Mydriatic Pupil as the Presenting Sign of Common Carotid Artery Dissection

H.-C. Koennecke, MD; S. Seyfert, MD

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
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 ophthalmic 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 reinstition 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...
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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