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Pupil-involving Third-nerve Palsy and Carotid Stenosis: Rapid Recovery Following Endarterectomy

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We report a patient who presented with a painful pupil-involving third-nerve palsy. Cerebral angiography revealed a high-grade stenosis of the ipsilateral internal carotid artery with a 4-cm intraluminal thrombus. Following emergent carotid endarterectomy, the patient's partial third-nerve palsy resolved in 1 hour. A pupil-involving third-nerve palsy may be an unusual presentation of impending carotid occlusion.


Unilateral third-, fourth-, and sixth-nerve paresis accompanied by ipsilateral blindness or contralateral hemispheric signs has occurred on occasion in association with acute thrombosis of the internal carotid artery [1, 2]. In addition, patients with carotid dissection have also presented with major neurologic and ocular deficits, including oculomotor pareses [3–6]. We describe a unique patient with a high-grade internal carotid artery stenosis and ipsilateral pupil-involving third-nerve palsy as the result of ischemia.

Patient Report

A 59-year-old right-handed man was referred for a neuroophthalmic consultation. Two days prior to evaluation, he experienced sudden onset of a severe right periorbital headache and binocular horizontal diplopia, worse in left gaze. The patient recalled, after prodding, that 1 day prior to the headache he had noted the painless appearance of a "gray cloud" over the inferior visual field of his right eye lasting seconds. There were no other visual, neurologic, or constitutional symptoms. The past medical history included cigarette smoking for 45 years.

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On examination, the blood pressure was 140/85. A left, but not right, carotid bruit was detected. Visual acuities at distance were 20/15 in both eyes. Confrontation visual fields were normal. The right pupil measured 4.5 mm in ambient light, was slightly elliptical, and reacted sluggishly to bright light. The left measured 4 mm and contracted briskly to light. There was no afferent pupillary defect. Ocular motility examination revealed partial limitation of adduction, supraduction, and infraduction of the right eye; abduction and intorsion were intact. Maddox rod measurements confirmed the partial right third-nerve palsy. There was no ptosis. Slit-lamp examination and intraocular pressures were normal. Funduscopic examination revealed no abnormalities. The remainder of the neurologic examination was normal.

The presence of an incomplete pupil-involving right third-nerve palsy in the setting of sudden-onset severe headache prompted admission for emergent cerebral angiography. A computed tomographic scan of the brain without contrast performed prior to angiography revealed no evidence of intracranial hemorrhage, infarction, or mass lesion. Three-vessel angiography excluded the presence of an aneurysm; however, right common carotid artery injection demonstrated high-grade stenoses of both the internal and external carotid arteries (Fig 1) with filling defects representing extensive intraluminal thrombus formation. The right ophthalmic artery filled in an anterograde fashion and exhibited moderate atherosclerotic changes. Slow flow through the right internal carotid artery was noted with filling intracranially only of right middle cerebral branches. Injection of the left common carotid artery revealed moderate atherosclerosis with 50% stenosis at the origin of the internal carotid artery. The right anterior and middle cerebral arteries filled via the left injection.

Intravenous heparin was initiated. Right carotid endarterectomy was performed 12 hours later. Intraluminal thrombus was found within the carotid endarterectomy specimen (Fig 2). Examination 1 hour following surgery, including Maddox rod measurements, demonstrated complete resolution of the ocular motility deficit and anisocoria. A magnetic resonance imaging scan of the brain revealed no evidence of brainstem or hemispheric infarction. The patient has had no further episodes of amaurosis fugax or diplopia.

**Discussion**

Our patient demonstrates the unusual finding of a pupil-involving third-nerve palsy in association with a surgically treatable ipsilateral internal carotid artery stenosis. Wilson and colleagues [1] described 3 patients with transient ocular motor paresis and angiographically confirmed occlusion of the ipsilateral internal carotid artery. All 3 patients in their series demonstrated involvement of the third and sixth nerves; fourth-nerve function was affected in 1 patient. Third-nerve involvement was characterized in all cases by pupillary dilation and ptosis. In addition, all developed permanent ipsilateral blindness with evidence of central retinal artery occlusion, and 2 patients had contralateral hemiparesis. Kapoor and co-workers [2] reported a patient with ipsilateral central retinal artery occlusion and corresponding blindness, pupil-involving third-nerve palsy, and contralateral hemiparesis. Cerebral angiography confirmed that the internal carotid and ophthalmic arteries were occluded. In that patient [2], the third-nerve palsy represented the only demonstrable ocular motility deficit. Ophthalmoplegia with third-nerve involvement may occur in association with internal carotid artery dissection, but it is commonly accompanied by other orbital, cranial nerve, or hemispheric signs [3–6].

Several aspects of our patient’s presentation are unique. Initially, the combination of sudden-onset severe headache and incomplete pupil-involving third-nerve palsy prompted cerebral angiography to exclude a posterior communicating artery aneurysm. The angiogram demonstrated a high-grade stenosis of the right internal carotid artery with intraluminal thrombus. Since our patient did not have complete occlusion of the internal carotid artery, carotid endarterectomy was possible, including removal of the thrombus. Our patient had no hemispheric signs or involvement of other cranial nerves; the third-nerve palsy was an isolated examination finding. A brief episode of ipsilateral amaurosis had occurred 3 days prior to presentation, a feature consistent with symptomatic internal carotid artery stenosis.

That ipsilateral internal carotid artery occlusion or high-grade stenosis may result in symptomatic third-nerve ischemia is consistent with the known blood supply to the oculomotor nerve within the cavernous sinus [7–14]. As shown previously [7] in a serial section study of an elderly diabetic patient with recent oculomotor palsy and autopsy control specimens, the nutrient circulation to the third nerve arises from (1) small branches of the basilar and posterior cerebral arteries and the posterior circle of Willis, (2) microscopic branches of the artery to the inferior cavernous sinus [8], and (3) recurrent branches of the ophthalmic artery intraorbitally. Widespread severe thickening and hyalinization of vasa nervorum was noted along with a focal zone of demyelination in the intracavernous portion of the affected third nerve. It is interesting that this lesion was located within a watershed zone between the carotid and posterior cerebral branches. Others [1–6, 9–12] have suggested compromised flow in this circulation as a mechanism of ocular motor nerve ischemia. Ischemia to the third nerve in our patient probably occurred as a result of low flow distal to a severe internal carotid artery stenosis.

In the setting of vasculopathic ocular motor palsy secondary to small-vessel ischemic disease, the time to improvement is generally 4 to 12 weeks [15]. That the anisocoria and ocular motility deficit resolved completely within 1 hour after carotid endarterectomy pro-
Fig 1. Right common carotid artery injection. (A) This magnified view of the right common carotid artery injection demonstrates filling defects within the common carotid (lower arrow), internal carotid (upper arrow), and external carotid arteries (thin arrow). These filling defects represent extensive clots within the carotid circulation. (B) On this oblique view, one can identify the high-grade stenosis of internal carotid artery (arrow) with the filling defect from the clot above. Note the small caliber of the cervical portion of the right internal carotid artery extending superiorly.
Fig 2. Gross photograph of carotid endarterectomy specimen immediately following surgery. The atheromatous plaque has been opened longitudinally; note the presence of intraluminal thrombus. Bar measures 1 cm.

provides reasonable evidence that low flow in the distal internal carotid preoperatively contributed to third-nerve ischemia in our patient.

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