De novo Vertebral Artery Dissecting Aneurysm after Internal Trapping of the Contralateral Vertebral Artery

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Abstract

We present the case of a *de novo* vertebral artery dissecting aneurysm (VADA) after endovascular trapping of a ruptured VADA on the contralateral side. The first ruptured VADA involved the posterior inferior cerebellar artery, which was successfully treated by endovascular internal trapping using a stent. A follow-up study of 3 months' duration revealed *de novo* VADA on the contralateral side. The second VADA was successfully embolized using coils while normal arterial flow in the vertebral artery was preserved using a stent. Increased hemodynamic stress may cause the development of *de novo* VADA on the contralateral side.

Keywords

vertebral artery dissecting aneurysm, *de novo* aneurysm, hemodynamic stress, sent assisted coil embolization

Introduction

Vertebral artery dissecting aneurysm (VADA) with a hemorrhagic presentation has a high rate of re-bleeding and mortality [10]. Immediate treatment is necessary to avoid lethal re-bleeding. Endovascular internal trapping is currently the first choice for treating ruptured VADAs [13, 14]. However, VADA treatment involving the origin of the posterior inferior cerebellar artery (PICA) is still controversial because it is difficult to preserve the PICA via internal trapping. A new endovascular technique using a stent was developed to overcome this problem [6, 9, 15]. Another problem related to internal trapping is hemodynamic changes in the cerebral circulation, which may cause another dissection on the contralateral side. We present the case of a patient with a *de novo* VADA after endovascular trapping of a ruptured PICA involved VADA on the contralateral vertebral artery (VA).

Case Report

A 55-year-old woman presented with a disturbance of consciousness. She complained of sudden occipitalgia on the left side one week before the examination. She had no family history and past history, including connective tissue disorders. Computed tomography (CT) revealed a subarachnoid hemorrhage (Fig. 1a). 3-dimentional CT angiography demonstrated a dilatation and narrowing of the left VA, indicating a VADA (Fig. 1b). The patient was diagnosed as having World Federation of Neurosurgical Societies grade IV subarachnoid hemorrhage caused by a ruptured left VADA. She was sedated and intubated, and an emergency cerebral digital subtraction angiography was performed. A vertebral angiography (VAG) showed a left VADA that included the PICA origin (Fig. 2). The diameter of both VAs was similar (right 3.5 mm, left 3.3 mm). The diameter of the left PICA was 1.4 mm and it perfused a wide area of the cerebellum. We decided to treat the patient using endovascular internal trapping while preserving the PICA. Triple antiplatelet therapy was administered using 300 mg aspirin, 300 mg of clopidogrel, and 100 mg of cilostazol, and their function was confirmed using the Verify Now Platelet Function Assay (Accumetrics, San Diego, CA). Under general anesthesia, Headway17 (MicroVention, Tustin, CA, USA) microcatheter was threaded into the left PICA from the

right VA via the VA union and the left VA in a retrograde fashion to place the stent. Another microcatheter (Excelsior SL-10; Boston Scientific, Fremont, CA, USA) was placed in the aneurysmal dilatation from left VA for the internal trapping. A 2.5×17 mm LVIS Jr. (MicroVention, Tustin, CA, USA) stent was inserted via the left PICA to the left distal VA, just proximal to the origin of the anterior spinal artery (ASA). The withdrawn Headway17 microcatheter was introduced into the aneurysm for double-catheter coil embolization (Fig. 3a). The VADA was then completely occluded using 13 detachable coils (Fig. 3b). One week follow-up angiography results showed an occluded VADA with preserved left PICA flow. The patient recovered well with no neurologic deficits and she was discharged to her home 1 month later. She was prescribed triple antiplatelet therapy for 1 month and her blood pressure remained normal.

Three months after the initial treatment, follow-up angiography revealed a complete occlusion of the left VADA with patent PICA flow, but it also showed a *de novo* VADA on the right side (Fig. 4a). We intended to occlude this *de novo* VADA and preserve the right VA patency. Under general anesthesia, we placed a microcatheter (Headway17) just proximal to the VA union for stenting, and another microcatheter (Headway17) was placed into the aneurysmal dilatation for coil embolization. A 3.5×28 mm LVIS Jr. stent was placed in the right VA covering the dissecting segment. Four detachable coils were then placed into the aneurysmal dilatation from the situated microcatheter. Complete occlusion of the aneurysm while preserving the right VA flow was achieved without any complications (Fig. 4b,c). The patient showed an uneventful post-operative course with no neurological deficits. She continued double antiplatelet therapy for 6 months.

Discussion

A ruptured VADA should be treated as soon as possible because its rate of re-bleeding is extremely high [10]. When treating a VADA, the angioarchitecture such as VA dominancy, location of the PICA origin, ASA involvement, and bilateral lesions should be considered. Recent developments in endovascular treatment include a new therapeutic strategy called stent assisted coil embolization, which can preserve normal VA flow using a stent. However, stent usage in the acute stage of subarachnoid hemorrhage may cause ischemic complications [6, 9]. If the VADA is unilateral and the affected side can be sacrificed, surgical or internal trapping of the affected VA is believed to be the best treatment in the acute stage of subarachnoid hemorrhage [10, 13, 14].

Because PICA is one of the major arteries supplying the cerebellar hemisphere, the involvement of PICA is an important factor in the treatment of VADA. Mizutani et al. categorized VADAs into four types according to the location of the PICA origin [10]. Pre-, post-, and non-PICA type VADAs can be treated using internal trapping of the affected VA while preserving the PICA. In case of a PICA-involved type VADA, PICA reconstruction is necessary. Conventional treatment for PICA-involved type VADA is an endovascular segmental occlusion using coils followed

by PICA-to-PICA anastomosis or occipital artery-PICA bypass. Stent-assisted coil embolization is currently a helpful and alternative way to treat PICA-involved type VADA [13, 14].

Some authors have reported the efficacy and safety of VA-PICA stenting for PICA-involved type VADA [2, 6, 15]. There are several methods of stent deployment, and the direction of stenting depends on the PICA origin site from the aneurysm and its angulation. In the present patient, the PICA was located at the distal part of the aneurysmal dilatation and it originated at an acute angle from the left VA. The contralateral approach seemed to be straighter for stenting. Stenting into PICA is sometimes challenging because of its small caliber, and stent patency is a major concern when the stent is placed in small vessels. The LVIS Jr. microstent has several advantages over the currently available intracranial stents [11]. The smallest delivery system facilitates navigation and precise advancement in small vessels. Although the LVIS Jr. is intended for use in aneurysms located on arteries from 2–3.5 mm in diameter, some authors applied it in vessels less than 2 mm diameter without stent occlusion [1, 11]. They showed a high degree of safety and a high rate of aneurysm occlusion. The LVIS Jr. stent appears to be effective and suitable for VADA in posterior circulation. In addition, adequate antiplatelet therapy was needed to prevent ischemic complications. Triple antiplatelet therapy was started and the platelet function was monitored using Verify Now.

Although our treatment strategy for the left VADA seemed to be reasonable, a *de novo* VADA had developed on the contralateral side. Only a few reports have described cases of *de novo* VADA after an occlusion of the contralateral VA [3-5, 8, 12]. Previously reported cases are summarized in Table 1. The interval between the initial dissection and the detection of *de novo* contralateral dissection varies from patient to patient. Most patients were treated using conservative therapy. For an asymptomatic *de novo* VADA, conservative therapy may provide good outcomes. However, once the patients develop symptoms (SAH or infarction), the clinical course is life-threatening and surgical treatment is difficult because of an inability to trap the remaining VA. In the present case, a *de novo* VADA had developed even with good blood pressure control. Because there was no appropriate choice of conservative treatment, we decided to perform the endovascular treatment for the right VADA although it was asymptomatic.

The mechanism of development for these aneurysms is not clear. The present patient had no risk factors for arterial dissection such as hypertension, diabetes mellitus and connective tissue disorders. Occlusion of the affected VA may cause increased blood flow in the contralateral VA, resulting in increased hemodynamic stress, which has been shown in several studies. Yasui et al. reported that the VA diameter increased after occlusion of the contralateral VA [16]. Kono et al. performed computational fluid dynamic simulations of contralateral VA trapping. In their simulated case of bilateral VADAs, trapping would increase the wall sheer stress in the dome surface of the contralateral aneurysm [7]. Excessive hemodynamic stress can lead to disruption of the internal elastic lamina and consequently result in artery dissection. In the acute phase of SAH, aggressive volume loading for the treatment of vasospasm also contributes to artery dissections [3].

Conclusion

Increased hemodynamic stress may cause development of dissecting aneurysms. After internal trapping for VADA, radiologic follow-up is necessary to detect *de novo* dissection because of post-therapeutic hemodynamic changes.

Informed Consent: The patient has consented to submission of this case report to the journal.

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Conflict of Interest: The authors declare that they have no conflict of interest.

References

1. Alghamdi F, Mine B, Morais R, Scillia P, Lubicz B (2016) Stent-assisted coiling of intracranial aneurysms located on small vessels: midterm results with the LVIS Junior stent in 40 patients with 43 aneurysms. Neuroradiology 58:665-671
2. Chung J, Kim BS, Lee D, Kim TH, Shin YS (2010) Vertebral artery occlusion with vertebral artery-to-posterior inferior cerebellar artery stenting for preservation of the PICA in treating ruptured vertebral artery dissection. Acta Neurochir (Wien) 152:1489-1492
3. Horie N, Sadakata E, Izumo T, Hayashi K, Morikawa M, Nagata I (2015) De Novo Vertebral Artery Dissection after Endovascular Trapping for Ruptured Dissecting Internal Carotid Artery Aneurysm: Case Report. Neurol Med Chir (Tokyo) 55:524-527
4. Inui Y, Oiwa Y, Terada T, Nakakita K, Kamei I, Hayashi S (2006) De novo vertebral artery dissecting aneurysm after contralateral vertebral artery occlusion--two case reports. Neurol Med Chir (Tokyo) 46:32-36
5. Katsuno M, Mizunari T, Kobayashi S, Takahashi H, Teramoto A (2009) Rupture of a vertebral artery dissecting aneurysm developing immediately after trapping of a dissecting aneurysm on the contralateral vertebral artery: case report. Neurol Med Chir (Tokyo) 49:468-470
6. Kim MJ, Chung J, Kim SL, Roh HG, Kwon BJ, Kim BS, Kim TH, Kim BM, Shin YS (2012) Stenting from the vertebral artery to the posterior inferior cerebellar artery. AJNR Am J Neuroradiol 33:348-352
7. Kono K, Shintani A, Fujimoto T, Terada T (2012) Stent-assisted coil embolization and computational fluid dynamics simulations of bilateral vertebral artery dissecting aneurysms presenting with subarachnoid hemorrhage: case report. Neurosurgery 71:E1192-1200; discussion E1200-1191
8. Kubo Y, Miura K, Suzuki M, Tsuiki K, Kuwata N, Kubo N, Kuroda K, Ogawa A (1998) Development of a dissecting aneurysm on the vertebral artery immediately after occlusion of the contralateral vertebral artery: a case report. Neurosurg Rev 21:177-180
9. Lim SH, Shin HS, Lee SH, Koh JS (2015) Endovascular Treatment of Vertebral Artery Dissecting Aneurysms That Cause Subarachnoid Hemorrhage : Consideration of Therapeutic Approaches Relevant to the Angioarchitecture. J Korean Neurosurg Soc 58:175-183
10. Mizutani T, Aruga T, Kirino T, Miki Y, Saito I, Tsuchida T (1995) Recurrent subarachnoid hemorrhage from untreated ruptured vertebrobasilar dissecting aneurysms. Neurosurgery 36:905-911; discussion 912-903
11. Mohlenbruch M, Herweh C, Behrens L, Jestaedt L, Amiri H, Ringleb PA, Bendszus M, Pham M (2014) The LVIS Jr. microstent to assist coil embolization of wide-neck intracranial aneurysms: clinical study to assess safety and efficacy. Neuroradiology 56:389-395
12. Otawara Y, Ogasawara K, Ogawa A, Kogure T (2002) Dissecting aneurysms of the bilateral vertebral arteries with subarachnoid hemorrhage: report of three cases. Neurosurgery 50:1372-1374; discussion 1374-1375
13. Sugiu K, Tokunaga K, Date I (2004) [Endovascular treatment for ruptured vertebral artery dissecting

aneurysms]. No Shinkei Geka 32:1229-1238

14. Sugiu K, Tokunaga K, Watanabe K, Sasahara W, Ono S, Tamiya T, Date I (2005) Emergent endovascular treatment of ruptured vertebral artery dissecting aneurysms. Neuroradiology 47:158-164

15. Wu Z, Lv X, Yang X, He H (2009) Ruptured vertebro-inferoposterior cerebellar artery dissecting aneurysm treated with the Neuroform stent deployment and vertebral artery occlusion. European Journal of Radiology Extra 70:e100-e103

16. Yasui T, Sakamoto H, Kishi H, Komiyama M, Iwai Y, Yamanaka K, Nishikawa M (1998) Bilateral dissecting aneurysms of the vertebral arteries resulting in subarachnoid hemorrhage: case report. Neurosurgery 42:162-164; discussion 165

Figure Legends

Fig. 1 a) Brain CT demonstrated typical subarachnoid hemorrhage. **b)** 3-dimentional CT angiography showed a dilatation (arrow) and narrowing of left vertebral artery, indicating a vertebral artery dissecting aneurysm

Fig. 2 a) Bilateral vertebral angiogram showed the left VADA, which includes the origin of the posterior inferior cerebellar artery. **b)** 3-D reconstruction image of the left VA showed that the PICA origin was included in the aneurysmal dilatation (arrow). Simulation image of stenting indicates our plan to place the LVIS Jr. stent. The arrow indicates the anterior spinal artery originating from the left distal VA

Fig. 3 a) The maximum intensity projection image demonstrated good deployment of the LVIS Jr. stent between left distal VA and left PICA (arrow). Two microcatheters for the coiling are also seen in the aneurysm (arrowheads). **b)** 3-D reconstruction image after internal trapping of the left VADA showed good patency of the stented PICA and occlusion of the left VA

Fig. 4. a) At a three-month follow-up, the right VAG revealed *de novo* VADA (arrow) in the right VA. Good patency of the left PICA through the left distal VA is shown. **b)** After stent assisted coil embolization of the right VADA, right VAG demonstrated good patency of the right VA with successful occlusion of the aneurysmal dilatation. **c)** 3-D reconstruction image also showed good patency of the right VA with successful occlusion of the aneurysmal dilatation by detachable coils

Table 1. Reported cases describing *de novo* VADA after occlusion of the contralateral VA

Figure1

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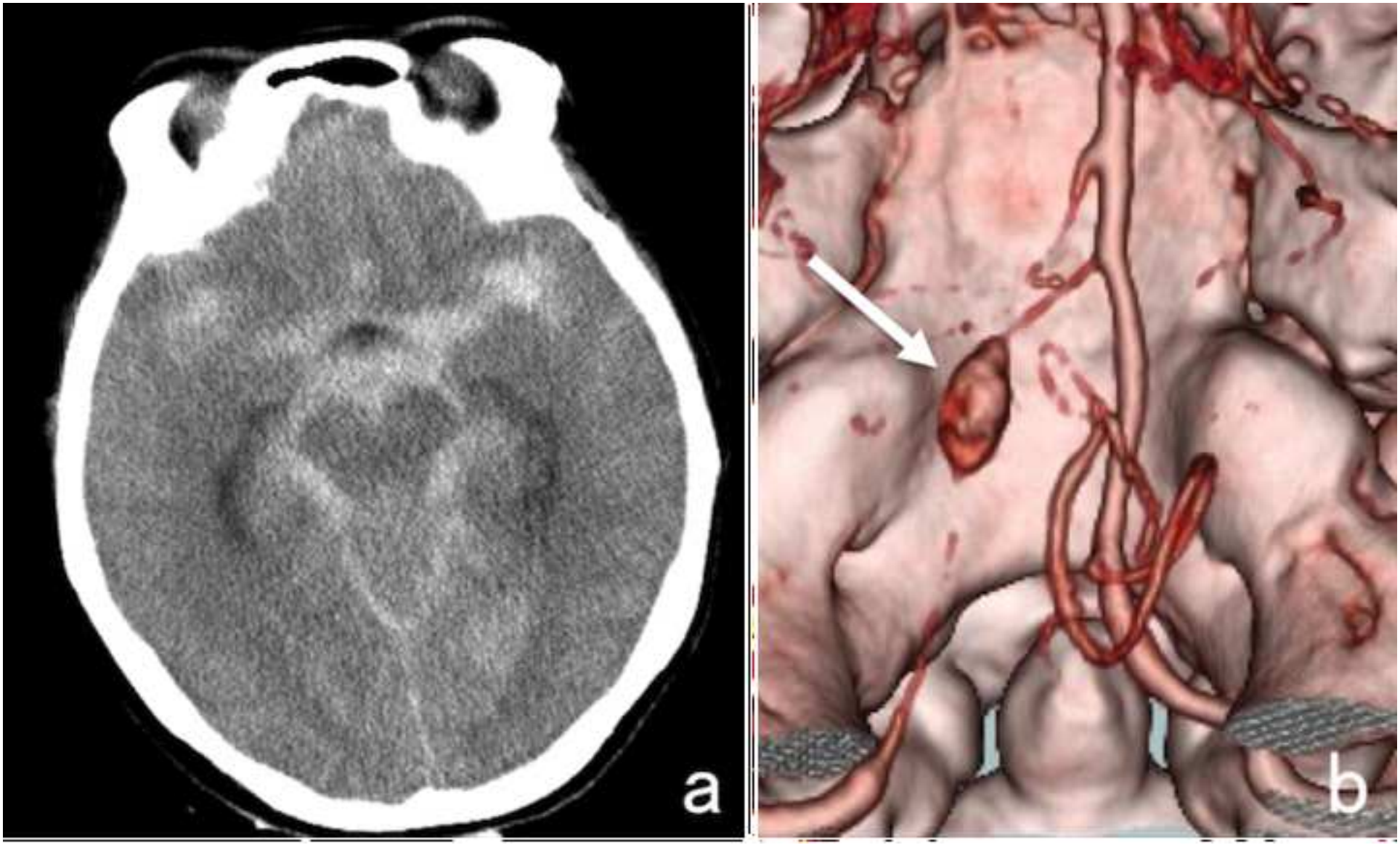

Figure2

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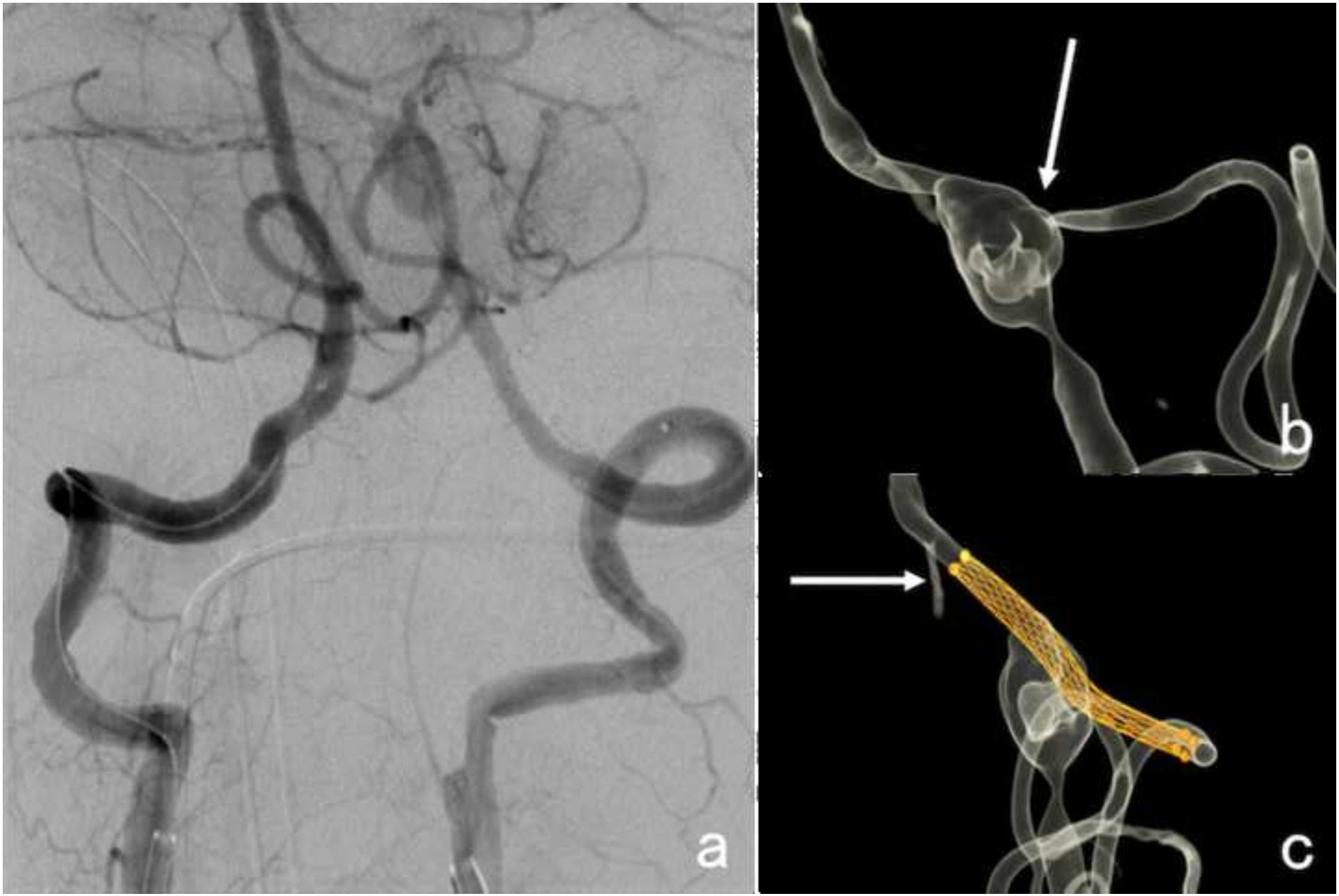

Figure3

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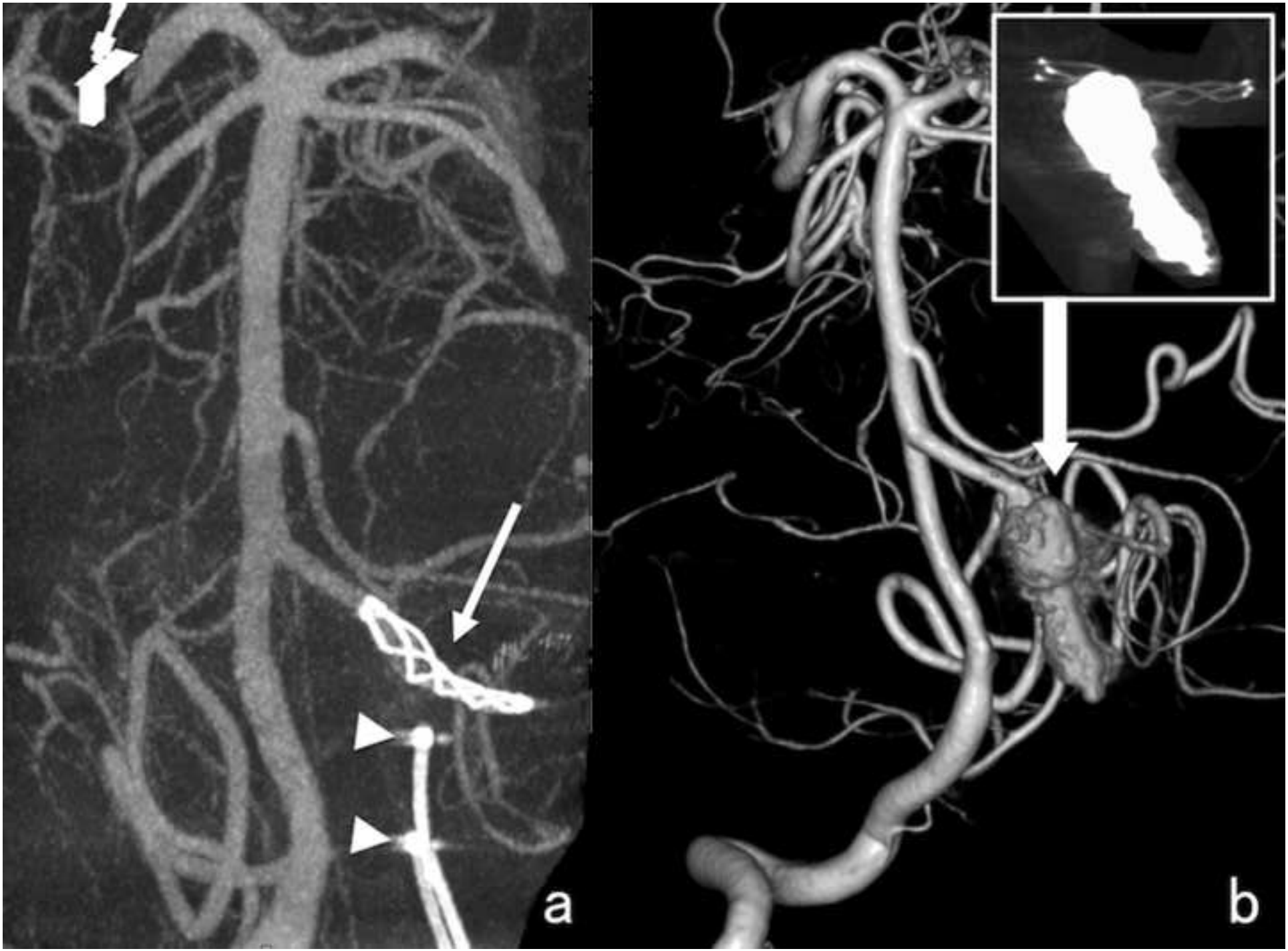

Figure4

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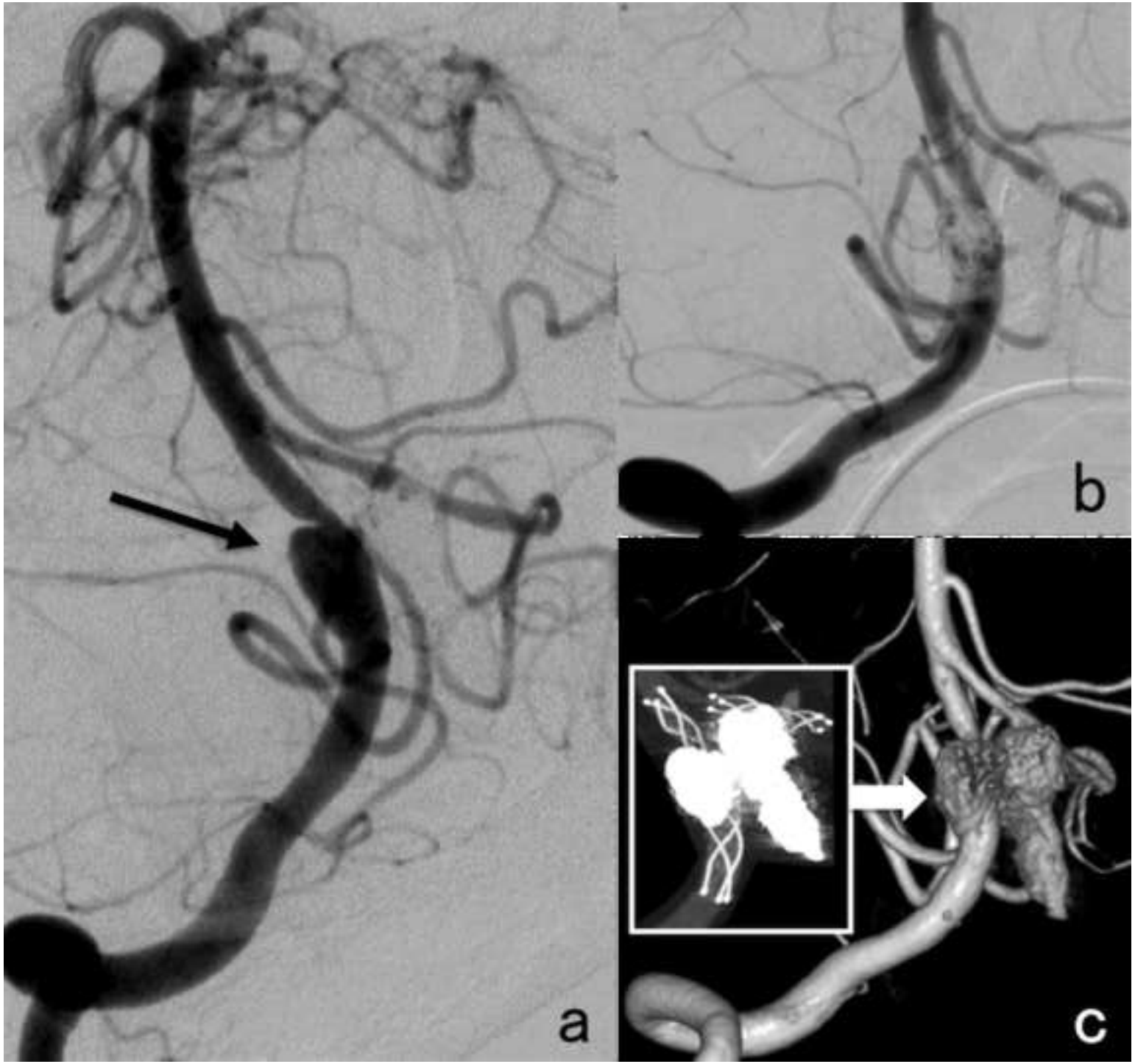

Table 1

Age, Sex	Author	Initial onset	Initial treatment	Interval	2nd onset	2nd treatment	Outcome
49,F	Kubo et al.[8]	SAH	Proximal occlusion	3w	Asympt	Proximal occlusion	GR
51,F	Otawara et al.[12]	SAH	Surgical trapping	1m	Asympt	Conservation	GR
36,M	Inui et al.[4]	Infarction	Conservation	13m	Infarction	Conservation	D
45,M	Inui et al.[4]	SAH	Endovascular trapping	2w	Infarction	Conservation	SD
39,M	Katsuno et al.[5]	SAH	Surgical trapping	8h	SAH	Conservation	D
55,F	Present	SAH	Endovascular trapping	3m	Asympt	Stent assisted coil	GR

SAH, subarachnoid hemorrhage; h, hours; w, weeks; m, months; Asympt, asymptomatic; GR, good recovery; SD, severely disabled; D, died