Bilateral Cervical Internal Carotid Artery Pseudoaneurysms

Lucas M. Walden ▪ Jeffrey W. Prescott, MD, PhD ▪ Vikas Jain, MD

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.

AFFILIATIONS:
Radiology Department, Department of Radiology, MetroHealth Medical Center, Case Western Reserve University, Cleveland, OH

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CORRESPONDENCE:
Lucas M. Walden, MD, MetroHealth Medical Center/Case Western Reserve University, 2500 MetroHealth Dr, Cleveland, OH 44109 (jprescott@metrohealth.org)

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

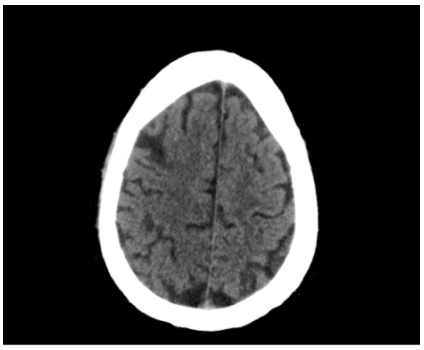


Figure 1. Noncontrast-enhanced CT of the head showing a small, hypodense lesion in the right frontal lobe suggestive of a chronic infarct.

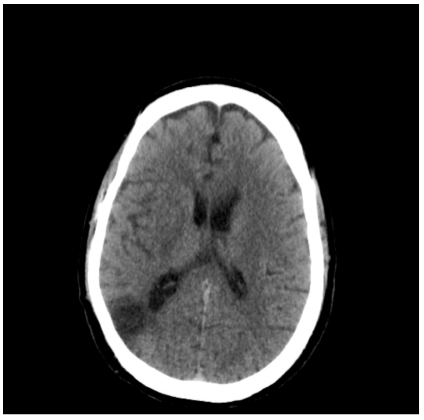


Figure 2. Noncontrast-enhanced CT of the head demonstrating right temporo-occipital lobe encephalomalacia suggestive of a chronic infarct.

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)**.

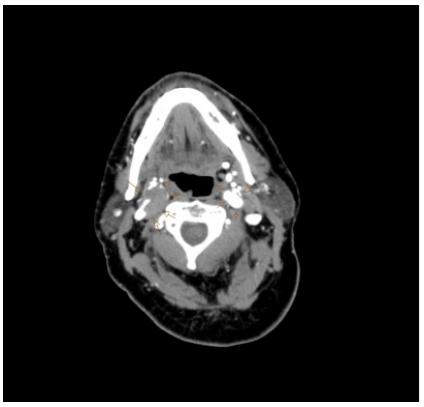


Figure 3. Axial CTA of the neck demonstrating bilateral ICA pseudoaneurysms that contain eccentric mural thrombus/atherosclerotic plaque.

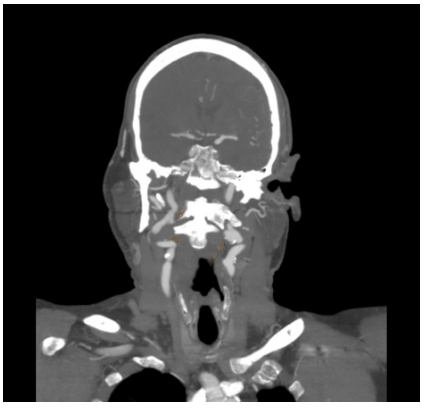


Figure 4. Coronal maximum intensity projection CTA of the neck demonstrating extensive bilateral ICA vessel tortuosity and mural thrombus/atherosclerotic plaque containing pseudoaneurysms.

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

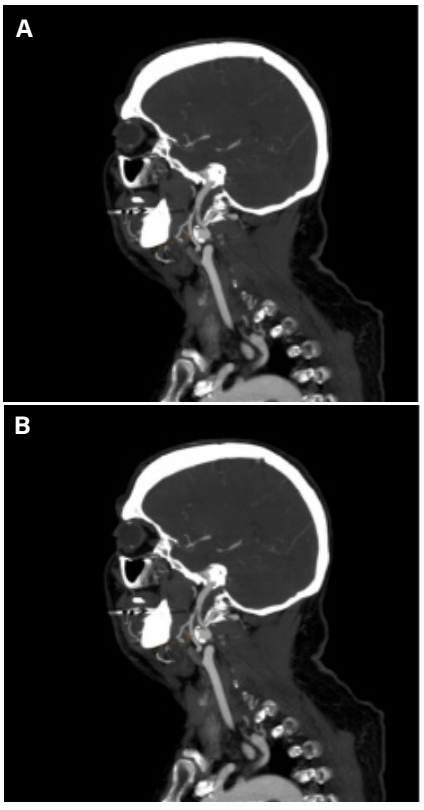


Figure 5. Sagittal CTA of the neck showing left ICA pseudoaneurysm (A) with dense atherosclerotic plaque and right ICA pseudoaneurysm (B) associated with large mural thrombus.

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

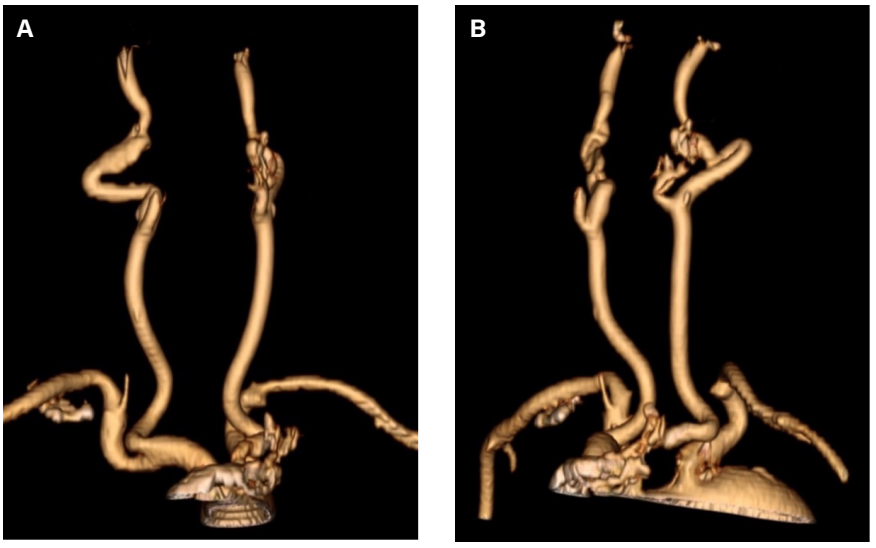


Figure 6. Three-dimensional images reconstructed using Vitrea (Canon Medical Informatics; Minnetonka, MN) and demonstrating significant ICA vessel tortuosity and mucosal irregularities associated with mural thrombus/atherosclerotic plaque containing ICA pseudoaneurysms. Image A demonstrates the left ICA anatomy. Image B demonstrates the right ICA anatomy.

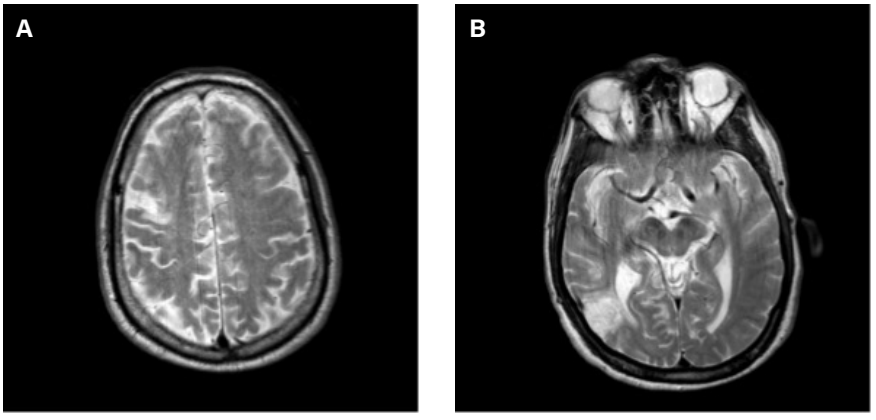


Figure 7. Axial T2-weighted images confirming chronic infarcts in the right frontal (A) and temporo-occipital (B) lobes.

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.

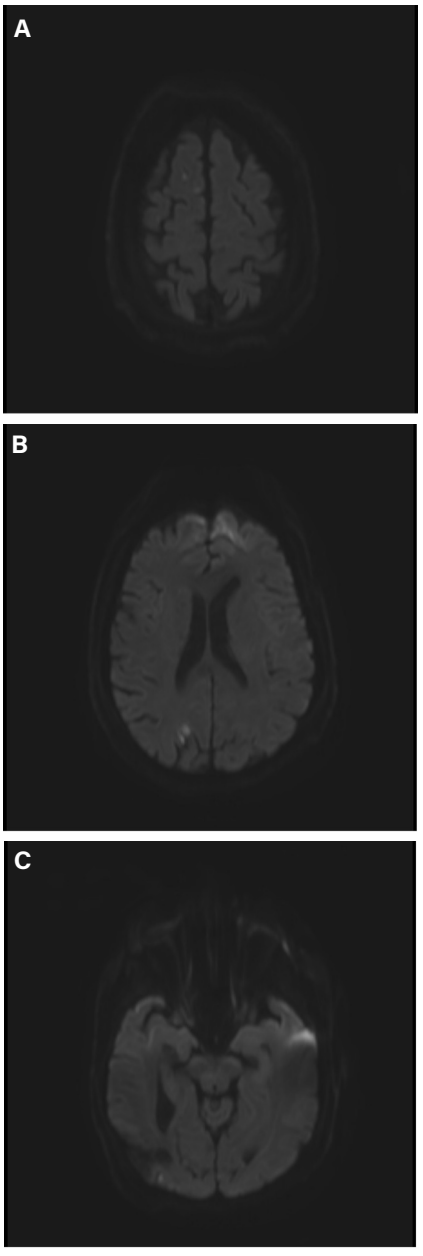
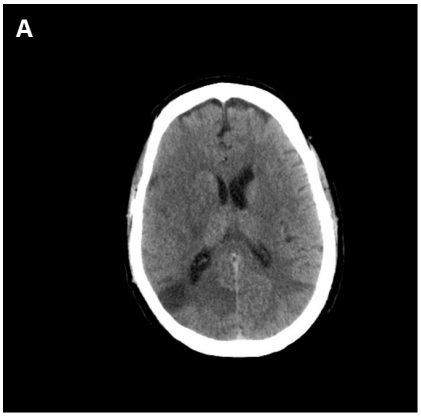


Figure 8. Diffusion-weighted imaging showing several tiny foci of diffusion restriction within the right frontal (A), parietal (B), and occipital (C) lobes likely related to showering emboli.

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



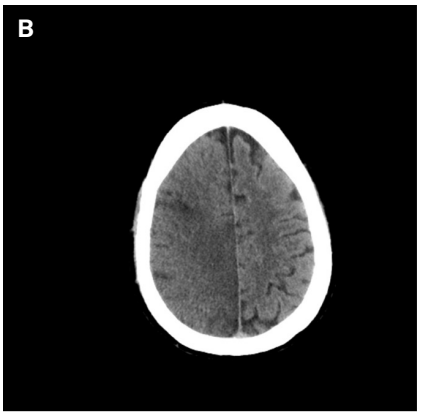


Figure 9. Axial noncontrast-enhanced CT of the head demonstrating loss of gray-white matter interface and effacement of sulcal spaces involving the right MCA and PCA territories due to acute infarct. A chronic infarct in the right temporo-occipital lobe is again seen.

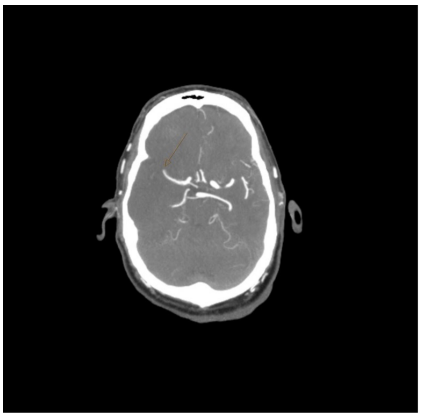


Figure 10. CTA showing abrupt cutoff of the right MCA at the bifurcation, representing an acute MCA occlusion.

and false aneurysms. True aneurysms represent full-thickness dilation of the expected vessel diameter by ≥50%.[1,2] 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.[1,2] 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.[3,4]

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.[5,6] 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).[7-10]

In other vascular beds, such as the aorta, penetrating atherosclerotic ulcers have been found to cause pseudoaneurysms through disruption of the internal elastic lamina.[11] 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.[3] Other symptoms include a painful or palpable neck mass, airway compression secondary to rupture, or neck infection or cellulitis.[3,6] Physical examination findings include focal neurologic or cranial nerve deficits, a pulsatile neck mass, or a systolic bruit.[12] 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.[6,13] Treatment decisions are based on such factors as symptoms, aneurysm size, location, and overall clinical status. Medical management

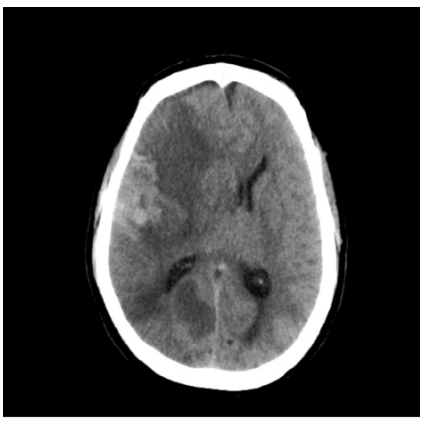


Figure 11. Axial noncontrast-enhanced CT head demonstrating hemorrhagic conversion of a large MCA territory infarct with right to left midline shift. CT, computed tomography; MCA, middle cerebral artery.

primarily involves antithrombotic therapy with antiplatelets, anticoagulants, or both.[5]

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.[5,14]

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.[5] Operative management 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.[3,15] Factors that may favor an endovascular approach include severe comorbidities, unfavorable neck anatomy from radiation or prior surgery, or a pseudoaneurysm related to trauma.[15,16] Overall, endovascular stenting remains the primary treatment modality for ICA pseudoaneurysms.[3,4,16]

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.[5] 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.[5]

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.[17] 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.[4,17] 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).[4] 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%.[17]

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.

References

1. Johnston KW, Rutherford RB, Tilson MD, Shah DM, Hollier L, Stanley JC. Suggested standards for reporting on arterial aneurysms. Subcommittee on Reporting Standards for Arterial Aneurysms, Ad Hoc Committee on Reporting Standards, Society for Vascular Surgery and North American Chapter, International Society for Cardiovascular Surgery. *J Vasc Surg.* 1991;13(3):452-458. doi:10.1067/mva.1991.26737
2. Writing Committee to Develop Clinical Data Standards for Peripheral Atherosclerotic Vascular Disease, Creager MA, Belkin M, et al. 2012 ACCF/AHA/ACR/SCAI/SIR/STS/SVM/SVN/SVS key data elements and definitions for peripheral atherosclerotic vascular disease: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Clinical Data Standards (Writing Committee to Develop Clinical Data Standards for Peripheral Atherosclerotic Vascular Disease). *Circulation.* 2012;125(2):395-467. doi:10.1161/CIR.0b013e31823299a1
3. Fankhauser GT, Stone WM, Fowl RJ, et al. Surgical and medical management of extracranial carotid artery aneurysms. *J Vasc Surg.* 2015;61(2):389-393. doi:10.1016/j.jvs.2014.07.092
4. Zhou W, Lin PH, Bush RL, et al. Carotid artery aneurysm: evolution of management over two decades. *J Vasc Surg.* 2006;43(3):493-496; discussion 497. doi:10.1016/j.jvs.2005.11.023
5. Daou B, Hammer C, Chalouhi N, et al. Dissecting pseudoaneurysms: predictors of symptom occurrence, enlargement, clinical outcome, and treatment. *J Neurosurg.* 2016;125(4):936-942. doi:10.3171/2015.10.JNS151846
6. Garg K, Rockman CB, Lee V, et al. Presentation and management of carotid artery aneurysms and pseudoaneurysms. *J Vasc Surg.* 2012;55(6):1618-1622. doi:10.1016/j.jvs.2011.12.054
7. Cafasso DE, Golarz S, Wolfe S. Endovascular treatment of bilateral carotid artery dissections progressing to pseudoaneurysms after blunt carotid injury: a case report and review of the literature. *J Vasc Surg.* 2012;56(2):584-585. doi:10.1016/j.jvs.2012.05.037
8. Németh T, Szakács L, Bella Z, Majoros V, Barzó B, Vörös E. The treatment of pseudoaneurysms with flow diverters after malignant otitis externa. *Interv Neuroradiol.* 2017 Dec;23(6):609-613. doi:10.1177/1591019917729804
9. Cohen JE, Grigoriadis S, Gomori JM. Petrous carotid artery pseudoaneurysm in bilateral carotid fibromuscular dysplasia: treatment by means of self-expanding covered stent. *Surg Neurol.* 2007;68(2):216-220; discussion 220. doi:10.1016/j.surneu.2006.08.082
10. Hospital Universitario Severo Ochoa, Rodriguez-Padilla J. Endovascular repair of radiation-induced bilateral common carotid artery stenosis and pseudoaneurysm with covered and uncovered stent graft using

double embolic protection device. *Angiol Vasc Surg.* 2021;6(1):1-4. doi:10.24966/AVS-7397/100062

11. Macura KJ, Corl FM, Fishman EK, Bluemke DA. Pathogenesis in acute aortic syndromes: aortic dissection, intramural hematoma, and penetrating atherosclerotic aortic ulcer. *AJR Am J Roentgenol.* 2003;181(2):309-316. doi:10.2214/ajr.181.2.1810309
12. Mokri B, Piepgras DG, Sundt TM, Pearson BW. Extracranial internal carotid artery aneurysms. *Mayo Clin Proc.* 1982;57(5):310-321.
13. El-Sabrout R, Cooley DA. Extracranial carotid artery aneurysms: Texas Heart Institute experience. *J Vasc Surg.* 2000;31(4):702-712. doi:10.1067/mva.2000.104101
14. Foreman PM, Griessenauer CJ, Falola M, Harrigan MR. Extracranial traumatic aneurysms due to blunt cerebrovascular injury. *J Neurosurg.* 2014;120(6):1437-1445. doi:10.3171/2014.3.JNS131959
15. Troutman DA, Mohan CR, Samhouri FA, Sohn RL. Endovascular repair of carotid artery pseudoaneurysm after carotid endarterectomy with self-expanding covered stents-a long-term follow-up. *Ann Vasc Surg.* 2010;24(7):954.e13-16. doi:10.1016/j.avsg.2010.03.020
16. Kim DY, Biffl W, Bokhari F, et al. Evaluation and management of blunt cerebrovascular injury: A practice management guideline from the Eastern Association for the Surgery of Trauma. *J Trauma Acute Care Surg.* 2020;88(6):875-887. doi:10.1097/TA.0000000000002668
17. Li Z, Chang G, Yao C, et al. Endovascular stenting of extracranial carotid artery aneurysm: a systematic review. *Eur J Vasc Endovasc Surg.* 2011;42(4):419-426. doi:10.1016/j.ejvs.2011.05.008