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Ipsilateral Pupillary Dilation Following Carotid Endarterectomy: A Case Series, Review of the Literature, and Proposed Etiology

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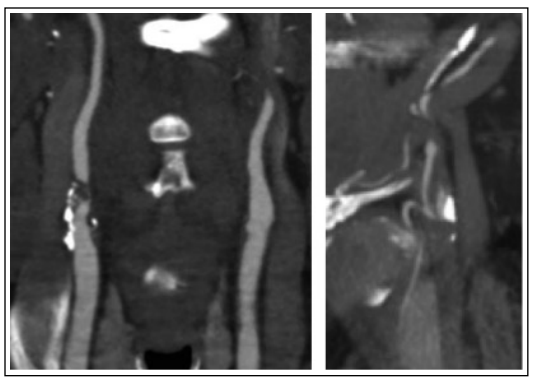
Introduction

We present three cases of carotid endarterectomy (CEA) followed by postoperative development of ipsilateral mydriasis without referable pathology.

A review of the literature was conducted as related to this finding.



Methods

Three patients were identified who underwent CEA with subsequent ipsilateral mydriasis upon awakening.

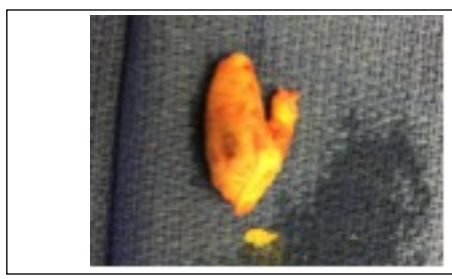
Each patient presented with TIA/stroke symptomatology related to carotid atherosclerosis. Two of the patients demonstrated greater than 90% stenosis in addition to significant intracranial atherosclerosis, and one patient presented with an occluded CCA, but with retrograde flow from his ECA to the ICA.

Results

Endarterectomy was offered to each patient for refractory TIA/strokes. Shunting was not utilized in the cases as neuromonitoring remained at baseline. Clamp time was 90 minutes in one case because of complex anatomy (occluded CCA).

Post operatively all three of the patients awoke with a dilated, ipsilateral, nonreactive pupil. CT scan did not demonstrate any referable pathology. The patients were otherwise at their neurologic baseline and the mydriasis resolved over the ensuing days.

Cases of post CEA Horner's syndrome with a miotic pupil have been reported, with symptoms attributed to manipulation of the sympathetic plexus along the carotid artery. We report the first 3 cases of mydriasis following CEA.



Conclusions

We hypothesize that these cases are secondary to an ischemic phenomenon, specifically to the ciliary ganglion and/or the oculomotor nerve resulting in parasympathetic dysfunction. Blood supply to these nervous structures is from the ciliary arteries (originating from the ophthalmic artery), and branches off the intracavernous portion of the ICA (inferior cavernous sinus artery and meningohypophyseal artery), respectively.

Carotid clamping, poor collateral blood flow, intracranial atherosclerosis, embolic thrombi, and intraoperative lability of blood pressure are all potential causes of ischemia to these parasympathetic structures, both of which derive their blood supply from the clinoidal region of the ICA.

A similar finding of mydriasis following acute carotid injury has been reported, with ischemia also postulated as the etiology. These cases involved carotid dissection, aortic dissection with extension into the carotid, and giant cell arteritis.

Whether certain patients undergoing CEA are more prone to this phenomenon remains unclear. Treating physicians should be aware that post CEA mydriasis may result from local ciliary ganglion or oculomotor nerve ischemia, rather than a devastating intracranial event.

Learning Objectives

By the conclusion of this session, participants should understand:

1) that unilateral mydriasis can occur after carotid endarterectomy or other carotid injury

2) the proposed etiology of this post CEA mydriasis phenomenon

References

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3) Perry., Horner's Syndrome After Carotid Endarterectomy: A Case Report. Vasc Endovascular Surg 35:325–327, 2001

4) Prasad et al., Mydriatic pupil in giant cell arteritis. J Neurol Sci 284:196–197, 2009

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