Giant Cell Arteritis as the Cause of Amaurosis Fugax in a Patient with Significant Bilateral Internal Carotid Artery Disease

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Background

Amaurosis fugax (AF) occurring in a patient with severe carotid artery stenosis ipsilateral to the side of the vision abnormality is typically considered a case of symptomatic carotid stenosis. In this context, without a known central embolic source or diagnosis of atrial fibrillation, carotid artery disease is typically the most likely etiology of this transient visual disturbance. In a risk appropriate patient, carotid endarterectomy is the recommended treatment.

However, Giant Cell Arteritis (GCA) is another possible cause of AF. In a study from the United Kingdom from 2014, the prevalence of GCA on diagnosis per American College of Rheumatology criteria was found to be 0.25% in patients older than 55 years of age. Among patients with GCA and some form of ocular complaints, the literature has demonstrated that around 32% of these patients will specifically complain of AF. Symptomatic carotid disease is a much more common cause of AF overall, but the case presented here demonstrates that GCA is another possible AF etiology.

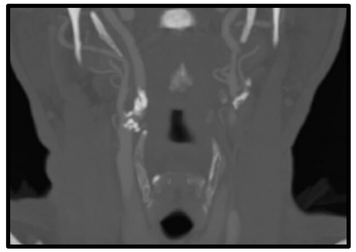
Initial Case Presentation

Here we have an 81-year-old active male who presented with acute onset of complete transient loss of vision in the right eye. He described a recent likely viral illness with some nasal congestion and mild associated headaches. Initial workup included an EKG that did not show atrial fibrillation and a computed tomography angiography (CTA) study of the head and neck that showed greater than 70% stenosis of the right internal carotid artery with a heavily calcified plaque burden. In the context of symptoms of AF, a right carotid endarterectomy was performed for a likely symptomatic carotid lesion.

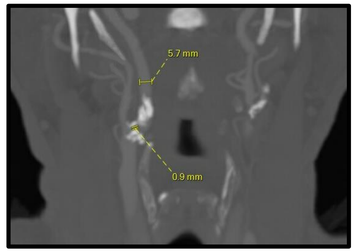
Right Carotid Endarterectomy

The right carotid endarterectomy was performed under general anesthesia with EEG monitoring. Tissues and plaque appeared typical for a classic case of carotid stenosis. The artery was repaired with a bovine patch in standard fashion. The vessel wall was not noted to be unusually thickened or inflamed.

CTA Imaging



Heavily calcified right carotid artery bifurcation and proximal internal carotid artery origin



Estimated 84.2 % stenosis ((5.7 mm – 0.9mm)/5.7mm)x100

Complicated Postoperative Course

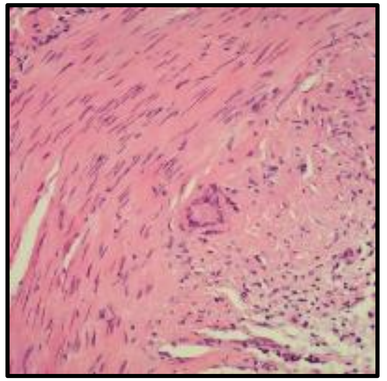
Within a week after discharge he was discovered to have atrial fibrillation based on a cardiology remote evaluation of data from his pacemaker and after discussion with vascular surgery he was anticoagulated for management of this new diagnosis of atrial fibrillation. An echocardiogram was also performed with no central embolic source found and a normal ejection fraction (EF) of around 60%.

And then, two days following initiation of anticoagulation, he had an another episode of temporary vision loss in the right eye similar the event he had prior to right carotid endarterectomy. He was subsequently readmitted and on workup was recognized to be at risk for a GCA etiology to his symptoms. At this time he was describing associated right sided jaw pain and right sided temporal headaches. Laboratory studies showed elevated inflammatory markers.

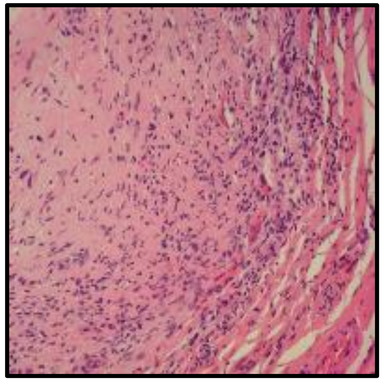
CRP Trend				
Days from Readmission	0	2	12	21
CRP	4.3	4.9	0.2	<0.1

Diagnosis and Management of GCA

The patient was started empirically on steroids and underwent temporal artery biopsy. Intraoperative duplex of the temporal artery and direct inspection both demonstrated wall thickening and inflammatory changes. An immediate frozen section was therefore performed and this confirmed GCA. Contralateral temporal artery biopsy was avoided given these findings. The patient was given a full course of steroids. He has had a few more minor vision loss episodes after discharge, but is felt to be optimized per rheumatology with normalized ESR and CRP and is overall progressing well.



An example of a giant cell



Lymphocyte infiltration observed in the arterial wall

Conclusion

This is an interesting case that demonstrates the multiple potential causes of amaurosis fugax and the varying treatments, all of which were eventually undertaken in this individual.