

Hemodynamic Impact of a Spontaneous Cervical Dissection on an Ipsilateral Saccular Aneurysm

Alfred P. See¹, Bradley A. Gross², David L. Penn¹, Rose Du¹, Kai U Frerichs¹

¹Department of Neurosurgery, Brigham and Women's Hospital and Harvard Medical School, Boston, MA, USA

²Department of Neurosurgery, Barrow Neurological Institute, Phoenix, AZ, USA

The dynamic, hemodynamic impact of a cervical dissection on an ipsilateral, intracranial saccular aneurysm has not been well illustrated. This 45-year-old female was found to have a small, supraclinoid aneurysm ipsilateral to a spontaneous cervical internal carotid artery dissection. With healing of the dissection, the aneurysm appeared to have significantly enlarged. Retrospective review of the magnetic resonance imaging (MRI) at the time of the initial dissection demonstrated thrombus, similar in overall morphology to the angiographic appearance of the "enlarged" aneurysm. As the dissection healed far proximal to the intradural portion of the internal carotid artery, this suggested that the aneurysm was likely a typical, saccular posterior communicating artery aneurysm that had thrombosed and then recanalized secondary to flow changes from the dissection. The aneurysm was coiled uneventfully, in distinction from more complex treatment approaches such as flow diversion or proximal occlusion to treat an enlarging, dissecting pseudoaneurysm. This case illustrates that flow changes from cervical dissections may result in thrombosis of downstream saccular aneurysms. With healing, these aneurysms may recanalize and be misidentified as enlarging dissecting pseudoaneurysms. Review of an MRI from the time of the dissection facilitated the conclusion that the aneurysm was a saccular posterior communicating artery aneurysm, influencing treatment approach.

J Cerebrovasc Endovasc Neurosurg.
2016 June;18(2):110-114

Received : 1 May 2015

Revised : 29 August 2015

Accepted : 27 December 2015

Correspondence to Bradley A. Gross

Department of Neurosurgery, Barrow Neurological Institute, 350 W Thomas Rd Phoenix, AZ 85013, USA

Tel : 1-602-406-3000

Fax : 1-617-734-8342

E-mail : Bgross83@gmail.com

ORCID : <http://orcid.org/0000-0003-2599-5182>

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Keywords Aneurysm, Endovascular, Dissection, Saccular aneurysm, Thrombosis

INTRODUCTION

Arterial dissection (AD), a separation of the layers of an arterial wall, most often occurs spontaneously but may also result from trauma.⁸⁾ Although rare, an estimate from a North American study of cervical AD reported an annual incidence of 2.6 per 100,000.¹¹⁾ Although embolic events are the most commonly implicated cause of ischemic events as a result of cervical AD, cervical vessel thrombosis or hypoperfusion may also result in ischemic stroke.¹⁾¹²⁾

Hemorrhagic stroke from rupture of an intracranial

aneurysm (IA) is a well-known source of significant neurological morbidity and mortality.⁵⁾ Parent artery occlusion and resultant aneurysm thrombosis was an early approach for the treatment of aneurysms with rare, though continued, modern applications today.²⁾³⁾¹³⁾

In this unique report, we illustrate the fascinating interaction of a spontaneous cervical AD and an incidental, ipsilateral intracranial saccular aneurysm. The history initially draws analogy to the hemodynamic effect of proximal parent artery occlusion in the treatment of cerebral aneurysms with an interesting turn of events as the dissection heals with concomitant recanalization

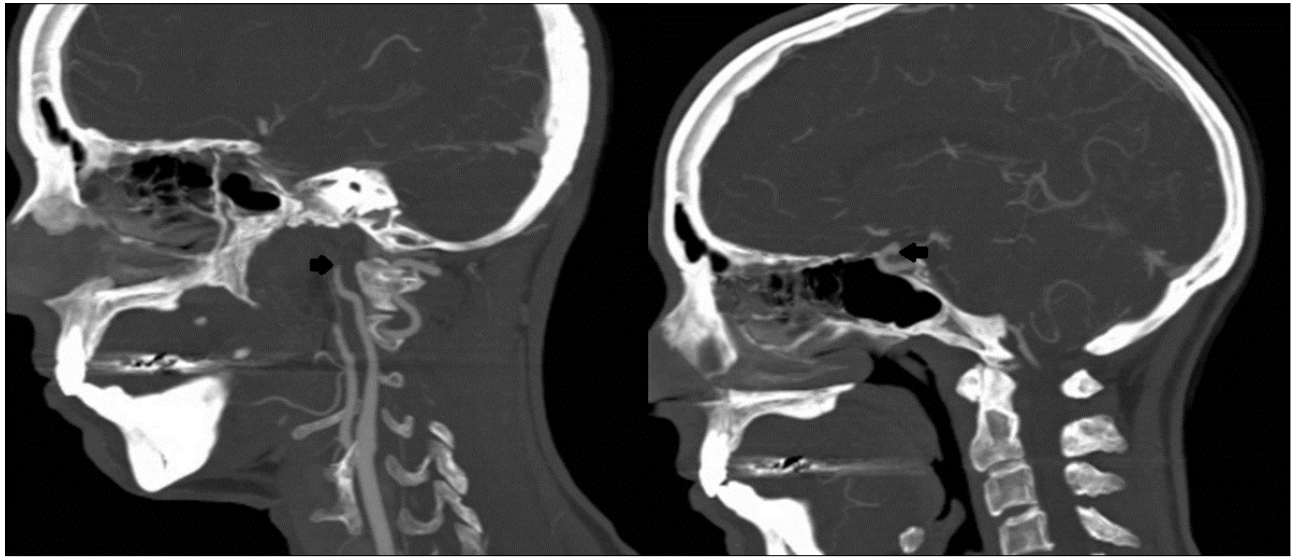


Fig. 1. CTA demonstrates a cervical left internal carotid artery dissection (arrow, sagittal, left panel) and a 4 mm left supraclinoid aneurysm (sagittal, right panel, arrow). CTA = computed tomography angiography.

of the aneurysm.

CASE REPORT

This 45-year-old female with a past medical history of pre-eclampsia and hypertension presented with left eye pain, chemosis, and ptosis. She was initially placed on empiric zoster treatment; however, a magnetic resonance imaging (MRI) scan obtained during the workup for persistent headache demonstrated a left internal carotid artery (ICA) dissection extending from the distal cervical to petrous segment. She was initially managed with anticoagulation (warfarin).

Two days later, she presented to our institution after a syncopal event. Physical examination was remarkable for left pupillary miosis and left-sided ptosis; she was otherwise intact. Computed tomography angiography (CTA) demonstrated her dissection, as well as an irregularly-shaped, ipsilateral supraclinoid aneurysm (Fig. 1). Given its morphology, the possibility of a dissecting pseudoaneurysm was raised. Her anticoagulation was ceased, and she was started on aspirin 325 mg daily.

A two week interval CTA demonstrated no significant change in her dissection or the aneurysm.

However a four month interval CTA demonstrated a significant increase in size of the aneurysm (Fig. 2), prompting neurosurgical consultation. Digital subtraction angiography at that time demonstrated an irregularly shaped, 9.4×6.2 mm aneurysm in the region of the posterior communicating artery with a 3.6



Fig. 2. Scheduled follow-up CTA at 4 months redemonstrates the left supraclinoid aneurysm with significant increase in contrast filling compared to the prior CTA; it is now measured at 10 mm. CTA = computed tomography angiography.

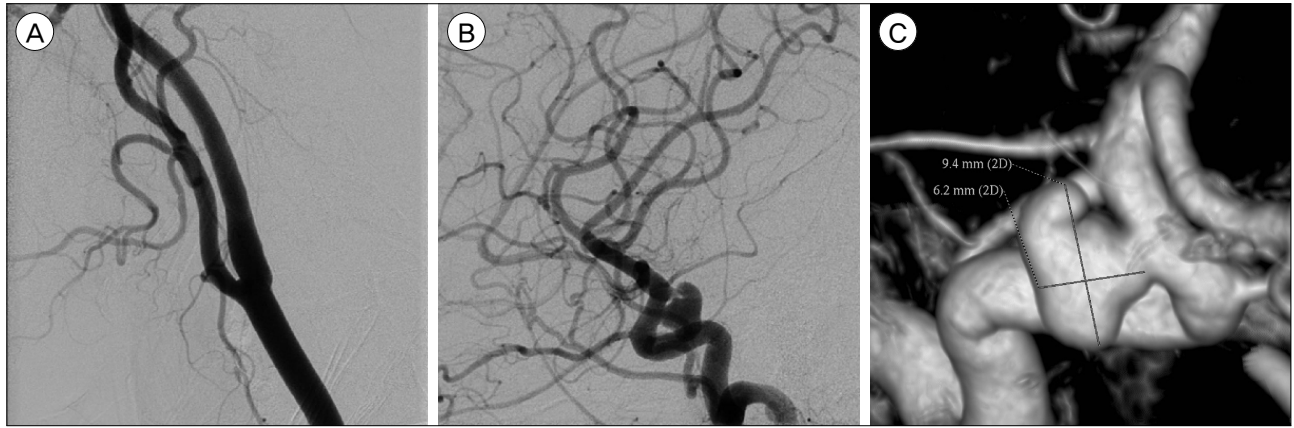


Fig. 3. Digital subtraction angiography for further evaluation of the dissection and aneurysm demonstrates a healed left ICA dissection (A, lateral view of common carotid artery injection) and an irregular, 9.8 × 6.2 mm posterior communicating artery aneurysm (B, lateral view of common carotid artery injection); C, 3D-reconstruction. ICA = internal carotid artery.

× 2.7 mm daughter dome and near complete resolution of the cervical ICA dissection (Fig. 3). 3-dimensional rotational angiography as well as the 2-dimensional angiograms made extension of the cervical ICA dissection to the communicating segment with resultant aneurysm formation seem dubious. A retrospective review of the patient's first MRI at the time of the initial dissection confirmed what was likely an incidental, ipsilateral posterior communicating artery

aneurysm with significant thrombus (Fig. 4); the morphology and size seen on MRI were quite similar to that seen on the angiogram months later. On the premise that the patient had a true saccular posterior communicating artery aneurysm that had originally partially thrombosed at the time of dissection and then recanalized as the dissection healed, the aneurysm was treated via direct coiling. This proceeded uneventfully (Fig. 5); however, interestingly, the patient

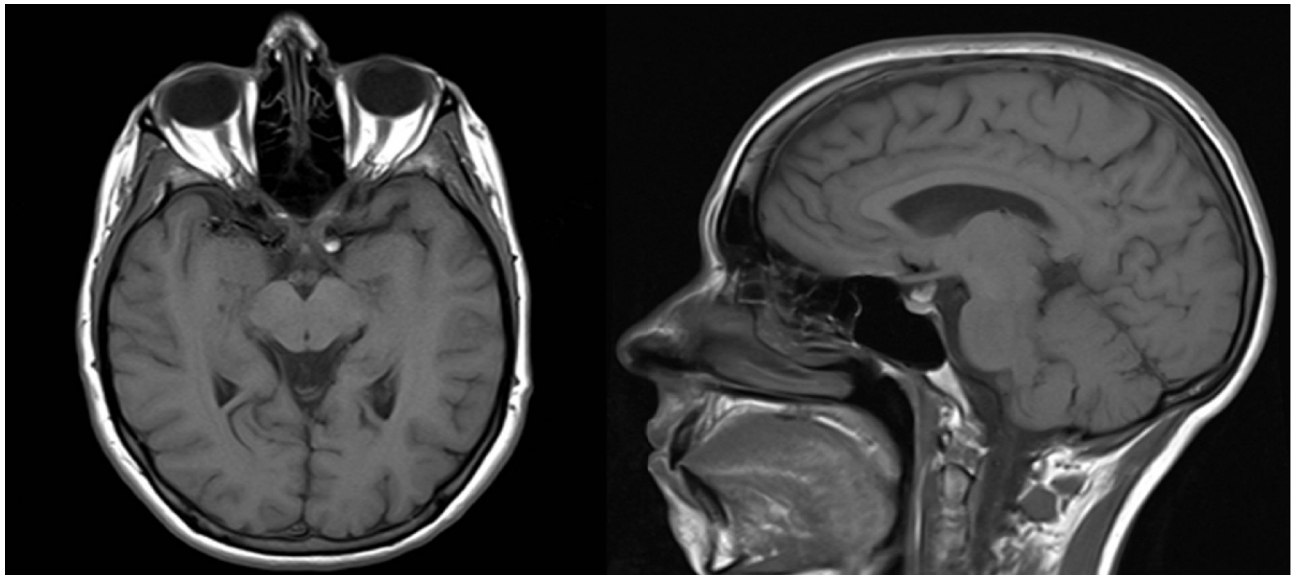


Fig. 4. Axial T1 MRI demonstrates thrombus posterior to the communicating segment of the left internal carotid artery (left panel). Sagittal T1 MRI demonstrates thrombus inferior to the communicating segment of the left internal carotid artery, similar in morphology to the aneurysm seen in Fig. 3A (right panel). MRI = magnetic resonance imaging.

initially complained of a post-coil headache that was very similar in quality to the headache she originally experienced at the time of the dissection. At the time of 11 month follow-up, the patient's Horner's syndrome had improved and MRI/A demonstrated stable coil occlusion of her aneurysm.

DISCUSSION

This case illustrates the dynamic interaction between a spontaneous AD and an ipsilateral saccular IA. The patient had no family history or known underlying connective tissue disorder that may have increased the propensity of comorbid IA and AD. Although she originally presented with a Horner's syndrome attributable to the dissection, her headache, retrospectively similar in quality to the headache she experienced after coiling, may have in fact been due to thrombosis of the aneurysm, seen in retrospect on her MRI. This case report nicely illustrates the impact of reduced antegrade flow through the ipsilateral ICA and resultant thrombosis of this saccular aneurysm, akin to traditional Hunterian approaches to aneurysm treatment.

Subsequent healing of the dissection adversely resulted in recanalization of the aneurysm, presumably secondary to a return of normal antegrade ICA flow and the return of hemodynamic stress to the ICA-posterior communicating artery branch point.

Without careful perusal of the initial MRI at the time of dissection, this recanalization may have been interpreted instead as growth of a dissecting pseudoaneurysm that had perhaps extended from the initial cervical AD. Such significant growth would prompt expedient treatment; however direct coiling would be unlikely to result in a durable, successful treatment of such an aneurysm. Instead, flow diversion with parent vessel reconstruction via a flow diverting stent or possibly proximal vessel occlusion with or without microsurgical bypass would be needed. Fortunately, recognition of the thrombosed aneurysm's morphology on the initial MRI at the time of dissection strongly suggested this was a saccular aneurysm that had thrombosed and then recanalized with healing of the dissection. As such, a technically more facile, direct coiling approach was employed in its treatment with successful results.

Such a clear interplay between a spontaneous cervical

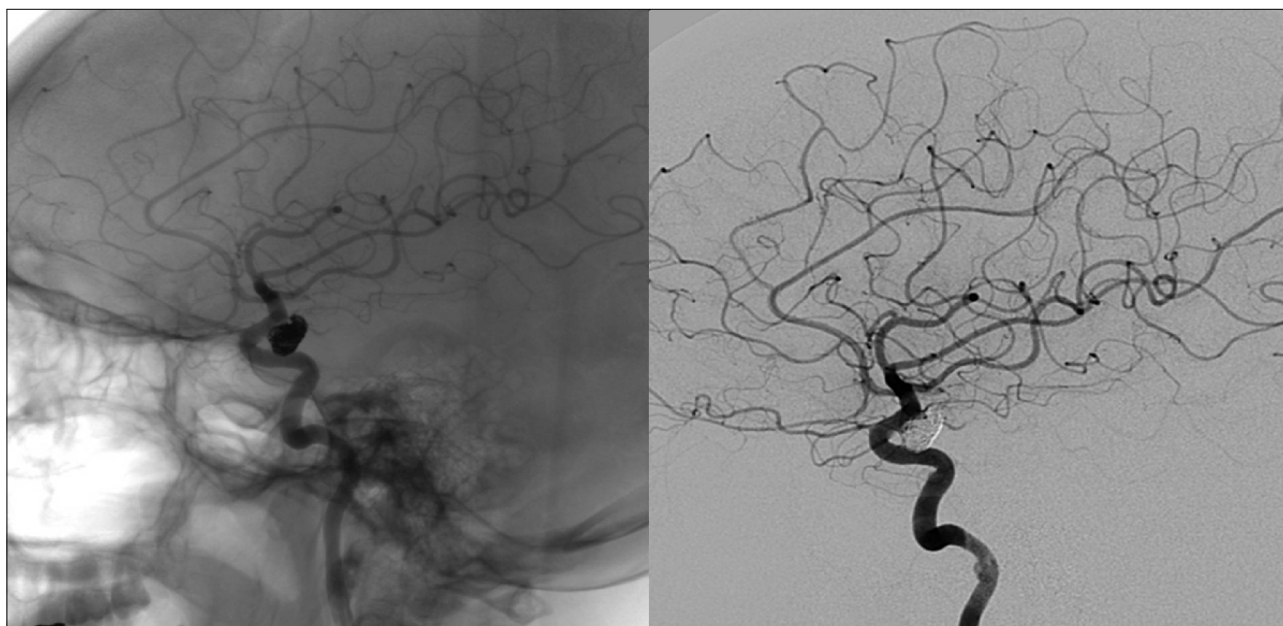


Fig. 5. Left panel: Lateral projection of the left internal carotid injection demonstrates coil pack within aneurysm. Right panel: Lateral projection of a subtracted image of the left internal carotid injection.

AD and an ipsilateral IA has not, to our knowledge, been previously documented in the literature. In one case report by Esposito and colleagues, a patient presented with diffuse subarachnoid hemorrhage and a right cervical carotid dissection, a right posterior communicating artery aneurysm, and a right paraclinoid aneurysm.⁴⁾ Both aneurysms were clipped with confirmatory indocyanine green angiography and post-operative digital subtraction angiography, the latter demonstrating relative resolution of the initial dissection. Three days after clipping, the patient suffered a recurrent subarachnoid hemorrhage. DSA at that time demonstrated a previously unseen right internal carotid artery aneurysm. The authors inferred that AD variability and hemodynamic changes may have led to IA formation.

The association between IA and AD may be related to a susceptibility to vascular injury, perhaps best illustrated by patients with inheritable connective tissue disorders.⁹⁾¹⁰⁾ Mazighi et al. reported a series of patients with simultaneous AD without intracranial extension and anatomically distinct subarachnoid hemorrhage from IA rupture.⁷⁾ They hypothesized that without acute external trauma, the sympathetic surge and resulting hypertension associated with aneurysmal rupture may disrupt the intimal structure of the artery, resulting in AD. This aligns with the observation of stress-induced myocardial injury as a known sequela of IA rupture.⁶⁾ It may be challenged that they did not observe increased rates of dissection in other arteries, but that may be due to sub-clinical dissection in vessels which are otherwise not routinely imaged in the management of IA rupture.¹⁴⁾

CONCLUSION

This case illustrates flow reduction as a result of a spontaneous dissection resulting in partial aneurysm thrombosis and subsequent recanalization after flow restoration with dissection healing. Recognition of this phenomenon after review of the patient's initial MRI allowed for a safe, facile cure of the aneurysm via direct coiling as it was identified as a recanalized saccular IA rather than an enlarging, dissecting IA.

Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

REFERENCES

1. Benninger DH, Georgiadis D, Kremer C, Studer A, Nedeltchev K, Baumgartner RW. Mechanism of ischemic infarct in spontaneous carotid dissection. *Stroke* 2004 Feb; 35(2):482-5.
2. Crutchfield WG. Instruments for use in the treatment of certain intracranial vascular lesions. *J Neurosurg* 1959 Jul;16(4):471-4.
3. Drake CG, Peerless SJ, Ferguson GG. Hunterian proximal arterial occlusion for giant aneurysms of the carotid circulation. *J Neurosurg* 1994 Nov;81(5):656-65.
4. Esposito G, Sabatino G, Lofrese G, Albanese A. Carotid artery dissection-related intracranial aneurysm development: case report. *Neurosurgery* 2012 Feb;70(2):E511-4; discussion E514-5.
5. Hop JW, Rinkel GJ, Algra A, van Gijn J. Case-fatality rates and functional outcome after subarachnoid hemorrhage: a systematic review. *Stroke* 1997 Mar;28(3):660-4.
6. Lee VH, Connolly HM, Fulgham JR, Manno EM, Brown RD Jr, Wijndicks EF. Tako-tsubo cardiomyopathy in aneurysmal subarachnoid hemorrhage: an underappreciated ventricular dysfunction. *J Neurosurg* 2006 Aug;105(2):264-70.
7. Mazighi M, Saint Maurice JP, Rogopoulos A, Houdart E. Extracranial vertebral and carotid dissection occurring in the course of subarachnoid hemorrhage. *Neurology* 2005; Nov;65(9):1471-3.
8. Rao AS, Makaroun MS, Marone LK, Cho JS, Rhee R, Chaer RA. Long-term outcomes of internal carotid artery dissection. *J Vasc Surg* 2011 Aug;54(2):370-4; discussion 375.
9. Rodrigues VJ, Elsayed S, Loeys BL, Dietz HC, Yousem DM. Neuroradiologic manifestations of Loeys-Dietz syndrome type 1. *AJNR Am J Neuroradiol* 2009 Sep;30(8):1614-9.
10. Schievink WI, Mokri B, Michels VV, Piepgras DG. Familial association of intracranial aneurysms and cervical artery dissections. *Stroke* 1991 Nov;22(11):1426-30.
11. Schievink WI, Mokri B, Whisnant JP. Internal carotid artery dissection in a community. Rochester, Minnesota, 1987-1992. *Stroke* 1993 Nov;24(11):1678-80.
12. Sharma GK, Deshmukh VR, Albuquerque FC, Wolf TR, McDougall CG. Resolution of mydriatic pupil after angioplasty and stenting of cervical internal carotid artery dissection: case report. *Neurosurgery* 2009 Mar;64(3):E562-3; discussion E563.
13. Surdell DL, Hage ZA, Eddleman CS, Gupta DK, Bendok BR, Batjer HH. Revascularization for complex intracranial aneurysms. *Neurosurg Focus* 2008 Feb;24(2):E21.
14. Völker W, Besselmann M, Dittrich R, Nabavi D, Konrad C, Dziewas R, et al. Generalized arteriopathy in patients with cervical artery dissection. *Neurology* 2005 May;64(9): 1508-13.