Mydriatic Pupil as the Presenting Sign of Common Carotid Artery Dissection

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Background—Ipsilateral mydriasis is known to accompany signs of cerebral ischemia in unilaterally compromised carotid blood flow. Mydriasis as the presenting sign of common carotid artery (CCA) dissection has not been reported thus far.

Case Description—we report the case of a patient who presented with a mydriatic pupil after intraoperative injury of the ipsilateral CCA. Mydriasis preceded complete third-nerve palsy and symptoms of cerebral ischemia for 12 hours. Cerebral angiography revealed occlusion of the CCA with slow collateral flow to the internal carotid artery and fetal origin of the posterior cerebral artery, suggesting a hemodynamic mechanism causing ischemia of the oculomotor nerve. Signs of cerebral ischemia and third-nerve palsy resolved completely after reconstructive surgery of the occluded vessel.

Conclusions—a mydriatic pupil may be the unusual first sign of compromised carotid blood flow and impending cerebral ischemia. (Stroke. 1998;29:2653-2655.)

Key Words: carotid artery ▪ dissection ▪ mydriasis ▪ oculomotor nerve paralysis

Transient and permanent pareses of ocular motor nerves with and without visual impairment have rarely been reported in atherosclerotic carotid artery disease.1–3 Moreover, dissection of the internal carotid artery (ICA) is known to be occasionally associated with ipsilateral disturbances of oculomotor nerve function.4–7 We describe a unique patient in whom pupillary dysfunction due to evolving paresis of the third nerve was the presenting clinical sign of acute dissection of the common carotid artery (CCA) and impending cerebral ischemia.

Case History
A 44-year-old right-handed, nondiabetic woman with a history of alcoholism underwent elective surgery for hypopharyngeal carcinoma. During the procedure a massive hemorrhage due to erosion of the right CCA occurred, leading to emergency tangential ligation of the vessel. Surgery protocol reported a patent artery after the bleeding had been staunched. Postoperatively, the patient was documented to have no focal neurological deficit. The next day the right pupil was noted to fluctuate. On neurological examination, the patient reported no double vision or headache. At first the right pupil was intermittently mydriatic and reacted poorly to bright light, but minutes later it became 9 mm and fixed to light and near, while an immediate dilation to 10 mm was noted in the dark. The left pupil remained at 4 mm, with normal reactions to light, dark (dilation to 8 mm), and near. Vision, ocular motility, lid function, and corneal reflexes were normal in both eyes, as was the remainder of the neurological examination. Twelve hours later the patient developed complete right third-nerve palsy, visual hemiextinction to the left, and mild left brachiofacial hemiparesis. Plantar responses were flexor. Blood pressure was 110/80 mm Hg. Head CT was unremarkable, but angiography demonstrated occlusion of the right CCA 2 cm below its bifurcation and left-to-right cross-flow to the right middle cerebral artery via the anterior communicating artery. The right ICA filled slowly in an antegrade fashion through collaterals from the right vertebral artery connecting to the right external carotid artery (Figure 1A), and the proximal segment of the right posterior cerebral artery (PCA) was found to be hypoplastic (Figure 1B). No aneurysm was detected. Under controlled elevation of blood pressure, the right hemispheric syndrome resolved almost completely, but the right third-nerve palsy persisted. The next day, reconstructive surgery of the CCA revealed intimal dissection with occlusion at the level of the emergency ligation. The vessel was reconstructed with a saphenous graft, and right-sided neck dissection was performed. Postoperatively, left hemiparesis had resolved completely, and the right pupil was fixed at 6 mm (in light and dark), with no changes in oculomotor function. Brain MRI demonstrated no focal lesion in either the right hemisphere or the upper brain stem. Two days after surgery the right pupil consticted to bright light and was slightly miotic, still with no dilation when the patient was placed in the dark. During the next 4 weeks, right ptosis partially resolved and all other extraocular muscles regained normal function. Five weeks after surgery the patient had a normal neurological status, with the exception of a right-sided Horner’s syndrome, probably as a persisting deficit after neck dissection.

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Discussion

Our patient demonstrates the unique finding of a mydriatic pupil as the first documented clinical sign of impending cerebral ischemia, preceding third-nerve palsy and hemispheric symptoms for 12 hours. A recent report describes a patient with high-grade atherothrombotic stenosis of the right carotid bifurcation and a history of ipsilateral amaurosis fugax, periorbital headache, and sudden-onset diplopia 2 days before admission. In this patient, a right pupil-involving third-nerve palsy with preserved lid function was noted. Carotid endarterectomy led to immediate and complete resolution of all eye signs. It remains unclear whether pupillary dysfunction preceded the oculomotor disturbances in this patient, because diplopia was noted 48 hours before neurological evaluation.

Third-, fourth-, and sixth-nerve palsies, either isolated or combined, are known to occur in <3% of patients with ICA dissection. Miosis due to oculosympathetic paralysis is the most common pupillary dysfunction in carotid dissection, even in cases with oculomotor disturbances. Only 1 patient with isolated pupil-involving third-nerve palsy caused by ICA dissection has been reported, but no details of the clinical course were given. To the best of our knowledge, no case of isolated third-nerve palsy in dissection of the CCA has been reported.

Both hemodynamic failure and embolism may account for the presumed ischemia of nerve trunks in patients with oculomotor abnormalities associated with carotid artery occlusive disease. Blood supply to the oculomotor nerve is provided by small arteries arising from the basilar artery, the PCA, and the posterior communicating artery (subarachnoid nerve segment), the inferolateral trunk (cavernous segment), and the opthalmic artery (extradural and orbital segment). The combination of acute CCA occlusion with fetal origin of the PCA may account for the unusual susceptibility of the oculomotor nerve to ischemia in our patient, because blood flow presumably was compromised in all nerve-supplying arteries. In addition, the initially fluctuating course of eye signs and their recovery following reinstitution of blood flow favor a hemodynamic mechanism, as in the case of Balcer and colleagues. Ischemia to the ciliary ganglion or to the iris has been suggested as a possible mechanism in patients with ICA dissection and pupillary dysfunction. However, none of these patients developed third-nerve palsy, although visual symptoms with signs of retinal or optic nerve infarction were common, suggesting a prolonged hypoperfusion to the orbital structures rather than ischemia to the third nerve alone.

Complete resolution of oculomotor signs within 4 weeks in our patient is reminiscent of the course in microvascular third-nerve palsy, while involvement of pupillary function

Angiography with injection into the right vertebral artery. A, Lateral view. The carotid circulation is filled through anastomoses between vertebral artery muscular branches and the occipital artery. Note slow antegrade filling of the ICA. B, Anterior view. Aplasia of the P1 segment (arrow) of the PCA. RE indicates right.
and lack of periorbital pain are rather untypical features. Asbury and coworkers, who meticulously described the pathological aspects in a diabetic woman with typical (ie, pupil-sparing) and painful oculomotor palsy, attributed the circumscribed demyelinating lesion detected in the cavernous portion of the third nerve to ischemia as a result of small-vessel disease. Preserved pupillary function was explained by the lesion’s distribution in the core of the nerve, thereby sparing parasympathetic fibers presumed to be located more peripherally. Compromised blood flow proximally to the arterioles supplying the nerve, as in our case, is more likely to cause primarily diffuse nerve ischemia, thereby involving parasympathetic and oculomotor fibers in the same way. A possible explanation for the occurrence of mydriasis prior to ocular motor failure could be microembolism to the vasa nervorum, causing additional ischemia to the parasympathetic fibers. Microemboli, demonstrated by transcranial Doppler ultrasonography, have been reported in patients with carotid dissection and were associated with cerebral ischemia. The superficial location of parasympathetic fibers may thereby increase the probability of microembolism to these structures. In addition, the combination of compromised blood flow with microembolism is likely to decrease collateral flow to ischemic nerve segments.

Lack of periorbital pain in our patient may indicate a more posterior location of the site of nerve affection, because ischemia of trigeminal trunks in their cavernous portion is believed to cause periorbital pain in at least 50% of diabetic third-nerve palsies.

In conclusion, our case demonstrates that a mydriatic pupil may be the presenting feature of ischemic third-nerve palsy due to compromised carotid blood flow with impending cerebral ischemia. Lack of periorbital pain in combination with involvement of pupillary function may help to differentiate the hemodynamic mechanism of nerve injury in large-vessel disease from presumed microangiopathy in diabetic third-nerve palsy.

References
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