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A man with double vision

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Hypertension

A man presented with double vision. After a careful physical examination, it was discovered that the patient had a pupil-sparing cranial nerve III palsy. This article will review the case presentation and explore etiologies and treatments for this relatively common condition.

Case presentation

A 54-year-old male presented to the office with complaints of a headache and double vision. He reported that he had these symptoms for a few weeks but he thought they would go away. In addition, he complained that his left eye was not "working right." He could not open the left eye completely. As noted, he had double vision (images side by side) and could make it disappear by covering the left eye. The double vision was also worse while looking to the right and a little better when looking to the left. He denied seeing any flashes of light or having any visual disturbances other than the double vision.

His headache started without injury and had been constant and severe. It started at the back of the head and shot straight through his head to the left eye, and he reported pressure behind it, with a feeling that the eye was being pushed out. He had not found anything that made it better except covering his eye and was not sure what made it worse because it hurt all of the time. Because he thought the pain might be coming from his neck, he went to a chiropractor three times, but the treatment provided no relief of his symptoms. He thought that if he could get rid of his headache, the double vision would go away as well.

His past medical history was significant for type 2 diabetes (10 years), hypertension, melanoma in situ removed 12 years earlier, dyslipidemia, and degenerative joint disease/degenerative disk disease cervical spine. He had fairly good control of his diabetes (last HbA1c was 6.1%), but he admitted that he did not check his blood often enough. He reported that his blood pressure was always better outside of the doctor's office.

His medications were pioglitazone, metformin, atorvastatin, lisinopril, and omega 3 oils. He was also supposed to be taking aspirin but this upset his stomach so he did not take it often.

Physical examination

Vital signs: Height 6′1″, weight 262 lbs, blood pressure 142/90 mm Hg, body mass index 35.

General: Overweight Caucasian male who was in no obvious distress

Head, ears, eyes, nose throat: Exotropia and ptosis of the left eye; only able to move the left eye to the left and downward and to the left; left pupil constricted normally to light and accommodation; no other asymmetries to the face; visual actuity was good for each eye individually.

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Cardiovascular: No carotid bruits; the rest of the exam was normal.

Respiratory: Lungs were clear to anterior and posterior fields.

Neurological: Muscle spasms in the cervical and upper thoracic area; no muscle weakness of the limbs.

Osteopathic exam: He had significant changes to the cervical musculature with increased tension bilaterally and an inhaled thoracic inlet on the right. He had poor motion to rotation bilaterally to the cervical spine and limited extension. The upper thoracic musculature showed chronic tissue texture changes with T2FRlSl (Figs 1 and 2; Table 1).

Lab findings: His labs showed a normal complete blood count, sedimentation rate, and comprehensive metabolic profile. His lipids were not at goal at the time of presentation. Random glucose was 146 mg/dL (Table 2).

In light of his history of melanoma and his significant cardiovascular risk from hypertension and diabetes, a magnetic resonance image/angiogram (MRI/MRA) of his neck and head was ordered to rule out central nervous system tumor, brain bleed, or aneurysm. The MRA of the neck and brain were normal. The MRI of the brain confirmed the presence of an old meningioma but could not provide an explanation for his symptoms (Figs. 3, 4).

He was then referred to an ophthalmologist who confirmed a pupil-sparing third nerve palsy but found no other abnormalities. He was then sent to a neurologist, who confirmed his history and physical examination findings and reviewed all previous testing. After reviewing all of the reported symptoms and findings, the neurologist felt that the patient had a metabolic cranial nerve 3 palsy. The condition was a result of hypertension and diabetes, and the patient was told that his symptoms would likely resolve with time.

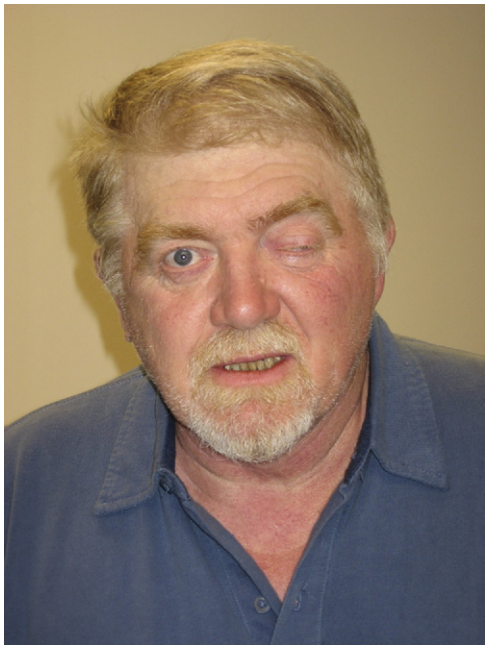

Figure 1 Patient at presentation.

Figure 2 Patient at presentation.

Diagnosis: pupil-sparing unilateral third nerve palsy

This patient had a pupil-sparing unilateral third nerve palsy that resulted from ischemic changes to the oculomotor nerve. This occurs most often as a result of chronic diseases such as diabetes and hypertension.

Topic discussion

Complete third nerve palsies classically present with unilateral ptosis, down and out deviation of the eye, iridoplegia, and cycloplegia. Patients may complain of pain, double vision, glare, and blurred near vision.[1] Variations of this presentation can occur based on the location and type of lesion creating the palsy. One such variation exists with diabetic ischemic oculomotor nerve infarcts. These infarcts, involving the vasa nervosa, occur in the central portion of the peripheral nerve and spare the pupiloconstrictor parasympathetic fibers that lie on

Table 1 Differential diagnosis

CNS vascular lesion	Cerebrovascular accident, aneurysm
CNS tumor	Primary CNS, metastatic melanoma, or other occult malignancy
CNS trauma	
Systemic inflammatory processes	Multiple sclerosis, myasthenia gravis, vasculitis, others
CNS infections	Lyme disease, syphilis, others

CNS = central nervous system.

Table 2 Lab values

Lab	Result	Reference range
HbA1c	6.1%	4.0%-5.7% (normal) DM goal <7.0%
Total cholesterol	188 mg/dL	200-239 mg/dL
Triglycerides	117 mg/dL	40-184 mg/dL
HDL-C	37 mg/dL	>40 mg/dL
LDL-C	128 mg/dL	<100 mg/dL in DM
CBC: white blood cells	10.0	4.5-11.0
Hemoglobin	14.9	13.0-17.0
Hematocrit	44.2	38.0-50.0
Platelets	220	130-400
Sedimentation rate	5	0-15

DM = diabetes mellitus; HDL-C = high-density lipoprotein cholesterol; LDL-C = low-density lipoprotein cholesterol; CBC = complete blood count.

the lateral to inferior peripheral portion of said nerve. Therefore, they tend to present as pupil-sparing, but otherwise complete unilateral, palsies.[2] Many patients will present with eye pain as well, but the etiology of this is unclear (Fig. 5).

To better understand the varied presentations of third nerve palsies, a quick review of oculomotor nerve anatomy is included here. The origin of the nerve lies in separate subnuclei located in the midbrain. Nerve fibers that innervate the pupil, ciliary bodies, levator palpebrae, superior rectus, inferior rectus, inferior oblique, and medial rectus muscles all originate in different areas of the nucleus in a dorsal to ventral order as listed here. The levator palpebrae muscle has innervations from both

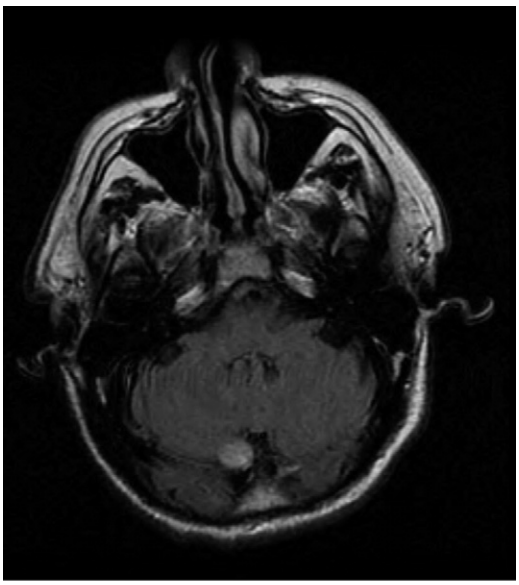

Figure 3 MR 1—right-sided occipital lesion.

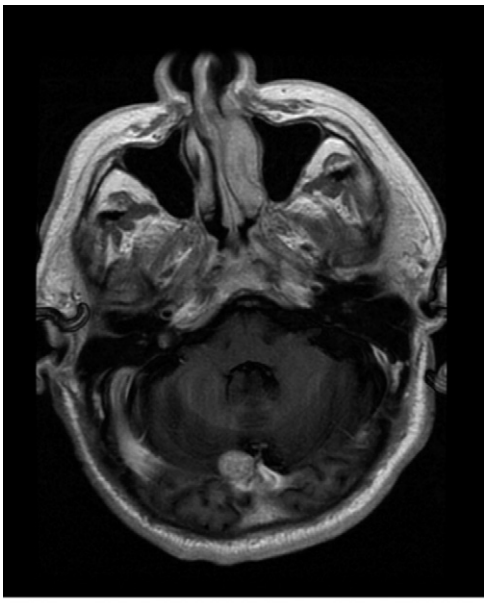

Figure 4 MRI 3 and MRI 4—Lesion appears to be meningeal in source.

sides, whereas the superior rectus receives all of its innervations from the contralateral side. All other muscles solely receive ipsilateral innervations. The parasympathetic fibers innervating the pupil and ciliary bodies arise from the ipsilateral Edinger Westphall nucleus. Once the oculomotor nerve fibers leave the subnuceli, they traverse through the midbrain, passing the medial longitudinal fasciculus, red nucleus, and substantia nigra and they then exit via the medial portion of the cerebral peduncle. Once it exits the brainstem, the oculomotor nerve passes between the superior cerebellar and posterior cerebral arteries. The nerve then enters the orbit via the superior orbital fissure and branches into superior and inferior divisions. The superior branch innervates the superior rectus and levator palpebrae muscles, whereas the inferior branch reaches the pupil, ciliary body, inferior rectus, inferior oblique, and medial rectus muscles.[3]

Along the origination and course of this nerve, there exist many locations where lesions to the nerve can result in variations of palsy. Infarcts, aneurysms, tumors, trauma, inflammatory processes, and infections should all be included in a differential diagnosis for third nerve palsy.[4] The location of the lesion may be the primary variable in determining the pathological etiology and thus directing treatment options. Lesions that occur in the nuclear portion, such as small midbrain infarcts or tumors, tend to present with contralateral partial ptosis and levator palsy. Intramedullary lesions usually have other associated neurological symptoms related to other involved nuclei. Radicular lesions in the subarachnoid space and along the tentorial edge can be isolated but are more likely the result of a hemorrhage or infectious or inflammatory etiology and thus have the associated

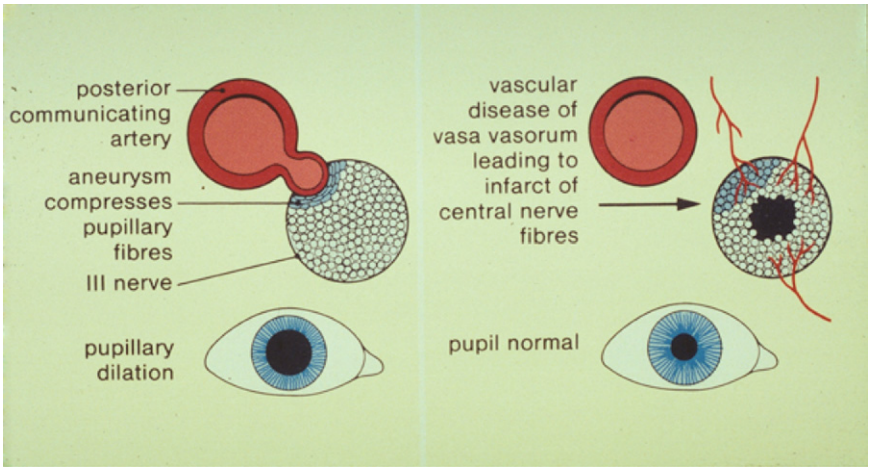

Figure 5 Compressive lesions such aneurysm, and tumors (*left*) cause a complete CN 3 palsy while microvascular lesions (*right*) spare the pupil. From Slide Atlas of Ophthalmology, Gower Medical Publishing, 1984.

symptoms of loss of consciousness, fever, stiff neck, and so on. Orbital lesions tend to have associated proptosis, lid swelling, conjunctival injection, and chemosis. Most isolated cranial nerve palsies occur in the area of the cavernous sinus and superior orbital fissure.[3]

Laboratory tests helpful in making the diagnosis

Location can be derived from a careful clinical history and physical examination. Imaging with MRI or computed tomography scan of the brain can be helpful to rule out other causes.[5] Potential tests to consider to rule out other causes in the differential include: sedimentation rate, syphilis serology, Lyme titer, glucose, antinuclear antibodies, and lumbar puncture.[5]

Treatment: symptomatic

Patients should receive only symptomatic treatment: pain management, eye patching, and other supportive measures. Patients tend to have complete reversal of symptoms within three months of onset.[6] However, subsequent cranial mononeuropathies can occur in approximately 60% of patients.[7] Risk factors for recurrence include diabetes, hypertension, obesity, hyperlipidemia, and age older than 60 years. All modifiable risk factors should be addressed if possible.[8]

Figure 6 Patient after resolution of his cranial nerve palsy.

Figure 7 Patient after resolution of his cranial nerve palsy.

Patient outcome

The only treatment offered supportive care. It was recommended that the patient continue to tightly manage his diabetes and hypertension and he use a muscle relaxant to stop discomfort in his neck. Three months after his initial presentation, his symptoms had completely resolved and he returned to normal home and work duties. Interestingly enough, his symptoms reappeared a year later in the contralateral eye. This episode resolved in 6 weeks (Figs. 6, 7).

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