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Internal Carotid Artery Dilatation Induced by General Anesthesia: Technical Observation

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Technical Observation

General anesthesia exposure may increase the diameter of the parent vessel during flow-diverting stent placement of the large distal cervical and petrocavernous internal carotid artery (ICA) aneurysm. Remeasurement of the parent vessel during general anesthesia is advisable for the correct selection of the size of the flow-diverting implant.

Case Description

A 28-year-old previously healthy right-handed man presented with two episodes of left hemispheric transient ischemic attacks manifested by the right upper extremity clumsiness, numbness, and expressive aphasia in spite of medical treatment with antiplatelet medication. Neurologic examination was unremarkable and the MRI of the brain was negative for acute ischemic stroke. MR angiography showed a large aneurysm at the junction of the distal cervical and petrous segment of the left ICA.

Diagnostic cerebral angiogram under moderate sedation confirmed a focal arterial stenosis at the junction of the distal cervical ICA and the proximal petrous segment of the intracranial ICA complicated with a large pseudoaneurysm consistent with arterial dissection. The proximal parent vessel was severely tortuous and redundant with a 180° loop. The pseudoaneurysm measured 10 mm in its major diameter and the neck measured 5 mm (Figure 1). The neck of the pseudoaneurysm contains a large intimal flap that projected against the ascending (antegrade) arterial blood flow. The parent vessel proximal to pseudoaneurysm measured 5.3 mm at its major diameter. We proposed endovascular treatment using flow-diverting stent placement with a 5 × 30 mm pipeline embolization device (PED; eV3 Endovascular, Plymouth, MN, USA) to cover the aneurysm, since ICA vessel diameter was less than 5 mm, except for the segment proximal to pseudoaneurysm measuring 5.3 mm. Patient was placed on the dual antiplatelet regimen (Aspirin 85 mg plus Clopidogrel 75 mg daily) 5 days prior to the procedure and scheduled under general anesthesia. The follow-up angiogram showed an interval worsening of the luminal stenosis of the parent vessel and an increase in the size of the pseudoaneurysm. Moreover, the entire ICA was dilated. The parent vessel measured up to 6.7 mm, making it unsuitable for placement of even the 5 mm PED, which is the largest available size and can accommodate vessel lumen up to 5.25 mm (Figure 2). Not only the extracranial ICA but also

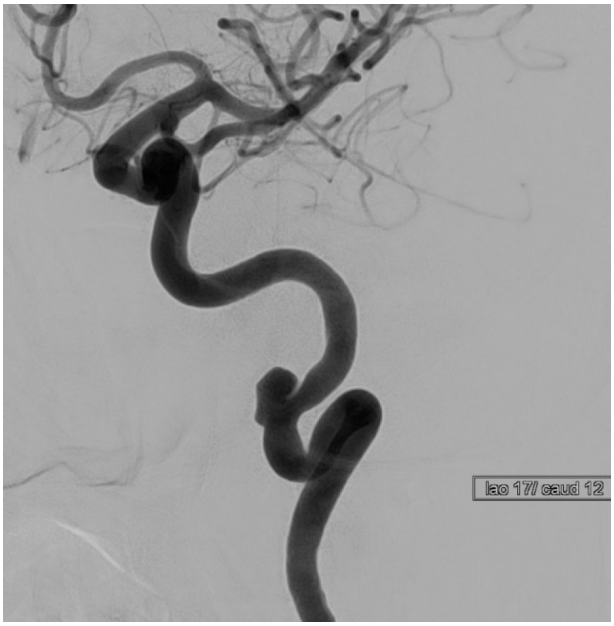

Figure 1. Left ICA injection showing a severe carotid redundancy with a focal lesion at the transition of the distal (suprabulbar) cervical and petrous segment. Stenosis and pseudoaneurysm were consistent with arterial dissection.

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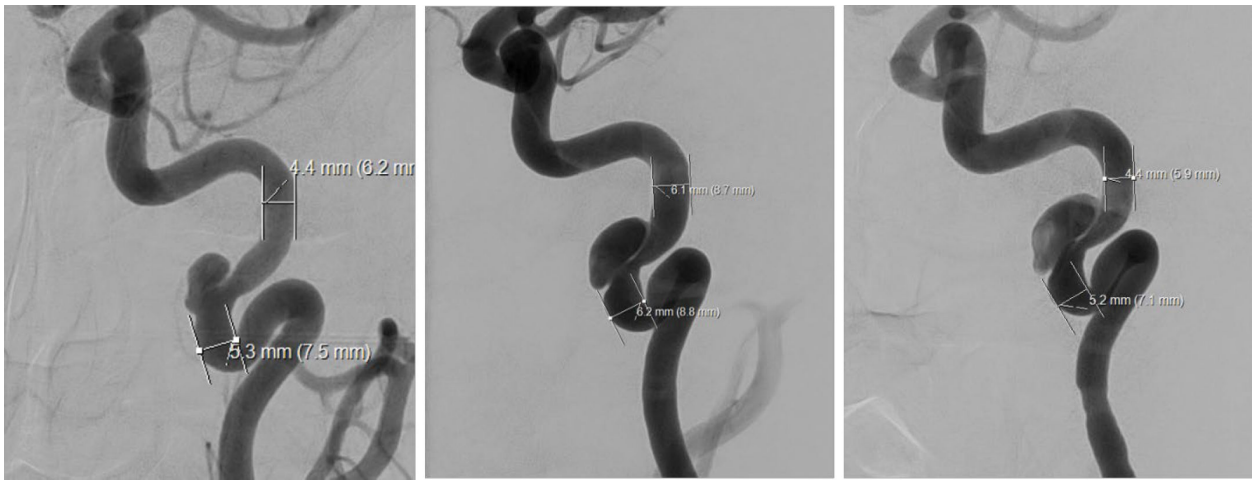

Figure 2. (A) Awake preoperative left internal carotid angiogram showing the luminal diameters of the parent artery at various planes. (B) Left internal carotid angiogram under general anesthesia showed marked dilatation of the artery with an increased major diameter up to 6.75 mm. (C) Subsequent awake angiogram of the left ICA showing returning of the luminal diameter at its baseline.

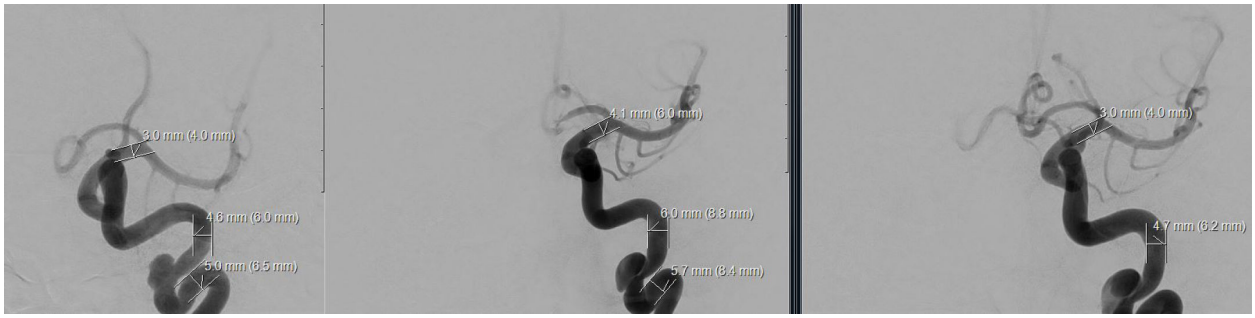

Figure 3. (A) Awake preoperative left internal carotid angiogram showing the major diameter of the supraclinoid segment of the intracranial ICA as 3.9 mm. (B) Left internal carotid angiogram under general anesthesia showed marked dilatation of the supraclinoid segment of the intracranial ICA with an increased major diameter up to 4.9 mm. (C) Subsequent awake angiogram of the left ICA showing returning of the supraclinoid segment diameter at its baseline.

the supraclinoid segment of the intracranial ICA was dilated (Figure 3). The increase in diameter in the ICA after placing the patient on general anesthesia was not subtle and exact measurement showed up to a 20% increase in the vessel diameter. The increase in the size of the carotid artery was reversible, as proven by a 24-hour follow-up cerebral angiogram performed with the patient awake that showed a return of the vessel diameter to its baseline. The anesthetic medication used during the induction and maintenance of general anesthesia comprised fentanyl, midazolam, propofol, lidocaine, and volatile inhaled anesthetic. Paralysis was achieved using rocuronium. All medications were administered at standard doses. There were no adverse effects during induction or maintenance of general anesthesia. The patient remained hemodynamically stable throughout the GA period. The lesion was later treated through endovascular stent-assisted coil embolization using the "jailing technique" with the patient awake during the procedure. A 5.5 × 33 mm low-profile visualized intraluminal support (LVIS Blue; Microvention, Tustin, CA, USA) stent was used along with bare metal detachable coils (Figure 4). The patient was discharged home the next day without complications.

Discussion

General anesthetics are known to cause changes in vascular reactivity and cardiovascular reflexes (i.e., baroreceptor reflexes). Literally, all general anesthetics, from

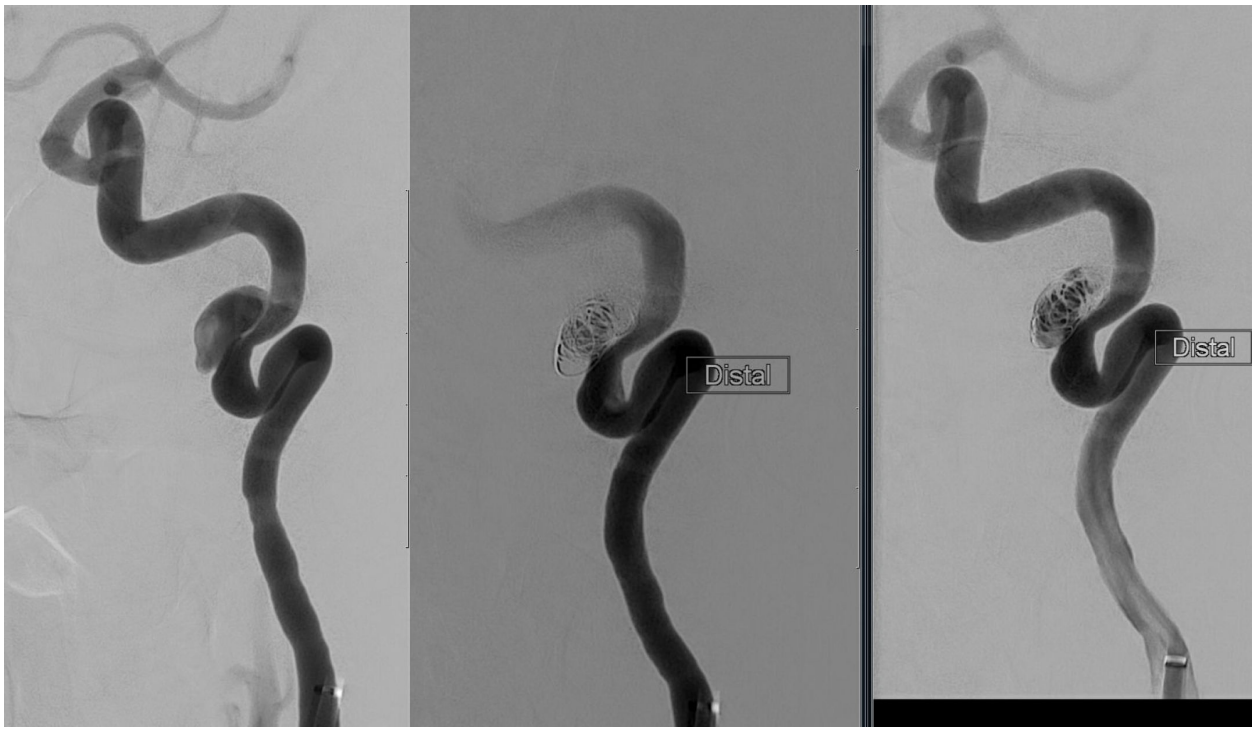

Figure 4. Treatment of the distal internal carotid dissection through endovascular reconstruction of the parent vessel using a 5.5 × 33 mm Lvis Blue stent and coil embolization of the pseudoaneurysm formation (Micrus Detachable coil) through a technique known as "Jailing Technique."

halogenated volatile (halothane and isoflurane) anesthetics to intravenous opioid (morphine, fentanyl) and nonopioid (barbiturates, propofol, ketamine, etomidate, and benzodiazepines) can cause changes in the vascular reactive properties of the cardiovascular system. The effects of general anesthetics in the vascular bed are mediated by the neural and nonneural mechanism. The neural mechanism is induced by a decreased sympathetic outflow from the central nervous system. The nonneural mechanism represents a direct effect of the general anesthetic drugs on the endothelial function and on the vascular smooth muscle cells. In general, intravenous anesthetics exert their vascular actions at supraclinical concentrations, whereas volatile anesthetics can cause changes in vascular reactivity at clinical concentrations. The target of general anesthetics is at multiple sites within the endothelial and vascular smooth cells and individual characteristics are responsible for the magnitude of their effects.

Spontaneous ICA dissection is a relatively common cause of ischemic stroke in young adults. The mechanism of spontaneous carotid dissection is unknown. Only 5% of the cases are related to collagen vascular disease with a specific inherited genetic pattern. Microscopic structural defects of the elastic and collagen fibers within the layers of the arterial wall are suspected. These structural changes might also account for different physiologic responses of the arterial wall to vasoactive substances.

We observed a significant increase in the luminal diameter of the ICA during GA exposure that returned to its baseline when the GA effect was removed making the effect of GA as the most likely explanation for this observation. Chronic partially healing carotid dissection with irregular lumen, associated stenosis, and enlarging pseudoaneurysm is the type of arterial lesions that would not be expected to resolve spontaneously. It is not uncommon for this carotid lesion to arise from redundant carotid arteries (from kink to 180° and 360° loops). Endovascular luminal reconstruction of spontaneous dissection of the distal cervical (suprabulbar) ICA can be challenging, particularly in cases in which severe tortuosity is present. The carotid stents designed for carotid artery stent placement for atherosclerotic stenosis of the distal common carotid artery and internal carotid origin (carotid bulb) are not trackable enough to navigate in the distal ICA. Biliary metal stents are sometimes helpful but again navigation in the distal carotid artery can pre-

clude their use. Intracranial stents are more likely to take pronounced curves and be more able to navigate in tortuous arterial anatomy. However, the bigger diameter of the extracranial ICA often precludes its use in the extracranial circulation.

The PED (eV3 Endovascular, Plymouth, MN, USA) is the only flow-diverting stent available for clinical use in the USA. The PED is approved for aneurysm arising from the anterior circulation (ICA) and situated below the posterior communicating segment of the intracranial ICA that measures more than 10 mm in size. Giant and very large aneurysms located in the distal cervical-petrous and petrous-cavernous transitions are successfully treated with PED. These stents can easily track and accommodate severe tortuous cervical ICA anatomy. Currently, the largest size is 5 mm and it can accommodate up to a 5.25 mm vessel size. The size of the distal cervical and petrous segment of the ICA typically ranges between 4 and 7 mm. Our case highlights the importance of recognizing the vasodilating effect of GA exposure as it will impact the ability to deploy these stents in an already borderline ICA during an awake cerebral angiogram.

Acknowledgements:

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Reference

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