Common carotid artery pseudoaneurysm secondary to *Mycobacterium tuberculosis* treated with resection and reconstruction with saphenous vein graft

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ABSTRACT

Extracranial carotid artery aneurysms secondary to *Mycobacterium tuberculosis* infection are exceedingly rare. Despite an uncommon location and offending pathogen, the treatment paradigm follows that of all mycotic aneurysms. We report the case of a right common carotid artery pseudoaneurysm caused by a tuberculous infection, successfully treated with antibiotics, resection, and autologous interposition graft. (J Vasc Surg Cases and Innovative Techniques 2017;3:192-5.)

Extracranial carotid artery aneurysms (ECCAs) are uncommon, with the majority of cases being secondary to atherosclerosis, trauma, or prior endarterectomy.[1-3] Infection is a much more infrequent cause, with only a few case reports in the literature.[4,5] These mycotic aneurysms are morbid conditions associated with a wide variety of complications and death if prompt and proper management is not provided. *Mycobacterium* is an extremely rare pathogen associated with ECCA.[6,7] Conventional management consists of antituberculosis therapy combined with aggressive surgical débridement and possible reconstruction based on the anatomic involvement. We present a case of a right common carotid artery (CCA) aneurysm secondary to tuberculosis infection from perivascular *Mycobacterium* lymphadenitis. The patient gave consent to the use of her clinical history and images for this case report.

CASE REPORT

A 55-year-old Chinese woman with recent travel back to China presented to us with an enlarging right-sided neck mass, odynophagia, and hoarseness. Two months before, she was diagnosed with extrapulmonary tuberculosis in the form of lymphadenitis by a right supraclavicular lymph node biopsy. She was started on supervised rifampin, isoniazid, pyrazinamide, and pyridoxine. On physical examination, she was thin with a body mass index of 19.5 kg/m^2. There was a pulsatile mass in the right anterior neck just above the clavicle measuring about 6 cm in diameter. Her voice was hoarse, but the rest of her cranial nerve examination was grossly intact. Computed tomography (CT) angiography revealed a 5 $\times$ 5-cm pseudoaneurysm of the proximal right CCA (Fig 1). A review of her previous CT scan performed 2.5 months earlier showed lymphadenopathy encasing an ectatic CCA (Fig 2). Of note, the excisional biopsy was performed on a superficial node away from the carotid artery and not thought to be related to the pseudoaneurysm. Based on her history, examination, and imaging, our diagnosis was a mycotic aneurysm secondary to tuberculosis infection, and surgical repair was recommended.

The case was performed under general anesthesia with electroencephalography and somatosensory evoked potential monitoring. A hemisternotomy was performed with extension up the right side of the neck. The innominate artery, right subclavian artery, and distal CCA were all isolated. The pseudoaneurysm was dissected sharply from the surrounding tissue and resected. The internal jugular vein and vagus nerve were incorporated into the wall of the pseudoaneurysm and therefore required sacrifice with our dissection (Fig 3, *A*). The specimen was opened longitudinally on the back table and a smooth, 12-mm defect was noted on the lateral wall (Fig 3, *B*). The left great saphenous vein was harvested, reversed, and used as a 6-cm-long interposition graft from the proximal to distal CCA (Fig 4). The postoperative course was unremarkable, and she was discharged home on postoperative day 7. The antituberculosis regimen was continued for 6 months, and aspirin 81 mg was added. The pathologic examination of the specimen revealed granulomatous inflammation of the arterial wall consistent with a *Mycobacterium* infection. The acid-fast bacillus culture had no growth, and the Gram stain did not reveal any organisms.

The patient's dysphagia completely resolved before discharge. The hoarseness gradually improved and was barely detectable at the 1-year follow-up visit. She also had transient facial fullness, which resolved by the 6-month follow-up visit. Duplex ultrasound examination at 1 month, 6 months, and 12 months showed the reconstruction to be widely patent.

DISCUSSION

Mycobacterium tuberculosis infection is an exceptionally uncommon cause of ECCAs, although the true

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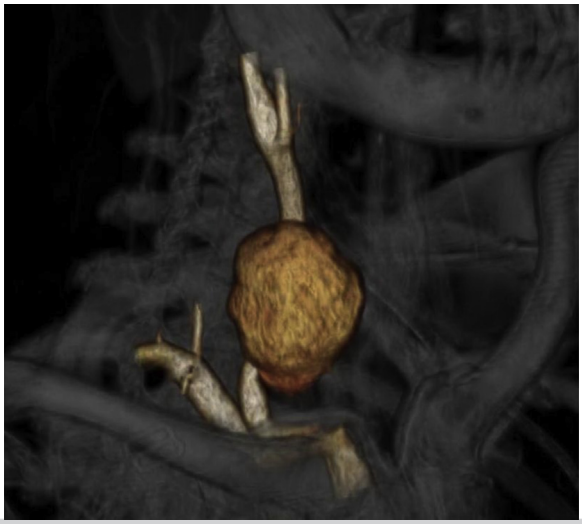

Fig 1. Three-dimensional reconstruction of computed tomography (CT) angiogram showing 5-cm pseudoaneurysm of the right common carotid artery (CCA).

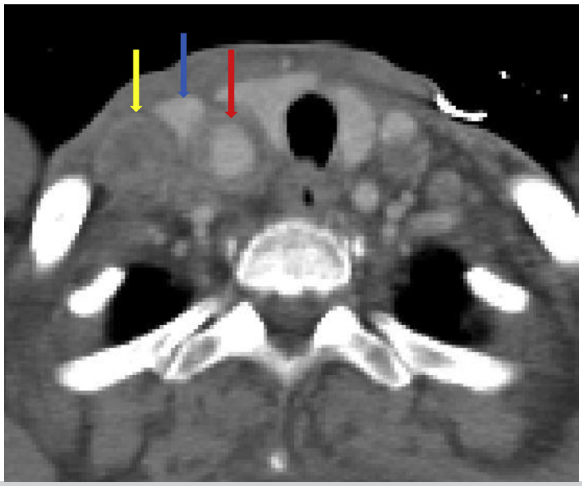

Fig 2. Computed tomography (CT) scan from 2.5 months before presentation, showing cervical lymphadenopathy (*yellow arrow*), internal jugular vein (*blue arrow*), and an ectatic common carotid artery (CCA; *red arrow*).

incidence is unknown. The most common bacterial pathogens associated with mycotic carotid aneurysms are *Staphylococcus*, *Streptococcus*, and *Salmonella*.[5,8,9] These same organisms are responsible for the majority of mycotic aneurysms at all anatomic locations.[10] Of the mycobacteria associated with arterial infections, *M. tuberculosis* is the most widely reported. However, other species of *Mycobacterium* are known to cause mycotic aneurysms. For example, there are multiple reports of *Mycobacterium bovis* resulting in mycotic aneurysms after intravesical bacillus Calmette-Guérin therapy for bladder carcinoma.[11,12]

Various mechanisms of the arterial infection by mycobacteria have been described: hematogenous seeding that leads to direct intimal colonization; implantation of mycobacteria on the adventitia or media through the vasa vasorum; and local vascular extension from adjacent infected structures, such as lymph nodes.[13] In our case, we suspect the pathologic process was a direct extension of the infection from tubercular lymphadenitis, considering that the lymph nodes adjacent to the CCA were biopsied and positive for tuberculosis (Fig 2). Regardless of the mechanism of infection or specific pathogen, the inflammatory process weakens the arterial wall, leading to aneurysmal degeneration, and may result in embolization or rupture.

Targeted antibiotic therapy should be started as soon as possible for any tuberculous mycotic aneurysm. Both medical and surgical therapy is necessary for acceptable outcomes. In tuberculous mycotic aneurysms of the aorta, Long et al reported 100% mortality if patients received no treatment, surgical therapy alone, or medical therapy alone.[13] As with any arterial infection, resection and débridement of all infected tissue are required, with or without revascularization. In situ reconstruction with autologous conduit seems optimal. On occasion, a focal defect without circumferential involvement of the artery wall can be repaired with patch angioplasty.[6] Surgical repair with the use of prosthetic conduits along with medical management in appropriately selected patients has demonstrated acceptable outcomes; but without medical management, recurrence rates have been shown to be high.[11,14] Finally, ligation of the carotid may be necessary when reconstruction is not possible but should be considered as a last resort because it carries a 25% mortality.[5]

Endovascular repair with stent graft has been reported as a treatment option for mycotic aneurysms. In general, this has been used as a temporizing maneuver for ruptures or in otherwise high-risk patients who are not candidates for open surgery.[6,15] Caution should be taken, as Labrousse et al pointed out in their treatment of a tuberculous thoracic aortic aneurysm with an endovascular stent graft. Four months after stopping antituberculosis medications, there was recurrence of infection, which led to aortic rupture and death.[16] For our patient, the dysphagia was caused by mass effect and would not have been relieved by stent graft alone.

A mini-sternotomy was used to gain proximal control. In retrospect, the excision likely could have been performed from a neck incision. However, this was not clear preoperatively, and we did not want to risk inadvertent rupture of the pseudoaneurysm from our dissection before having vascular control. Therefore, we chose the safer, more conservative approach. Consideration was also given to obtaining

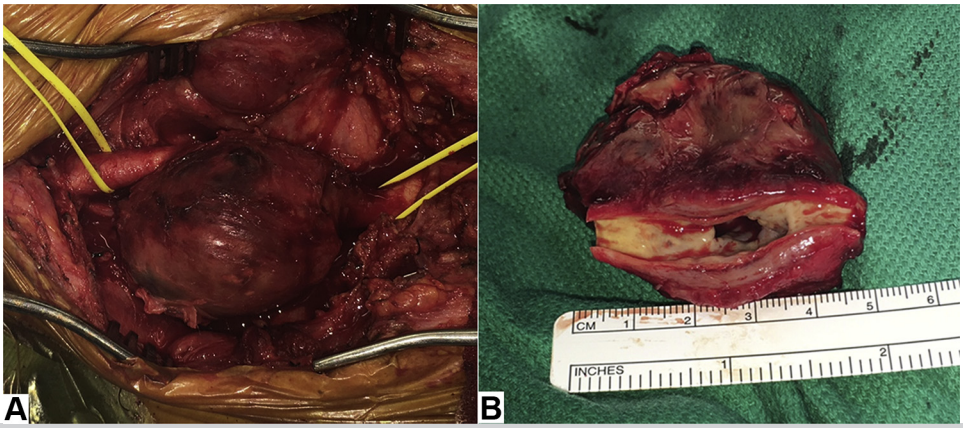

Fig 3. A, Right common carotid artery (CCA) pseudoaneurysm with vessel loops on proximal and distal CCA (head to left, feet to right). **B,** CCA opened longitudinally with defect leading to pseudoaneurysm.

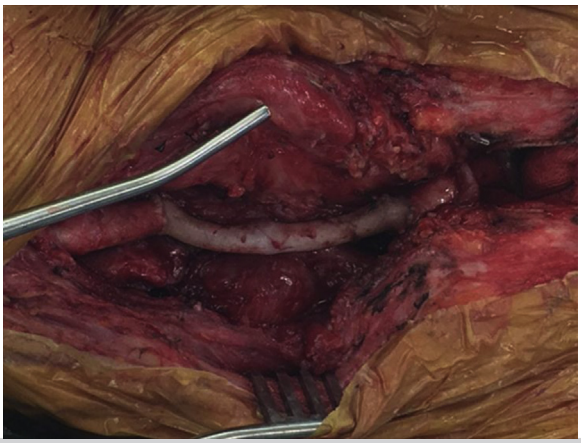

Fig 4. Completed repair, common carotid artery (CCA)-CCA interposition graft with reversed saphenous vein.

endovascular control, but we questioned the quality of the adjacent artery to accommodate an occlusion balloon. The Gram stain and culture from the pseudoaneurysm wall did not show any organisms, probably influenced by the 2.5 months of antituberculosis therapy before surgery. Several other cases of suspected tuberculous arterial infections have been reported with negative microbiology results.[17,18] Six months of therapy was recommended by our infectious disease consultant as this is the standard treatment for extrapulmonary tuberculosis. Follow-up out to 12 months has demonstrated excellent results.

CONCLUSIONS

ECCA secondary to *M. tuberculosis* infection is exceedingly rare, and clinical suspicion is warranted in patients with a neck mass and history of tuberculosis or recent travel to areas where tuberculosis is prevalent. Optimal management of infected ECCAs includes timely resection, débridement, reconstruction with autologous conduit, and targeted antimicrobial therapy.

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