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Retrograde aortic thrombosis with TEVAR for secondary stroke prevention: Case report

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ABSTRACT

Proximal aorta thrombi and atheromas have been identified as the source of cardioembolic ischemic stroke. Thrombi and atheromas distal to the carotid and subclavian origins have been overlooked as possible sources; however, descending thoracic aorta thrombi can embolize to the cerebral vasculature during diastolic retrograde aortic flow during diastole. Retrograde aortic flow is described and is capable of reaching the common carotid arteries. This phenomenon has recently been recognized for its stroke potential. Additionally, a complex thrombus in the descending aorta increases the retrograde diastolic flow with potential for these thrombi to cause ischemic stroke. Patients with stroke are more likely to have descending aortic thrombi than patients without stroke. Appropriate strategy for prevention of cardioembolic strokes from descending aortic atheroma is unclear. This case report describes a descending aortic thrombus as the likely etiology of a left middle cerebral artery and limb ischemia followed by successful secondary stroke prevention with thoracic endovascular aortic repair (TEVAR). Only one previous case report describes cardioembolic stroke with thrombus originating from a descending aortic atheroma. Endovascular aortic interventions have not been previously described for prevention of strokes in the setting of aortic atheroma.

1. Introduction

Retrograde aortic blood flow is more common than previously appreciated and can occur in a variety of circumstances. It was previously thought to be attributable to aortic valve insufficiency, but more recent studies have failed to find this correlation [1,2]. Retrograde flow from the descending aorta to the origin of the left subclavian artery is present in 98% of healthy subjects [3]. An examination of 94 patients with known aortic plaques with transesophageal echocardiogram (TEE) and magnetic resonance imaging (MRI) revealed flow reversal that reached the left common carotid artery nearly half, and about 25% had flow reversal that reached the brachiocephalic trunk [2]. Specifically, retrograde flow is capable of reaching the brachiocephalic trunk from 4 cm downstream of the descending aorta, and it is capable of reaching the left subclavian artery from 5 cm downstream of the descending aorta [3].

There is clear evidence that complex plaques in the descending aorta are both capable and likely causal of ischemic stroke previously thought to be cryptogenic. In an evaluation of 56 patients, transesophogeal echocardiogram (TEE) demonstrated diastolic retrograde aortic blood flow in all patients, and diastolic retrograde flow was higher in patients with atherosclerotic plaque, particularly in the aortic arch [4]. In a larger sample of 485 patients with acute ischemic stroke, TEE with 2D pulse-wave Doppler ultrasound demonstrated retrograde blood flow in the proximal descending aorta all patients evaluated. Additionally, patients with concomitant complex plaques or large aortic diameter had increased retrograde blood flow [5]. Age, aortic atherosclerosis, and decreased heart rate may also increase retrograde aortic flow [3,2]. It is therefore suggested that this blood flow is capable of causing cerebral emboli.

While the correlation of stroke and descending aortic atheroma is well described, the association remains unclear. Complex atheromatous plaques in the descending aorta have a 4-fold increase in patients with stroke as compared to patients without stroke. However, the risk factors for aortic atheromas and stroke are the same [1]. Only one previous case postulated recurrent posterior circulation embolic strokes stroke from retrograde thromboembolism from the descending thoracic aorta [6].

The treatment of complex atheromas is unclear. A prospective open

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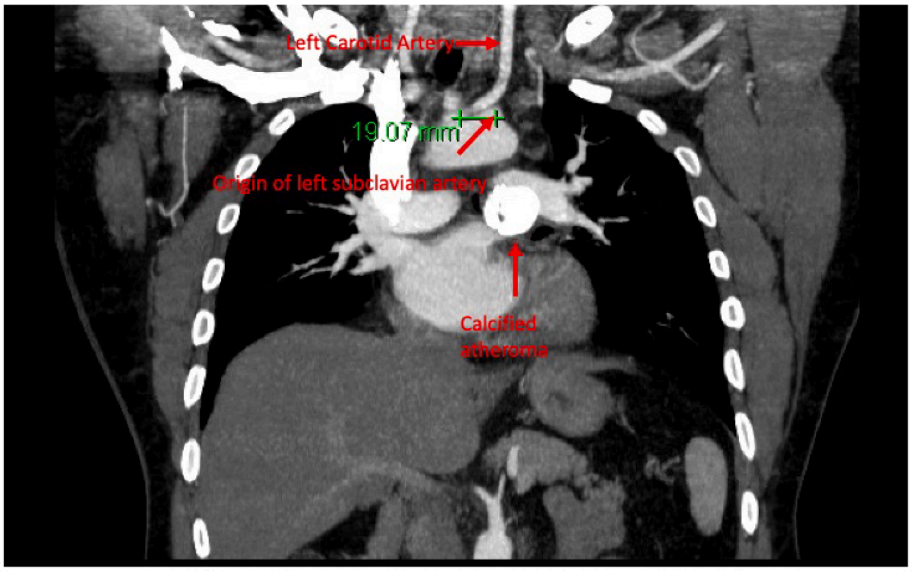

Fig. 1. Large calcified aortic atheroma distal to the origin of the left carotid and subclavian arteries.

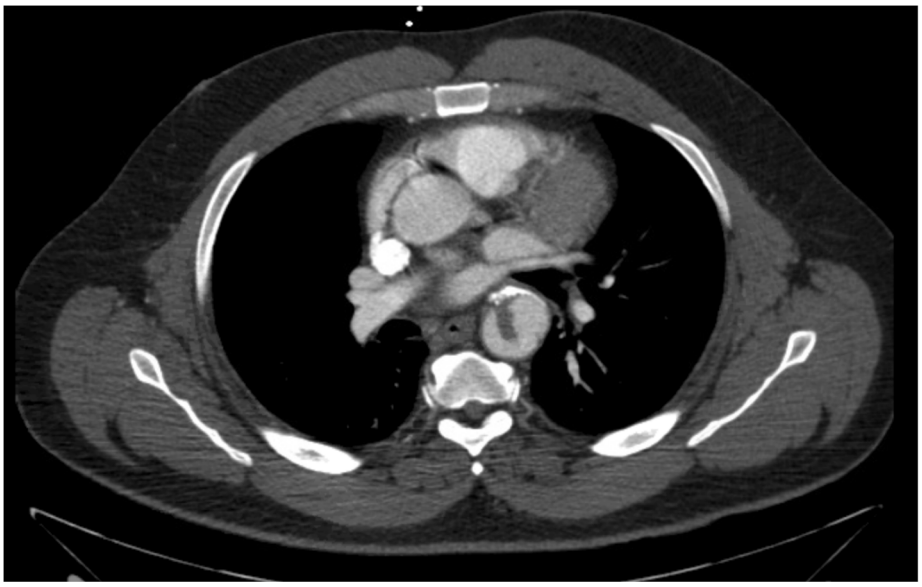

Fig. 2. Aortic atheroma and thrombus < 4 cm from the origin of the left subclavian artery.

label randomized control trial failed to demonstrate a difference between patients treated with aspirin and clopidogrel versus those treated with warfarin [7]. Statins remain a mainstay for all patients with atherosclerotic disease and have demonstrated regression of plaques [8]. Open thrombectomy has been described in the setting of large aortic thrombus [9].

Thoracic endovascular aortic repair (TEVAR) is a relatively new procedure and is now commonly used for type 2 aortic dissections, aortic aneurysms, intramural hematomas and ulcerative plaques, and aortic transections in the trauma setting [10–12].

2. Case report

A 44-year-old African American man with a past medical history of untreated hypertension presented with left sided facial droop and aphasia. He received intravenous tissue plasminogen activator which improved his symptoms. Initial MRI revealed wedge shaped infarcts in multiple vascular territories, consistent with likely cardioembolic etiology. Hospital complications included non-ST segment myocardial infarction. Cardiac catheterization demonstrated subacute occlusion of the proximal right coronary artery. Descending aortic thrombus was identified via transthoracic echocardiogram (TTE) and confirmed with transesophageal echocardiogram (TEE). TTE revealed a reduced left ventricular ejection fraction (LVEF 20–25%). No patent foramen ovale was found. Computer tomography angiogram (CTA) of the chest demonstrated descending aorta pseudoaneurysm with maximum diameter measuring 3.8 cm with anterior intimal calcification as well as multiple filling defects indicating partial thrombosis. A large pedunculated mass was found in the transverse and descending aorta 3.5 cm distal to the origin of the left subclavian artery and 5.5 cm from the origin of the left common carotid (Figs. 1 and 2).

The patient was anticoagulated with heparin infusion. The suspicion of a retrograde emboli to the carotids from the aortic atheroma was high considering that no other source of emboli was found on workup.

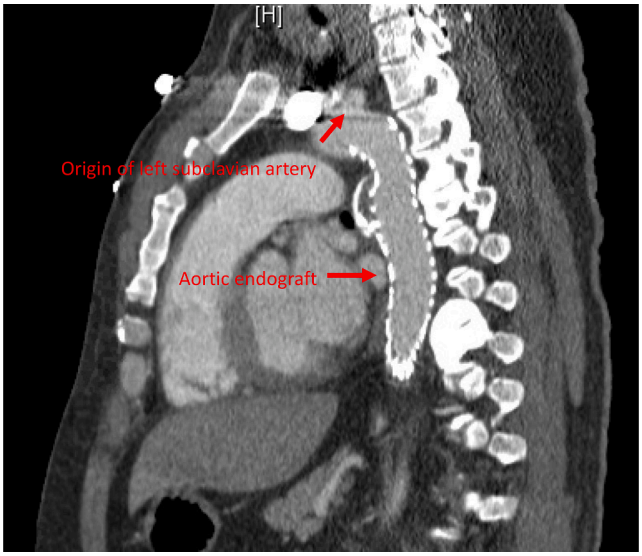

Fig. 3. Aortic endograft placed distal to the origin of the subclavian artery.

Decision was made to perform an elective TEVAR for exclusion of the aortic thrombus and prevention of further embolization. However, two days prior to the date of surgery, embolization to the lower extremity occurred, causing acute limb ischemia and requiring an emergent intervention with open left lower extremity thrombectomy removing a large thrombus from the artery.

Thoracic endovascular aortic repair (TEVAR) was performed. The patient had a bovine arch with the distance from the bovine trunk origin to aneurysm of 5 cm, the distance from the bovine trunk origin to the pedunculated thrombus was 7 cm. The pseudoaneurysm had calcification and thrombi within it. Care was taken to manipulate the wire on the outer curvature of the aorta, avoiding the thrombus on the inner curvature. The left subclavian was marked with angiography after performing an arch angiogram, manually without the power injector, to make sure not too much pressure was instilled within the aorta to avoid disrupting more thrombi and decreasing the chance of embolization. Embolic protection devices were not deemed necessary. The diameter of the descending aorta was 26 mm proximal to the thrombus-containing segment and 21 mm distal to it. Since there was a proximal seal zone of 3 cm, a 28 mmx150mm GORE CTAG thoracic endograft was used without oversizing.

He tolerated the procedures without further complications. Repeat CTA of his chest, abdomen and pelvis showed complete exclusion of aortic thrombus (Fig. 3). He progressed well and was discharged three days post operation. At one year follow up he remained on medical management with antiplatelets and statin and has had no additional ischemic events including stroke, spontaneous recovery of his LVEF to 60–65%, and a modified Rankin score of two.

3. Discussion

We describe a case of stroke likely associated with descending aortic thrombus located at the upper limit of known retrograde flow. Acute limb ischemia proved he failed initial medical management with heparin. While his long-term treatment included medical management with aspirin, clopidogrel, and statin, additional endovascular procedure of TEVAR halted further thromboembolic events for at least one-year post procedure. Limitations include other possible causes of cardioembolic sources of stroke but are less likely. Atrial fibrillation was not captured while he was admitted in the hospital. Transthoracic echocardiogram did not demonstrate intracardiac thrombus. Secondary stroke prevention for most cardioembolic sources is anticoagulation. He did not have additional strokes after TEVAR.

This case highlights retrograde embolization from thrombus overlying a descending aortic atherosclerotic plaque as a possible source of cardioembolic strokes. TEVAR may be beneficial for this rare event as a means of secondary stroke prevention.

CRediT authorship contribution statement

Taylor Stiegler: Conceptualization, Writing – original draft. **Tashfin Huq:** Conceptualization, Data curation. **Justin Salerian:** Conceptualization, Methodology. **Nicolas Zea:** Writing - review & editing, Visualization. **Aimee Aysenne:** Writing - review & editing, Supervision.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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