Congenital right subclavian artery aneurysm resection in a 30-year-old woman

Anaz Uddin, BS,[a] Steven Lu, MD,[b] Nicole Brennan, BS,[a] Jared Theriot, MD,[b] William Tracy, MD,[b] Ajit Rao, MD,[c] *and* David J. Finlay, MD,[b,c] *Valhalla and New York, NY*

ABSTRACT

Right-sided subclavian artery aneurysms (SAAs) are exceedingly rare. The most common cause of intrathoracic SAAs is atherosclerosis; however, causes can also include infection, trauma, cystic medial degeneration, Marfan syndrome, and Takayasu arteritis. Symptoms present most commonly with compression of surrounding structures, although adverse events, including rupture, thrombosis, and embolization, can also occur. We present a case of a 30-year-old woman with an asymptomatic, 15-mm, right-sided SAA, which was successfully resected with subsequent end-to-end primary anastomosis. (J Vasc Surg Cases Innov Tech 2024;10:101527.)

Keywords: Subclavian artery aneurysm; Congenital vascular malformation; Vascular surgery

CASE REPORT

The patient is a 30-year-old Bengali woman with a medical history of anxiety, gestational diabetes, an ovarian cyst found in 2020, and panic attacks. She originally presented to the emergency department for left-sided weakness and numbness. She had a history of chronic headaches and left-sided photophobia with associated nausea and vomiting but no prior episodes of hemiparesis. Her symptoms included intermittent blurry vision, dizziness, and nausea. The patient did not report cardiovascular symptoms, except for an unspecified peripartum cardiac condition 3 years previously that resolved on its own. She was evaluated by the stroke team and had imaging findings negative for acute stroke on non–contrast-enhanced computed tomography (CT), magnetic resonance imaging, and CT angiography. On CT angiography, a 13 × 12 × 15-mm asymptomatic right subclavian artery globous aneurysm, a hypoplastic right vertebral artery, a nonbifurcating right common carotid artery, and a congenitally small right hemispheric circulation were noted (Figs 1 and 2). Additionally, sclerosis of the right internal jugular vein was noted in the area of the aneurysm, which suggested prior thrombosis or inflammation. There was no evidence of tortuosity of the left subclavian artery and no evidence of compression at the thoracic outlet. She was prescribed aspirin, clopidogrel (Plavix; Sanofi Aventis US and Bristol-Myers Squibb), and atorvastatin. She was subsequently referred to the vascular surgery clinic for continuation of care. The patient provided written informed consent for the report of her case details and imaging studies.

When the patient was seen by vascular surgery, carotid duplex ultrasound was performed, which showed no significant stenosis of either carotid artery. Surgical repair of the aneurysm was recommended. The patient was amenable and presented to ambulatory surgery 1 month later for right-sided SAA repair with no interval changes in health.

In the operating room, a supraclavicular incision was used due to the high position of the aneurysm from redundancy. The right SAA was directly posterior and slightly medial to the anterior scalene muscle. The aneurysm was fully mobilized with assistance of intraoperative ultrasound guidance, and the proximal and distal subclavian artery segments were then skeletonized and looped for control.

The inferior thyroid artery was also identified and preserved, which emerged from the subclavian artery directly. After administering heparin, the aneurysm was resected, and sent for pathologic examination. The arterial ends were spatulated, and a running anastomosis was performed with 6-0 Prolene suture (Fig 3). Restoration of flow was confirmed with a Doppler probe and ultrasound, and hemostasis was achieved.

The platysma and skin were closed in layers. Blood loss was estimated at 20 mL. The patient tolerated the procedure well, had no immediate intraoperative complications, and was admitted to the surgical intensive care unit for regular postoperative monitoring (standard protocol for surgical patients at the hospital). The patient's postoperative course included challenges with nausea, dizziness, and pain, which were managed effectively in a multidisciplinary setting. The patient was discharged in stable condition on postoperative day 3. She returned to the clinic 2 weeks later doing well with minimal pain. Her pulse examination was improved from baseline but was +1 on the right vs +2 on the left. Her blood pressure was only slightly decreased on the right, with no perfusion issues.

This case highlights the importance of meticulous surgical planning and the use of intraoperative ultrasound in the management of complex vascular anomalies.

DISCUSSION

Right-sided subclavian artery aneurysms (SAAs) are exceedingly rare. The most common cause of intrathoracic SAAs is atherosclerosis; however, causes can also include infection, trauma, cystic medial degeneration,

From the New York Medical College School of Medicine, Valhalla[a]; the Department of General Surgery, Metropolitan Hospital,[b] and the Department of Vascular Surgery, Mount Sinai Hospital, New York[c].

Correspondence: Anaz Uddin, BS, New York Medical College, 40 Sunshine Cottage Rd, Valhalla, NY 10595 (e-mail: au396@nyu.edu).

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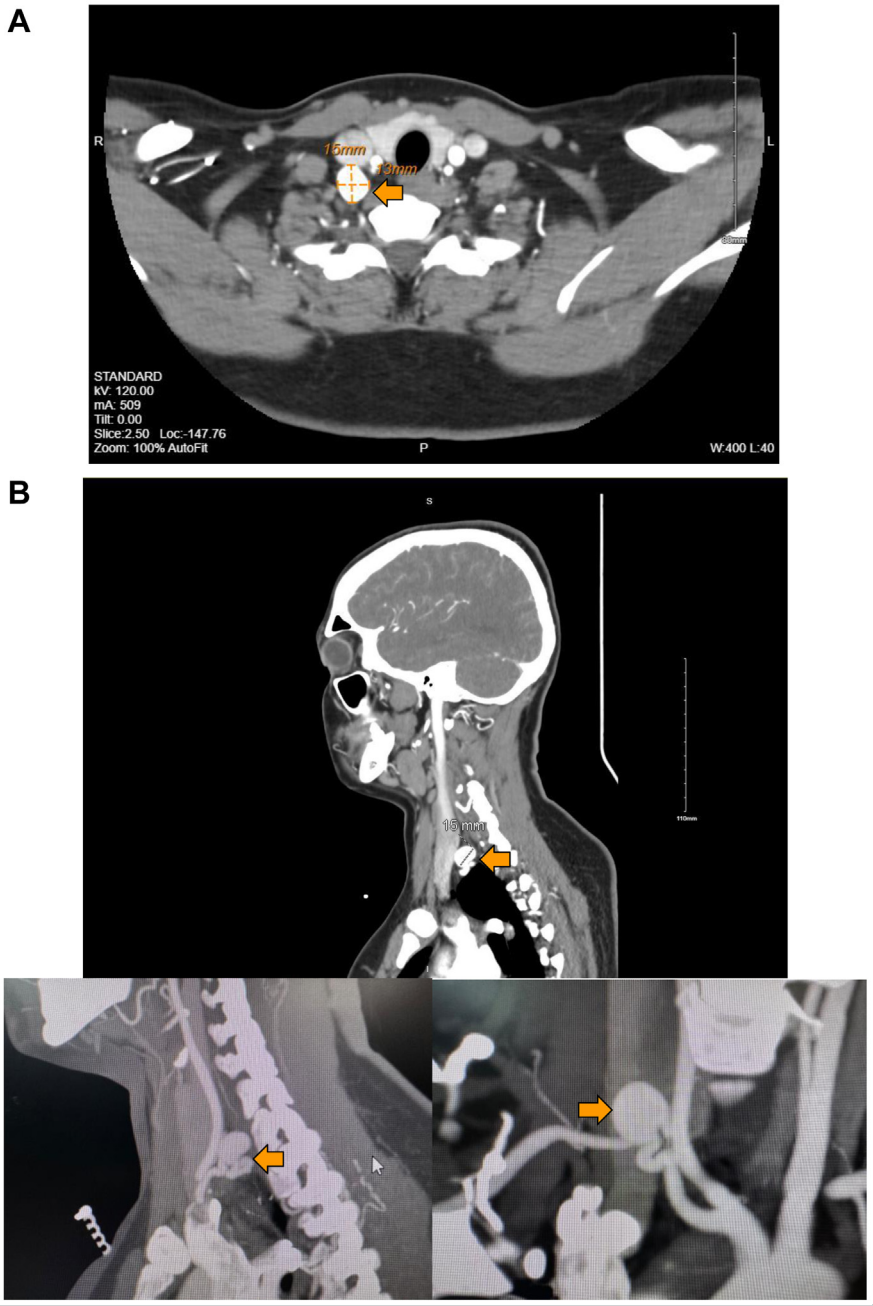

Fig 1. Computed tomography (CT) angiogram before intervention. **A,** Axial view revealing 13 × 15-mm right subclavian artery aneurysm (SAA; *orange arrow*). **B,** Sagittal view showing 15-mm, right SAA (*orange arrow*).

Marfan syndrome, and Takayasu arteritis.[1] Symptoms present most commonly with compression of surrounding structures, although adverse events, including rupture, thrombosis, and embolization, can also occur.[2] One percent of all peripheral aneurysms are SAAs, a serious condition with adverse outcomes, including rupture if not appropriately treated.[3] In this report, we present the case of a 30-year-old woman with an asymptomatic 15-mm fusiform SAA. According to a 2010 meta-analysis of 394 SAAs conducted by Vierhout et al,[4] the median diameter for an SAA is 40 mm (range, 10-180 mm), and the mean age of patients with SAAs is 52.2 years. A recent 2021 study by Sun et al[3] examining 22 true, isolated SAA cases without aberrancy found a mean diameter of SAAs of 30.0 ± 17.6 mm and an average age of 53.5 ± 14.3 years, consistent with the findings reported by Vierhout et al.[4] Although our patient's age and SAA diameter are also consistent with existing literature, both are at the lower end of both ranges. The diagnosis is often incidental, especially in asymptomatic

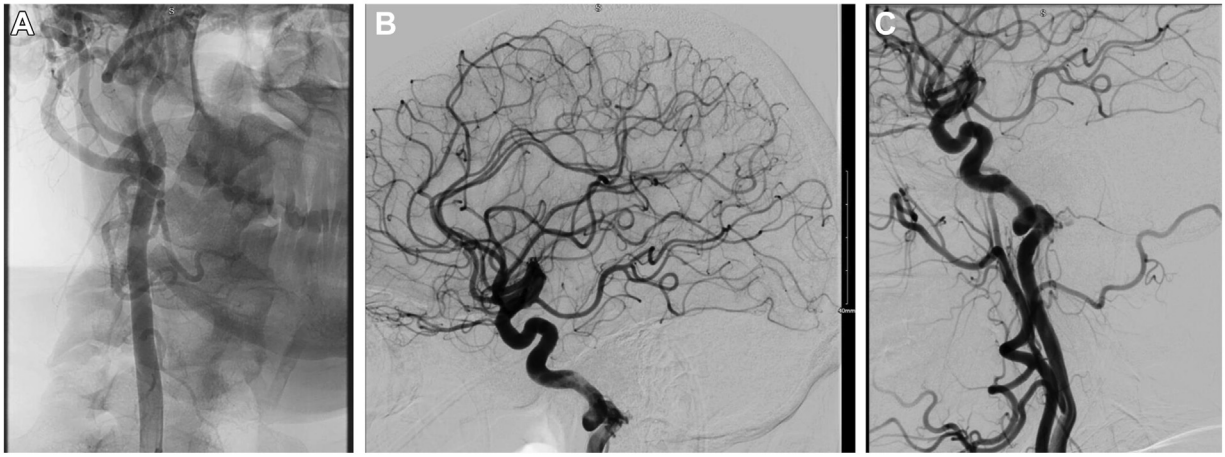

Fig 2. Congenital vascular abnormalities as seen on interventional radiology angiography. **A,** Coronal view revealing right common carotid artery (CCA) bifurcation at the superior C1 level. **B,C,** Sagittal view revealing hypoplastic anterior and posterior cerebral circulation.

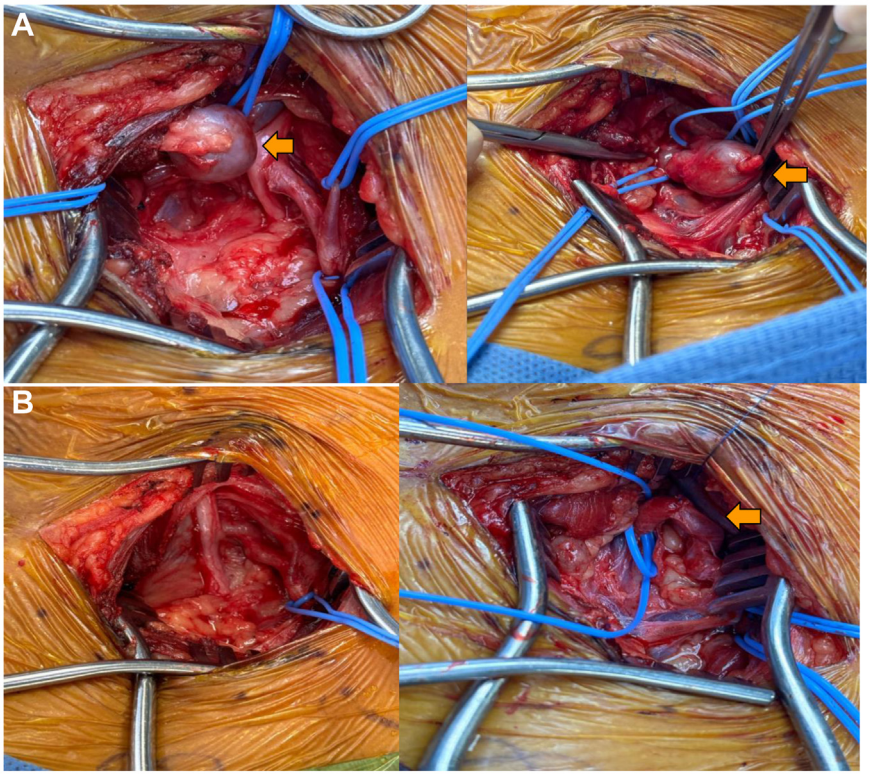

Fig 3. Operative image of right subclavian artery with inferior thyroid artery. **A,** Intraoperative photograph. *Orange arrow* indicates right subclavian artery aneurysm (SAA). **B,** Postoperative photograph of right SAA.

patients. Symptomatic cases can vary in presentation according to the SAA size and position (ie, palpable aneurysms, neurological deficits).

Our patient also has other uncommon vascular abnormalities, including a hypoplastic right vertebral artery, a nonbifurcating right common carotid artery, and congenitally small right hemispheric circulation. The right external carotid artery (ECA) branches extend from the right common carotid artery (CCA) to the superior C1 level. The right CCA bifurcates at the superior C1 level. The petrous segment of the right internal carotid artery (ICA) is hypoplastic compared with the left ICA. Normal bifurcation of the left CCA is seen at the superior C6 vertebral body. The patient had hypoplastic anterior and posterior cerebral circulation and evidence of a fetal posterior cerebral artery. Current literature related to

these abnormalities is limited mostly to case reports. Vertebral arteries are the primary source of blood to the structures of the posterior brain, and hypoplasia of these arteries can contribute to posterior circulation ischemia.[5] According to Chuang et al,[6] the frequency of this variation was reported to be 2% to 6% from autopsies and angiograms.

The finding of a nonbifurcating CCA is also rare, with the literature currently expressing two hypotheses for its generation. Nishizawa et al[7] theorized that it might be the result of agenesis of the proximal aspect of the ICA, with anastomosis of the distal ECA to the distal ICA. Seidel[8] describes it as agenesis of the ECA, with an arterial stump at the expected bifurcation level and all the ECA branches coming from the ICA. Uchino et al[9] reported a retrospective study of non–contrast-enhanced magnetic resonance angiograms and found six cases of nonbifurcating carotid arteries of 2866 patients, for a 0.2% incidence rate in a Japanese population. In conclusion, the vascular abnormalities found in our patient, in addition to the SAA, are also rare, with the current literature mainly consisting of case reports.

The indications to treat SAAs include the risk of embolization, thrombosis, and rupture, which can lead to catastrophic outcomes.[4] SAAs can also cause ipsilateral upper limb symptoms through compression of the brachial plexus. Ipsilateral facial neuralgia could stem from carotid artery or persistent trigeminal artery variants, although such symptoms were not observed in our patient.[10,11]

Challenges for this SAA include a particularly tortuous aneurysm and anatomical variations requiring thoughtful planning in preparation for surgical repair. A supraclavicular approach is conventional for accessing the middle segment of the subclavian artery, although an infraclavicular incision, clavicular separation, and restricted clavicular resection can be supplementary.[4] The preferred method of care for SAA repair is open surgery with an end-to-end anastomosis, although this is often difficult due to the lack of length. In our patient, there was a good amount of redundancy, which allowed for primary anastomosis and a supraclavicular approach. Usually, a transposition or a graft is required.[12-15] Endovascular repair of SAAs has also been documented in the literature, although this technique raises concerns for adverse outcomes due to aortic wall fragility, especially in patients with Marfan syndrome.[10,16] An endovascular approach using stent grafts has the advantages of being minimally invasive, a shorter length of stay in the hospital, and faster recovery. However, such an approach is less feasible for tortuous vessels, can result in a higher incidence of postprocedure complications (ie, endoleak) requiring reintervention, could require lifelong imaging follow-up, and fewer data are available on long-term durability compared with an open approach.[17] A hybrid approach can be deployed when purely open or endovascular approaches are not suitable; however, this also combines the risk of both methods, requires coordination, is highly technical, and is not available at all medical centers.[18] Furthermore, Vierhout et al[4] found comparable rates of complications and mortality between open and endovascular approaches. Ultimately, endovascular and hybrid approaches were not viable options for our patient due to the redundancy and tortuosity. We expressed concerned for an increased risk of rupture and embolization of our patient's SAA. However, currently, few data are available on the risk of rupture of a congenital fusiform aneurysm in the existing literature. For asymptomatic patients, the decision to perform surgery vs careful observation will depend on the diameter of the aneurysm and any changes found on serial imaging studies or follow-up. Future studies should explore the incidence and outcomes for congenital SAAs to help guide and elucidate clinical management.

CONCLUSIONS

A congenital fusiform right SAA is a rare finding that warrants repair due to the risks of rupture and possible thrombosis and/or embolization. This unique case illustrates the need for good planning to allow for a minimal supraclavicular approach with primary anastomosis. Intraoperative ultrasound can be a useful adjunct to accurately map patient vascular anatomy in the setting of aneurysm repair.

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DISCLOSURES

None.

REFERENCES

1. Salem A, Salem S, Moustafa S. Surgical treatment of subclavian artery aneurysms. *Zagazig University Medical Journal*. 2021;27:1582–1591.
2. Ramtoola MT, Bhatti M, Shetty R. A case report of a ruptured subclavian artery aneurysm presenting to the emergency department. *Clin Case Rep*. 2019;7:797–800.
3. Sun J, Qi H, Shi Y, et al. Isolated true subclavian aneurysm without aberrant subclavian artery or coarctation of descending aorta. *Ann Vasc Surg*. 2021;75:294–300.
4. Vierhout BP, Zeebregts CJ, van den Dungen JJAM, Reijnen MMPJ. Changing profiles of diagnostic and treatment options in subclavian artery aneurysms. *Eur J Vasc Endovasc Surg*. 2010;40:27–34.
5. Dinç Y, Özpar R, Emir B, Hakyemez B, Bakar M. Vertebral artery hypoplasia as an independent risk factor of posterior circulation atherosclerosis and ischemic stroke. *Medicine (Baltim)*. 2021;100:e27280.
6. Chuang YM, Chan L, Wu HM, Lee SP, Chu YT. The clinical relevance of vertebral artery hypoplasia. *Acta Neurol Taiwan*. 2012;21:1–7.
7. Nishizawa S, Yamamoto S, Sugiura Y, Uemura K. [Agenesis of the internal carotid artery with an unusual collateral pathway]. *Noshinkeigeka*. 1986;14:1011–1015.
8. Seidel K. [Arteriographic observation of a rare carotis anomaly]. *Fortschr Geb Rontgenstr Nuklearmed*. 1965;103:390–391.

9. Uchino A, Saito N, Watadani T, Mizukoshi W, Nakajima R. Non-bifurcating cervical carotid artery diagnosed by MR angiography. *AJNR Am J Neuroradiol*. 2011;32:1119–1122.
10. Domoto S, Asakura T, Nakazawa K, Niinami H. Hybrid repair of multiple subclavian and axillary artery aneurysms in a patient with Marfan syndrome. *J Vasc Surg*. 2018;68:255.
11. Ling MM, Gupta M, Acharya J. Trigeminal neuralgia associated with a variant of persistent trigeminal artery. *Radiology Case Reports*. 2020;15:2225–2228.
12. Davidović LB, Marković DM, Pejkić SD, Kovacević NS, Colić MM, Dorić PM. Subclavian artery aneurysms. *Asian J Surg*. 2003;26:7–11. discussion: 2.
13. Yap E, Patel ST. Bilateral subclavian artery aneurysms in a patient with Marfan syndrome. *J Vasc Surg*. 2022;75:1760–1761.
14. Morimoto K, Matsuda H, Fukuda T, et al. Hybrid repair of proximal subclavian artery aneurysm. *Ann Vasc Dis*. 2015;8:87–92.
15. Andersen ND, Barfield ME, Hanna JM, et al. Intrathoracic subclavian artery aneurysm repair in the thoracic endovascular aortic repair era. *J Vasc Surg*. 2013;57:915–925.
16. Rose JF, Lucas LC, Bui TD, Mills JL. Endovascular treatment of ruptured axillary and large internal mammary artery aneurysms in a patient with Marfan syndrome. *J Vasc Surg*. 2011;53:478–482.
17. Toor SM, Sazonov I, Luckraz H, Nithiarasu P, eds. *Aortic aneurysms: OSR, EVAR, stent-grafts, migration and endoleak—current state of the art and analysis*. 2013.
18. Welleweerd JC, Moll FL, de Borst GJ. Technical options for the treatment of extracranial carotid aneurysms. *Expert Rev Cardiovasc Ther*. 2012;10:925–931.

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