Atypical presentation of subclavian steal syndrome with left sided sensorineural deafness

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ABSTRACT

We present a rare manifestation of a common pathology: left sided sensorineural hearing loss secondary to subclavian steal syndrome after thoracic endovascular aortic repair for complicated acute aortic dissection. We describe the vascular physiology that can result in unilateral hearing loss and provide a brief review of subclavian steal syndrome. This case report highlights the importance of avid clinical recognition of an atypical presentation of a common vascular disease. (J Vasc Surg Cases Innov Tech 2023;9:101308.)

Keywords: Aortic dissection; Sensorineural deafness; Subclavian steal; TEVAR

CASE REPORT

A 52-year-old man with a history of uncontrolled hypertension, hyperlipidemia, and 20 pack-year smoking history presented to the emergency department with acute mid-sternal tearing chest pain radiating to the back. He was hypertensive to 170s/90s mm Hg, with normal cardiac findings. On physical examination, the abdomen was benign, and four-extremity vascular examination revealed palpable pulses without motor or sensory loss. Computed tomography angiography (CTA) revealed a type B2,6 aortic dissection, with a pseudoaneurysm of the distal bovine arch and codominant vertebral arteries at the V1 segments.[1] The entry tear was located just distal to the left subclavian artery (LSA) takeoff and extended down to the level of the celiac trunk. He was initially managed medically with nicardipine and esmolol infusion for anti-impulse therapy and systolic blood pressure and heart rate goals of <120 mm Hg and <70 bpm, respectively. However, because of radiographic evidence of an aortic arch pseudoaneurysm with a risk of rupture, he subsequently underwent zone 2 thoracic endovascular aortic repair (TEVAR) with aortic stent graft coverage of the LSA (Figs 1 and 2) and the PETTICOAT (provisional extension to induce complete attachment) technique with placement of bare metal aortic dissection stents to the level of the superior mesenteric artery.[2] Surgical intervention was performed within 12 hours of admission after optimization of his heart rate and blood pressure. The patient provided written informed consent for the report of his case details and imaging studies.

The index TEVAR procedure was performed under general anesthesia, with percutaneous access of the right and left common femoral arteries using micropuncture and the preclosure technique for large bore access. We used a 32 × 142-mm Zenith TX2 dissection endovascular graft (Cook Medical Inc) as the main body dissection stent graft. This was advanced from the right side and deployed at the level of the brachiocephalic and left common carotid artery common trunk, resulting in coverage of the LSA. Coverage was extended distally using two overlapping Zenith bare metal dissection stents (Cook Medical Inc). The proximal stent measured 36 × 180 mm and the distal stent 36 × 80 mm, with deployment just proximal to the renal arteries. Intravascular ultrasound was used before and after thoracic aortic repair. TEVAR sizing was performed using standard CTA in multiple projections and confirmed using centerline imaging on a separate three-dimensional workstation using TeraRecon software. Completion angiography revealed exclusion of the origin of the dissection and normal antegrade celiac trunk, superior mesenteric artery, and bilateral renal artery filling. Retrograde flow was present from the left vertebral artery to the LSA (Fig 3). Because of the urgency of the procedure, the primary goal of the initial surgical intervention was to prevent aortic rupture; thus, the decision was made to not revascularize the LSA at the time of the TEVAR.

Within 24 hours postoperatively, he developed left sided upper extremity paresthesia, claudication, sensorineural hearing loss, dizziness, and transient vision loss with left arm use. Physical examination revealed a left arm blood pressure of 98/62 mm Hg vs a right arm blood pressure of 132/64 mm Hg, and the lack of a radial artery pulse with a monophasic radial artery Doppler signal. CTA was also obtained, which ruled out a retrograde type A dissection and confirmed location of the aortic endograft and coverage of the LSA origin (Fig 4). Thus, subclavian steal syndrome (SSS) was diagnosed clinically.

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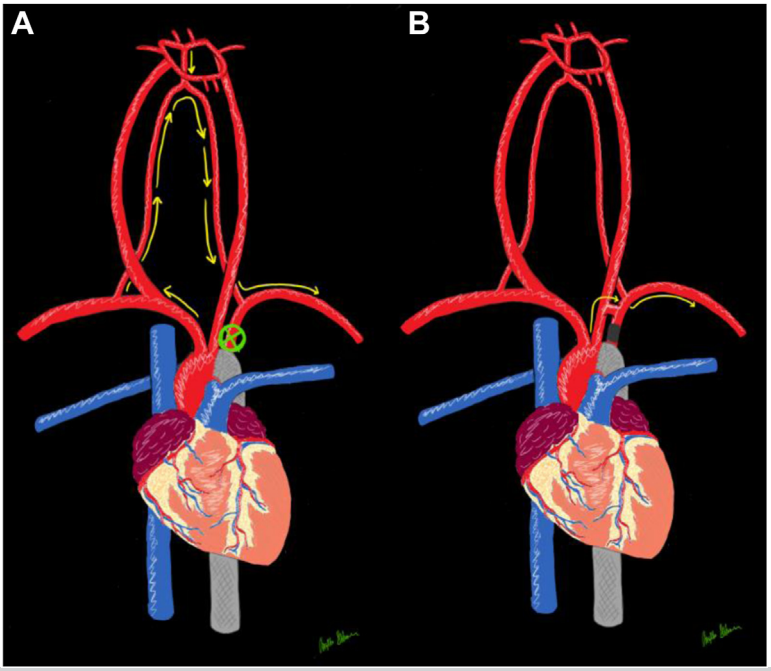

Fig 1. A, Depiction of subclavian steal syndrome (SSS) as it presented in our patient, with blood flow drawn to the left subclavian artery (LSA) from the contralateral vertebral artery and basilar artery. **B,** Drawing showing flow of blood after the bypass procedure directly into the ipsilateral common carotid artery, through the bypass, and into the LSA.

The patient underwent subsequent left carotid artery to subclavian artery bypass with an 8-mm polytetrafluoroethylene graft and LSA embolization. The LSA was accessed via the hood of the distal anastomosis, and angiography with the bypass patent revealed thrombus at the proximal LSA and antegrade flow in the vertebral artery (Fig 5). With the carotid–subclavian artery bypass clamped, retrograde flow was noted through the vertebral artery, as expected. The proximal LSA was embolized with a 12 × 8-mm Amplatzer plug (Abbott Laboratories). Immediately after surgery, his visual and auditory disturbances and left upper extremity paresthesia had resolved, and his bilateral upper extremity blood pressure was equal. He was discharged home on postoperative day 2, with once-daily aspirin of 81 mg, atorvastatin of 40 mg, and a four-drug oral antihypertensive regimen. At 2 years after the procedure, he remains without residual left arm pain, paresthesia, or deafness. He continues his aspirin and statin medications and a two-drug antihypertensive regimen (amlodipine and metoprolol).

DISCUSSION

SSS results from an inadequate upper extremity blood supply via the subclavian artery. An increased oxygen demand results in compensatory vascular recruitment by retrograde flow through the ipsilateral vertebral artery. Although subclavian steal is asymptomatic in most patients, peripheral symptoms include upper extremity paresthesia, claudication, weakness, and loss of muscle mass, resulting in SSS. Central manifestations can include confusion, dizziness, orthostasis, and, rarely,

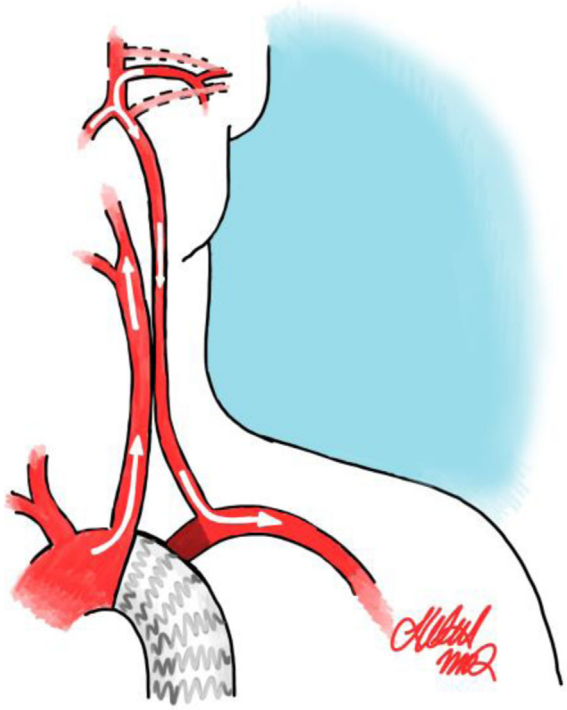

Fig 2. Reversal of flow through the vertebral artery in the setting of thoracic endovascular aortic repair (TEVAR). The labyrinthine artery is illustrated as it comes off the anterior inferior cerebellar artery. *Dotted lines* at the basilar and vertebral arteries depict possible anatomic variations.

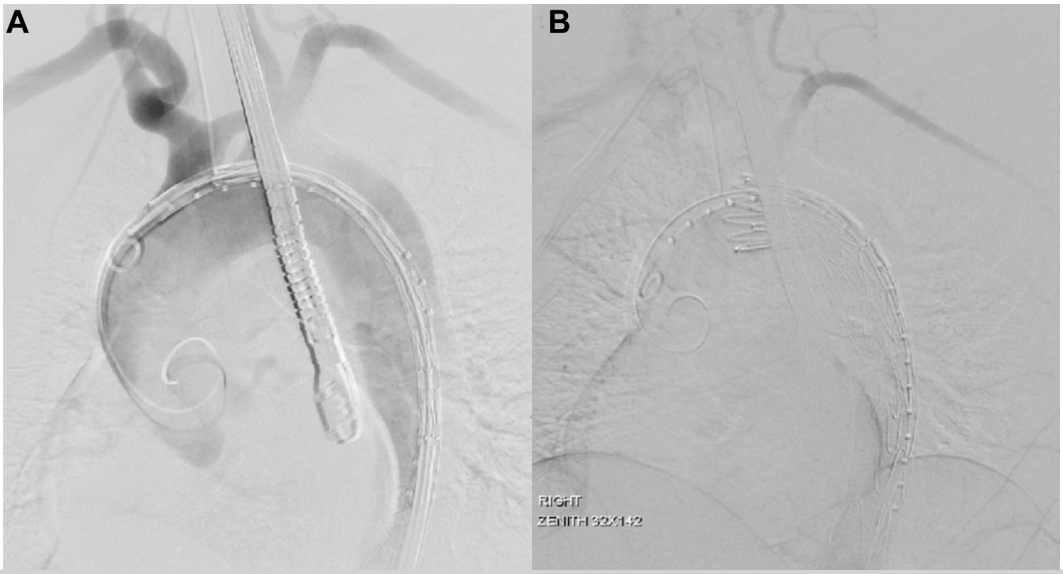

Fig 3. Intraoperative angiography demonstrating antegrade left subclavian artery (LSA) flow before stent deployment (**A**) and retrograde flow via the ipsilateral vertebral artery after stent deployment (**B**).

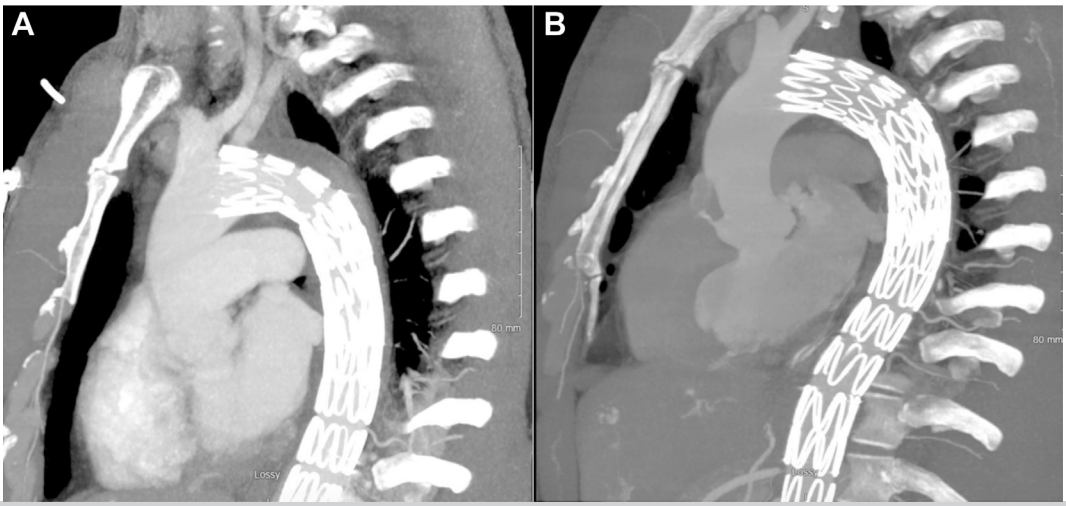

Fig 4. A, Computed tomography angiography (CTA) of the chest, abdomen, and pelvis after stent placement during thoracic endovascular aortic repair (TEVAR) demonstrating left subclavian artery (LSA) ostial coverage and bovine arch anatomy. **B,** CTA of chest, abdomen, and pelvis at 2 years of follow-up showing stable appearance of aortic endograft.

visual and/or auditory symptoms.[3-7] SSS most commonly results secondary to atherosclerotic disease and less commonly to aortic dissection, arteritis, anatomic anomalies, and iatrogenic insult.[8,9] The prevalence of SSS is poorly documented, given its vastly asymptomatic nature; however, the incidence has most recently been reported at ~5%.[10-12]

The prevalence of SSS after TEVAR varies widely in the literature, estimated at 4% to 38%.[13-15] One prospective study evaluated the short-term outcomes of complete LSA coverage after TEVAR and found that 11 of 55 patients subsequently developed symptomatic SSS.[14] The timing of symptom onset varied widely between these patients—within 1 month after TEVAR and as late as 6 months after endovascular intervention. In that study, symptoms related to SSS were successfully managed medically without revascularization.[14] One prior study investigated eight patients with LSA coverage after TEVAR, three of whom developed SSS.[15] One patient underwent elective carotid to subclavian artery bypass,

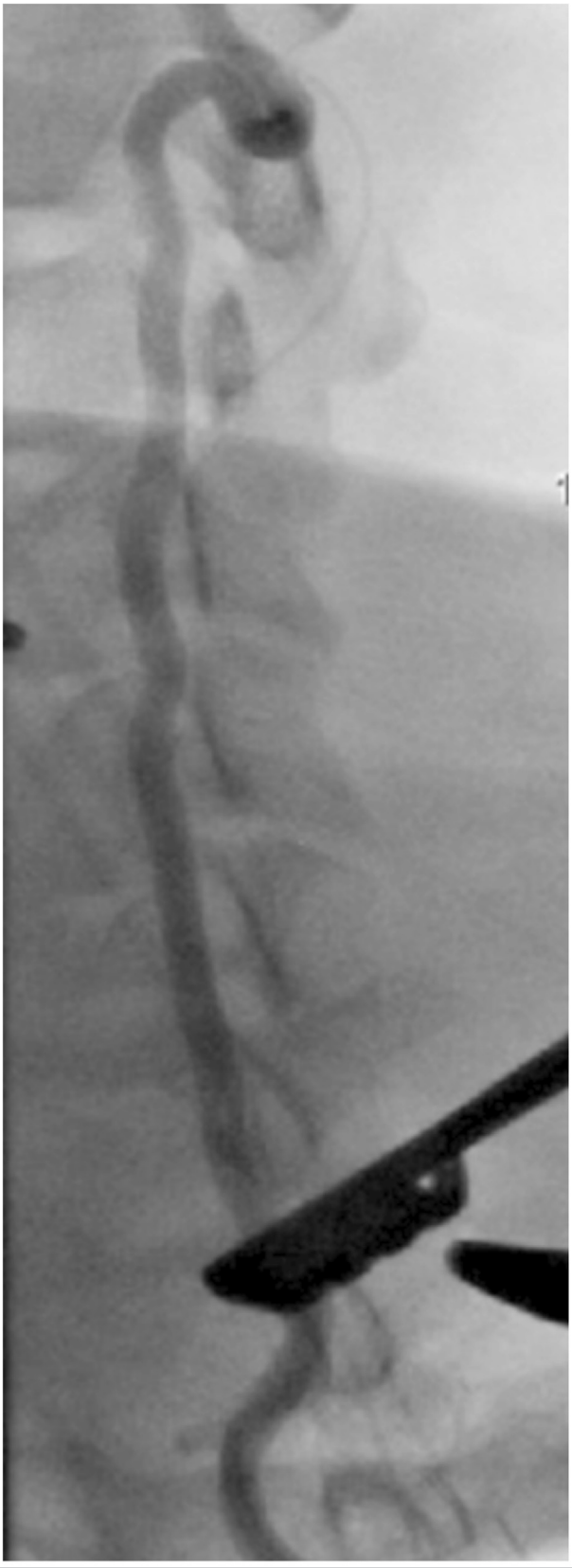

Fig 5. Intraoperative angiography demonstrating antegrade left vertebral artery flow after carotid artery to subclavian artery bypass.

and two other patients were managed medically in accordance with patient preference. A third study described eight patients who underwent LSA coverage as part of TEVAR.[13] Only one patient developed symptomatic SSS with subsequent elective revascularization.[13] In patients requiring surgical intervention for SSS, subsequent revascularization led to resolution of the patients' symptoms with little patient morbidity. Overall, the prevalence of SSS after TEVAR varies greatly in the literature, likely a result of patient heterogeneity and sparse observational data.[16] In the small subset of patients who develop symptomatic SSS, many have been managed successfully by either conservative medical treatment or subsequent elective revascularization. Thus, although intentional LSA coverage during TEVAR is deemed safe,[16-18] no definitive consensus has been reached for its management.[18-21]

Management of the LSA during TEVAR varies greatly within the literature.[22] Some studies have proposed prophylactic revascularization before TEVAR in all patients. This was thought to minimize the risk of acute limb ischemia, cerebrovascular insufficiency secondary to reversal of flow at the ipsilateral vertebral artery, and spinal cord ischemia resulting from decreased perfusion through the anterior spinal circulation.[23-26] Others have proposed only conditional LSA revascularization. In particular, LSA coverage is deemed safe if patent flow is present at the ipsilateral vertebral artery, with communication between the anterior and posterior circulation via the posterior communicating artery. However, prophylactic revascularization has been recommended for patients with a dominant left vertebral artery and concomitant contralateral vertebral artery stenosis or an occluded ipsilateral internal carotid artery.[27-29] Others have recommended against LSA revascularization during TEVAR. Specifically, although the incidence of upper extremity symptoms after LSA coverage is estimated at 21%, only 5% of these patients require revascularization.[30] Furthermore, most of these patients can be treated nonemergently via stenting. Additionally, prior studies that proposed a risk of stroke and/or cerebrovascular insufficiency after LSA coverage lacked sufficient power to reach statistical significance.[16,31,32] In these patients, the etiology of the cerebrovascular complications was more likely multifactorial.

In our patient, TEVAR with intentional LSA coverage resulted in acute-onset postoperative SSS with predominantly auditory symptoms. Auditory symptoms as a manifestation of SSS has been reported secondary to vertebrobasilar insufficiency. When the collateral flow to the posterior fossa through the posterior communicating artery is insufficient, retrograde vertebral artery circulation during SSS will manifest with vertebrobasilar symptoms, such as hearing loss. Vertebrobasilar insufficiency can also result from carotid artery disease or microvascular disease within the circle of Willis.

CONCLUSIONS

This report outlines a patient case with immediate onset of SSS after TEVAR with intentional LSA coverage. To the best of our knowledge, the presence of sensorineural hearing loss is unique to our case report. The cessation of symptoms after left carotid to subclavian bypass implies innate vertebrobasilar insufficiency in our patient. Also indicated by our findings is the necessity to consider occlusion of the LSA as the cause of a vast array of neurologic symptoms, including hearing loss.

DISCLOSURES

None.

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