A Rare Cause of Isolated Cranial Nerve III Palsy

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A 64-year-old female with a past medical history of hypertension, hypercholesterolemia, and type II diabetes presents with a chief complaint of double vision. Her current medications include lisinopril, simvastatin, and metformin. Vital signs on presentation are significant for a blood pressure of 164/86 mm Hg, and a heart rate of 72 bpm.

Physical exam reveals a well-developed African American female in no acute distress. Cardiopulmonary exam is normal. Initial cranial nerve testing demonstrates anisocoria, with slight enlargement of the left pupil relative to the right. Extraocular muscle testing demonstrates failed adduction, upward, and downward gaze of the left eye. The remainder of her neurologic exam is normal.

Non-contrast head CT is unremarkable. Gadolinium-enhanced brain MRI reveals subtly increased T2 signal in the region of the posterior left midbrain, with a concomitant small focus of restricted diffusion in this same region (Fig. 1).

The causes of isolated cranial nerve III palsy are myriad. Correct diagnosis hinges on a synthesis of neurological exam and radiologic findings. Differential diagnosis includes ischemic or diabetic neuropathy, extrinsic compression via intracranial aneurysm (most often of the posterior communicating artery), space-occupying midbrain lesions, and cavernous sinus lesions.1

In the present case, physical exam contains several clues that point toward the etiology of the patient’s CN III palsy: The presence of both internal and external CN III dysfunction (i.e., extraocular palsy and pupillary dilatation), for instance, speaks against ischemic or diabetic neuropathy. Indeed, ischemic neuropathy of CN III itself most commonly does not affect the pupil, as the circumferential parasympathetic fibers that supply the pupillary constrictors are spared.2,3

On the other hand, aneurysmal compression, which could cause internal and external CN III dysfunction--and is a dire cause given the risk of imminent rupture--is excluded based on MR findings. Sensitivity of MR angiography in the detection of intracranial aneurysms has been reported as high as 95%.4

Rather, it is the presence of a T2-bright, diffusion-restricted lesion in the left posterior midbrain, adjacent to the periaqueductal gray, that clinches the diagnosis: acute ischemic stroke of the left oculomotor and Edinger-Westphal nuclei.
Lesions of the third nerve nucleus (and the adjacent Edinger-Westphal nucleus) are rare, and are usually accompanied by other signs of mesencephalic involvement. Indeed, literature review discloses only 2 previously published case reports of isolated brainstem lesions presenting with solitary cranial nerve symptoms.\textsuperscript{5,6} The present case thus demonstrates a rare cause of isolated CN III palsy – ischemic CVA of the brainstem in the absence of other nuclear lesions.
References:


Figures:

*Figure 1.* Selected axial images from T2-weighted (left) and diffusion-weighted (right) brain MR demonstrating subtly increased T2 signal and restricted diffusion in the posterior left midbrain.