Bilateral Cervical Internal Carotid Artery Pseudoaneurysms

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Abstract

A 67-year-old man who developed new-onset neurological symptoms was found to have bilateral internal carotid artery (ICA) pseudoaneurysms on computed tomography angiography. Pseudoaneurysms of the ICA are a rare diagnosis and can lead to significant morbidity and mortality including infarcts and, in some cases, hemorrhagic transformation of infarcts. The primary risk factors for the formation of ICA pseudoaneurysms are trauma and surgery. Other causes include infiltrating metastatic disease, infection, fibromuscular dysplasia, Ehlers-Danlos syndrome, Marfan syndrome, irradiation, and Behçet disease. Endovascular repair with stenting is a highly effective treatment option and preferred in patients with large symptomatic pseudoaneurysms. Depending on the etiology, open repair may be necessary. Individuals with smaller asymptomatic lesions are managed nonoperatively with antithrombotic therapy and are followed with serial imaging for significant changes in aneurysm size, which would necessitate procedural intervention.

Key words: internal carotid artery pseudoaneurysm, pseudoaneurysm, endovascular repair

Internal carotid artery (ICA) pseudoaneurysms are a rare entity that have the potential for life-threatening complications. In most institutions, the two most encountered etiologies are trauma and surgery. It is important to understand the variety of clinical and imaging parameters that are used to guide therapy. Ultimately, endovascular stenting is the preferred procedural management for large pseudoaneurysms and is performed with high procedural success rates (>90%).

Case Presentation

A 67-year-old man presented to the emergency department (ED) with new-onset left hand weakness that began 2 hours prior.

History

While driving, the patient developed sudden-onset weakness and numbness in his left hand that led him to lose control of his car and collide with a pole at 15 to 20 mph. The patient did not sustain trauma related to the accident. In the ED, he stated that the weakness in his left hand had improved but the numbness persisted. He reported that he was now also experiencing dizziness. His medical history was significant for hypertension, hypercholesterolemia, and Raynaud syndrome; he also had bilateral retinal detachment that was repaired 1 year ago, and he underwent phacoemulsification 3 years ago for treatment of bilateral cataracts. His current medications are atorvastatin 20 mg daily, amlodipine 10 mg daily, hydrochlorothiazide 25 mg daily.

Physical examination

The patient was alert and oriented. His blood pressure was 138/88 mm Hg, and his
other vital signs were stable. Neurologic examination showed paresthesia involving the entire left hand extending to the wrist. There were no other focal neurologic deficits or external signs of trauma.

Diagnostic studies
Cranial computed tomography (CT) demonstrated small chronic infarcts within the right frontal lobe (Figure 1) and right temporo-occipital lobes (Figure 2). There was no evidence of intracranial hemorrhage or obvious large vascular territory acute infarct. Computed tomography angiography (CTA) of the head and neck revealed bilateral tortuous cervical internal carotid arteries (ICAs) with large pseudoaneurysms containing eccentric mural thrombus/atherosclerotic plaque (Figures 3-6).

Same-day magnetic resonance imaging (MRI) of the brain confirmed the presence of chronic infarcts within the right frontal and temporo-occipital lobes (Figure 7). This study also revealed several tiny foci of diffusion restriction within the right cerebral hemisphere, likely related to showering emboli (Figure 8).

Treatment and management
The patient was therapeutically anticoagulated (heparin infusion) with plans to transition to dual-antiplatelet therapy after 24 hours.

Neurointerventional radiology was consulted for possible intra-arterial intervention. The patient was deemed to not be a suitable candidate, however, because deploying a stent across a pseudoaneurysm that demonstrated extensive vessel tortuosity and thrombi would not only be technically challenging but would also carry an unacceptably high risk of iatrogenic stroke. A vascular surgeon was subsequently consulted for possible open pseudoaneurysm repair, but this option was tabled after the patient's conditioned worsened and he became nonverbal, responding only to pain on physical examination. The patient was transferred to the neurocritical care unit. Cranial CT demonstrated loss of gray-white matter interface involving most of the right cerebral hemisphere (Figure 9). CTA showed occlusion of the right middle cerebral artery (MCA) at the bifurcation and occlusion of the right posterior
cerebral artery (PCA) at the temporal occipital and calcarine bifurcation (Figure 10).

The patient was transitioned from a heparin infusion to dual-antiplatelet therapy and was given hypertonic saline to decrease intracranial pressure. Shortly thereafter, pupil reactivity was found to be newly decreased. Cranial CT at this time demonstrated hemorrhagic conversion of the MCA territory infarct and new midline shift (Figure 11).

**Patient outcome**

Neurosurgery was consulted for decompressive craniectomy; however, surgical intervention was not advised as the patient demonstrated clinical signs of devastating brain injury, had received high doses of antithrombotic therapy, and had large unstable thrombi in bilateral ICAs. Thus, the health care team felt that surgical management may place the patient at risk for severe hemorrhage and would be unlikely to prevent significant long-term disability.

Therapeutic options were discussed with the patient's family, who declined further intervention. Comfort care only orders were issued; the patient died the following day.

**Discussion**

Aneurysmal dilation of the ICA can be categorized into true aneurysms...
and false aneurysms. True aneurysms represent full-thickness dilation of the expected vessel diameter by ≥50%. By contrast, false aneurysms, also known as pseudoaneurysms, do not involve all vessel wall layers and are a result of arterial wall injury; the affected layers form an outpouching that contains the leaking blood and retains connection with the true arterial lumen. The reported incidence of true ICA artery aneurysms vs pseudoaneurysms varies widely based on demographic variables; however, pseudoaneurysms have been estimated to account for as many as 82% of all extracranial carotid artery aneurysms.

The most common causes of ICA pseudoaneurysms are trauma and surgery, such as carotid endarterectomies or head and neck surgery. Less common etiologies include infiltrating metastatic disease, infection, fibromuscular dysplasia, Ehlers-Danlos syndrome, Marfan syndrome, irradiation, and Behçet disease.

Interestingly, the patient presented in this case did not have any of these risk factors yet had developed bilateral pseudoaneurysms. A literature review produced several similar case reports of bilateral ICA pseudoaneurysms; however, all these patients had a classic risk factor for the development of pseudoaneurysm (ie, trauma, infection, fibromuscular dysplasia, or radiation exposure).

In other vascular beds, such as the aorta, penetrating atherosclerotic ulcers have been found to cause pseudoaneurysms through disruption of the internal elastic lamina. Although the patient presented in this case did not have any of the classic risk factors previously mentioned, he was noted to have significant atherosclerotic disease that could have been related to the formation of bilateral carotid pseudoaneurysms.

Patients with ICA pseudoaneurysms are commonly asymptomatic; however, patients who do experience symptoms most often suffer neurologic deficits, as in this case. Other symptoms include a painful or palpable neck mass, airway compression secondary to rupture, or neck infection or cellulitis. Physical examination findings include focal neurologic or cranial nerve deficits, a pulsatile neck mass, or a systolic bruit.

The differential diagnosis for a patient with a pulsatile neck mass and neurological symptoms has a narrow scope that includes ICA aneurysm/pseudoaneurysm and carotid body tumor.

A lack of large-scale studies to guide management of extracranial ICA pseudoaneurysms poses a therapeutic challenge. Treatment decisions are based on such factors as symptoms, aneurysm size, location, and overall clinical status. Medical management primarily involves antithrombotic therapy with antiplatelets, anticoagulants, or both.

For patients who have a small, asymptomatic ICA pseudoaneurysm or who are poor surgical candidates, nonoperative management may be appropriate. These patients are typically treated with antithrombotic agents and followed with serial imaging for any change in aneurysm character.

In patients who present with symptoms, those who have a large pseudoaneurysm (>1 cm) or one that is rapidly expanding, or those who have a pseudoaneurysm that contains thrombus, surgical repair may
be necessary. Furthermore, therapeutic success can be classified into two categories: open surgical repair and endovascular repair. Options for endovascular repair are stent placement (covered vs bare metal), coil embolization of the aneurysmal sac, or endovascular ICA occlusion. Open surgical repair includes carotid artery ligation, ICA bypass, and excision with reconstruction. The selection of the optimal operative approach rests on a variety of clinical variables, including comorbidities, aneurysm location, patient-specific anatomy, and etiology. An open surgical approach may be favored when the pseudoaneurysm is secondary to a carotid patch disruption or infection. Factors that may favor an endovascular approach include severe comorbidities, unfavorable neck anatomy from radiation or prior surgery, or a pseudoaneurysm related to trauma. Overall, endovascular stenting remains the primary treatment modality for ICA pseudoaneurysms. Outcomes from ICA pseudoaneurysms vary based on the treatment option pursued (nonoperative management vs endovascular repair vs open repair), aneurysm characteristics, and the patient’s condition. One study of 120 dissecting ICA pseudoaneurysms with a mean follow-up time of 29.3 months found that 3.3% of patients had recurrent transient ischemic attack, no patients had recurrent stroke, and 14.2% of patients had recurrence of nonischemic symptoms, such as headache, neck pain, or cranial nerve palsy. Of those pseudoaneurysms followed with imaging, 56% remained stable, 13.8% had enlarged, and 30.2% had healed. A total of 20.8% of patients required an endovascular or open intervention (eg, stenting, coiling, flow diversion, and/or clipping). Only 1 of 25 patients had a recurrent pseudoaneurysm, which was after stent-assisted coiling. Operative/procedural success in treating ICA pseudoaneurysm is high (>90%); however, to date, there are no randomized trials that compare open vs endovascular repairs of ICA pseudoaneurysms. Small studies have indicated that endovascular repairs may result in a lower rate of cranial nerve injury, major stroke, and 30-day mortality when compared with open repair. Zhou et al found that 30-day mortality/major stroke rates were 14% in the open repair group (n=22) vs 5% in the group that underwent endovascular repairs (n=20). Although the mortality rate may be lower in patients who undergo an endovascular repair, some device-related issues specific to the endovascular approach, such as endoleak or stent graft occlusion, have been found to occur at a rate of 6% to 8%

Conclusion
An ICA pseudoaneurysm is a rare entity that can cause serious and life-threatening complications, such as showering of emboli that lead to small infarcts and large vessel occlusion. Large infarcts may develop hemorrhagic transformation. ICA pseudoaneurysms are most commonly a result of trauma or surgery but can also be secondary to infection or connective tissue disease, and some could be associated with penetrating atherosclerotic ulcers. Approaches to treatment include both nonoperative and operative management (open vs endovascular approach) with high procedural success.

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CASE IN POINT


