Extensive Thoracic Aortic-Arterial Embolization: Imaging, Treatment, and Repair

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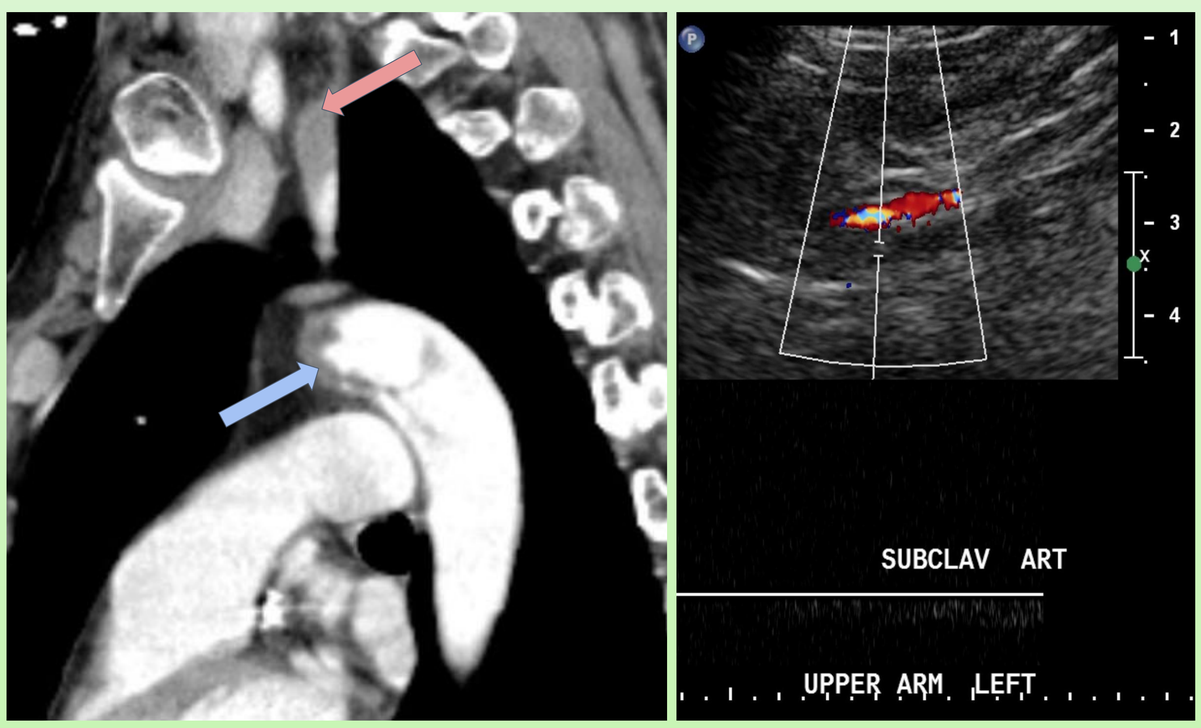
BACKGROUND

A 52 year old male with past medical history of diabetes mellitus II, hypertension, hyperlipidemia, chronic obstructive pulmonary disease, coronary artery disease status post two myocardial infarctions and multiple stents presented to the Emergency Department with acute onset left upper extremity hand and forearm weakness with tingling. He denied any similar symptoms in the past and stated that this did not feel anything like one of his previous heart attacks. Due to financial reasons, he stated that he has not taken any medication in months. His medications included antiplatelets such as aspirin and clopidogrel, as well as atorvastatin, a cholesterol-lowering drug that also exerts anti-inflammatory effects on blood vessels. He is a current, one pack-per-day smoker for the past forty years.

Ultrasound and Computed Tomography (CT) imaging discovered an occlusion of his left subclavian artery and an irregular mural thrombus of his distal aortic arch. On physical exam, he had no doppler pulses to his left brachial, radial, or ulnar arteries. He underwent emergent left brachial artery cutdown with thromboembolectomy, which resulted in removal of the occlusion and resolution of his symptoms. However, the irregular lesion of his aortic arch was likely the source of the occluding thrombus. This was addressed with placement of a Gore thoracic stent graft. After being discharged from the hospital, he was initially lost to follow-up for several months, but eventually was seen in the vascular clinic. At the time of his last visit, he denied any extremity pain or numbness. He was still smoking, but had cut down to half a pack-per-day. He also stated that he was being medication compliant with the aspirin, clopidogrel, and atorvastatin.



Left: Sagittal view of a CT pulmonary angiogram at the level of the aortic arch with (blue arrow) indicating irregular, hypodense thoracic aortic lesion. There is also thrombus occluding the left subclavian artery in this image (red arrow).
Right: Duplex Ultrasound of the left upper arm shows no detectable flow within the left subclavian artery.

IMAGING

Duplex Ultrasound (US) was initially performed, which demonstrated lack of flow compatible with occlusion of the left proximal subclavian artery. A small amount of flow was seen reconstituting at the distal axillary artery.

CT pulmonary angiogram (CTPA) was subsequently performed, which showed irregular mural thrombus involving the distal aortic arch. A long segment occlusion involving the left subclavian artery was noted, with reconstitution at the left axillary artery. The left brachial artery appeared patent, but radial and ulnar arteries are not visualized.

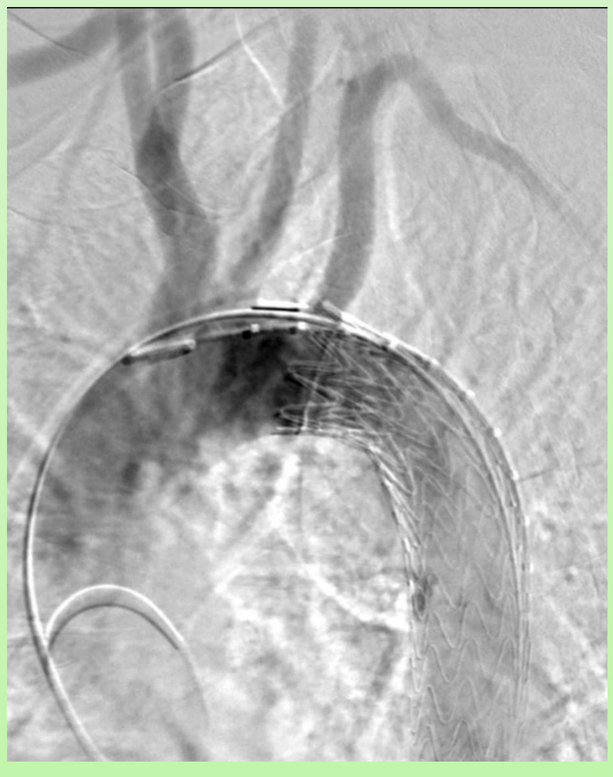
The irregular mural thrombus was implicated as the source of the left subclavian occlusion on the basis of showering emboli.

DISCUSSION

The term “atheroembolism” refers to the migration of fragments from an atheroscelrotic plaque or debris from platelets that may result in partial or complete obstruction of a downstream arterial vessel. Atheroembolization originating from the thoracic aorta is being recognized more and more often as a source of systemic emboli. Due to their central origin, these emboli can result in significant morbidity and mortality related to limb loss, strokes, and visceral ischemia.
In particular, the thoracic aorta is identified as the embolic source in approximately 10-15% of cases. The recurrence rate of systemic emboli in patients who undergo medical therapy alone is estimated at 50%, with most recurrent embolic events involving renal ischemia.

Thoracic atheroembolism can occur spontaneously, as with the described patient case. They can also complicate endovascular procedures or any operations involving manipulation of the aortic arch, which carry a risk of dislodging plaque or debris. The standard for diagnosis in suspected aortic-arterial high risk plaque or thrombus is contrast-enhanced CTA or transesophageal echocardiography. There are limited studies available to define standard of care, however, exclusion of the lesion is generally required for the associated risks of distal embolization. Treatment typically begins with anticoagulation and antiplatelet therapy followed by endovascular or surgical intervention with subsequent long term anticoagulation.

Once a thoracic lesion is identified, an endovascular approach to treatment should reduce instrument manipulation within the vessel lumen as much as possible, due to the risk of dislodging plaque and creating emboli. Devices such as the Gore thoracic endograft are useful in these situations because they are deployed from their central portion outward, which reduces the risk of lesion disruption. Routine follow-up is at the discretion of the provider due to lack of consensus data, but yearly contrast-enhanced CT to evaluate for new lesions or thrombus is generally practiced.



Digital Subtraction Angiography (DSA) of the aortic arch following placement of Gore thoracic endograft. This stent was placed over the area of irregular mural thrombus, excluding it from circulation and reducing the risk of atheroembolism from this source.

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