrain and left superior cerebellum suggestive of acute infarction (**J,** *red arrows*). A 6-months' follow-up angiogram revealed complete obliteration of the left SCA (**K** and **L,** *red arrow*).

of distal aneurysms, fusiform and dissecting aneurysm morphology, and good contralateral flow.[1,4] Complications of this procedure include cranial nerve III and IV palsies, cerebellar hemorrhage, and ataxia, but most of these resolve within a few days after the intervention and without long-term neurologic sequelae.[3,7,23,25] Ischemic stroke is also a potential complication; however, there is good collateral circulation among the SCAs, the anterior inferior cerebellar arteries, and posterior inferior cerebellar arteries that may prevent infarction after occlusion.[4] Our cases, however, did have acute infarction post treatment, from which both patients developed few symptoms and showed improvement at the last clinical follow-up. Parent vessel occlusion via the injection of glue or use of intraarterial balloons can be considered, but this is more prone to incomplete aneurysmal obliteration.[24] Four cases of fusiform SCA aneurysms treated via coil embolization have been published.[17,19,21] The first report of a fusiform SCA aneurysm treated with coil embolization and parent vessel occlusion demonstrated complete occlusion at 2-year imaging follow-up without

Table 1. Published Cases of Fusiform Superior Cerebellar Artery (SCA) Aneurysms

Publication and Year	Number of Patients	Number of Aneurysms	Rupture	SCA Segment	Presentation	Management	Clinical Outcome
Kalyan-Raman et al., 1983[9]	1	1	Yes	S1	Cerebellar infarct	Medical	Death
Hirose et al., 1990[10]	1	1	Yes	S1	SAH	Surgery (clipping)	Mild cerebellar signs
Drake et al., 1997[11]	2	2	Cases 1—2: no	Not reported	Cases 1—2: mass effect	Surgery. Case 1: clipping. Case 2: trapping	Case 1: severe disability. Case 2: death
Fukui et al., 1998[12]	1	1	Yes	S3	SAH	Surgery (trapping)	Uneventful
Ikeda et al., 1999[13]	1	1	Yes	S1	SAH	Surgery (clipping/ wrapping)	Uneventful
Sato et al., 1999[14]	1	1	Yes	S4	SAH	Surgery (trapping)	Uneventful
Mizutani et al., 2001[15]	1	1	Yes	Not reported	SAH	Surgery (trapping + bypass)	Not available
Danet et al., 2001[7]	1	1	No	S2	Cerebellar ischemia	Endovascular (parent vessel occlusion)	Uneventful
Araki et al., 2002[16]	1	1	Yes	S1	SAH	Surgery	Vegetative
Gotoh et al., 2003[17]	2	2	Case 1: no. Case 2: yes	Cases 1—2: S1	Case 1: cerebellar infarction Case 2: SAH	Case 1: medical. Case 2: endovascular (coiling)	Cases 1—2: uneventful
Atalay et al., 2007[18]	1	1	Yes	S2	SAH	Surgery (trapping)	Uneventful
Iko et al., 2007[19]	1	1	Yes	S3	SAH	Endovascular (coiling)	Uneventful
Nussbaum et al., 2011[20]	2	2	Cases 1—2: no	Cases 1—2: S2	Incidental	Cases 1—2: surgery (Clipping)	Cases 1—2: uneventful
Raphaeli et al., 2011[8]	4	4	Case 1: no. Case 2: yes. Case 3: no. Case 4: yes	Not reported	Case 1: TIA. Case 2: SAH. Case 3: cerebellar infarct. Case 4: SAH	Endovascular Cases 1—4: parent vessel occlusion	Case 1: cerebellar syndrome. Case 2: poor. Cases 3—4: uneventful
Alurkar et al., 2012[21]	3	3	Yes	Cases 1—3: S4	Case 1: SAH. Case 2: cerebellar hemorrhage Case 3: SAH	Endovascular. Case 1: parent vessel occlusion Cases 2—3: coiling	Cases 1—3: uneventful
Briganti et al., 2013[22]	1	1	Yes	S1	SAH	Endovascular (Pipeline embolization device)	Uneventful
Lamis et al., 2014[6]	1	1	Yes	S1	SAH	Surgery (bypass + trapping)	Uneventful
Kang et al., 2017[5]	1	1	Yes	S4	SAH	Surgery (clipping and trapping)	Uneventful
Present case	1	2	Case 1: yes. Case 2: no	Case 1: S1—S2. Case 2: S1—S4	Case 1: SAH. Case 2: right-side body numbness	Cases 1—2: endovascular (parent vessel occlusion)	Case 1: follow simple commands. Case 2: uneventful

S1, anterior pontomesencephalic segment of the superior cerebellar artery; SAH, subarachnoid hemorrhage; S3, cerebellomesoencephalic segment of the superior cerebellar artery; S4, cortical segment of the superior cerebellar artery; S2, lateral pontomesencephalic segment of the superior cerebellar artery; TIA, transient ischemic attack.

Table 2. Published Cases of Fusiform Superior Cerebellar Artery Aneurysms Stratified by Therapeutic Approach and Outcome

Clinical Outcome	Surgery	Endovascular	Medical	Total
Uneventful	8	10	1	19
Mild-to-moderate	1	2	0	3
Severe-death	3	1	1	5
Not reported	1	0	0	1
Total	13	13	2	28

associated neurologic deficits.[7] These findings were echoed in future reports.

In the present study, the authors decided to perform coil embolization and parent vessel occlusion in the treatment of 2 different dissecting fusiform SCA aneurysms; in both cases, the decision to intervene was predicated on observation of extension of the dissection. The first patient presented with SAH, similar to previously reported cases. Besides his baseline medical conditions, he continued to show neurologic improvement at last clinical follow-up. However, long-term outcomes are still due. The second patient did not present with SAH but presented with cerebellar symptoms that subsided over time. At last clinical follow-up, the patient was neurologically intact. In both patients, their neurologic outcomes at clinical follow-up and the absence of procedure-related complications support the use of this technique for the management of these rare and complex vascular lesions.

In our review, including in our cases, we identified 13 cases managed surgically, 13 cases managed with endovascular approaches, and 2 patients who did not undergo treatment and were managed medically. The outcomes ranged from uneventful, mild-to-moderate disability (cerebellar syndrome, moderate disability, and mild cerebellar signs) to severe (vegetative state, severe disability, poor clinical outcome, or death). Uneventful outcomes were reported in 8 cases who underwent surgery and 10 patients who underwent endovascular treatment. A higher number of severe disability-death outcomes was observed after surgical treatment (Table 2).

Of note, coil embolization of the SCA may raise concerns of occluding perforating and/or distal vessels leading to infarction of adjacent neural tissue. However, in these patients, the dissection itself may have extended and already occluded any critical perforating vessels and collateral vessels to more distal brain tissue. This may help to explain why patients who were treated via parent vessel occlusion did not worsen neurologically in prior reports, as well as in our cohort.

CONCLUSION

Here, we describe the endovascular management of 2 patients with SCA fusiform aneurysms. In both cases, the lesions were successfully treated via endovascular coil embolization in distal-to-proximal fashion and parent vessel occlusion.

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