Bilateral Third Nerve Palsy and Temporal Arteritis

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**Background:** Bilateral oculomotor palsy is a rare manifestation of temporal arteritis, and to our knowledge only 1 case has been described in the literature.

**Objective:** To investigate a possible case of temporal arteritis in a patient with bilateral third nerve palsy.

**Design:** Case report and review.

**Setting:** University hospital.

**Patient:** A 65-year-old man had subacute pupil-sparing bilateral third nerve palsy.

**Results:** Temporal artery biopsy findings and response to corticosteroids are consistent with temporal arteritis.

**Conclusion:** Temporal arteritis is a rare cause of ophthalmoplegia in elderly persons and may be unrecognized.

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**TEMPORAL ARTERITIS (TA)** is a common form of vasculitis, with an incidence as high as 24.1 per 100,000 population. The range of neuro-opthalmologic signs and symptoms in TA is broad and includes diverse manifestations of ischemic optic neuropathy, retinal infarction, ophthalmoplegia, pupillary autonomic and anterior ocular segment dysfunction, cortical blindness and associated posterior chiasmal field defects, and complex visual hallucinations. Oculomotor palsy is an uncommon manifestation of TA. This association was originally described by Fisher in 1959. We describe a patient with bilateral oculomotor palsy in association with histologically proved TA. The rapidly reversible nature of the ophthalmoplegia is in sharp contrast to the permanent damage caused by the other ophthalmologic manifestations, particularly ischemic optic neuritis and retinal artery occlusion.

**REPORT OF A CASE**

A 65-year-old Hispanic man with type 2 diabetes mellitus had a 6-week history of frontal and bitemporal headache. Four weeks before admission, ptosis of the left eyelid and horizontal diplopia developed. During the next few days ptosis of the right eyelid developed as well. The patient denied any visual impairment, jaw claudication, fever, weight loss, arthralgia, or myalgia. The medical history was remarkable for poorly controlled diabetes mellitus for 30 years and renal failure requiring regular hemodialysis. He had a moderate smoking history, but never abused alcohol or recreational drugs. At physical examination his blood pressure was 150/89 mm Hg and he was afebrile. Palpation demonstrated that the temporal arteries were cordlike bilaterally. Findings from the general examination revealed a 3/4 systolic flow murmur in the apex and Dupuytren contracture of the third and fourth fingers of the right hand. Mental status was normal. Visual acuity was 20/80 OD (20/70 OD with pinhole) and 20/60 OS (20/50 OS with pinhole). Ophthalmoscopic and slitlamp examination revealed bilateral cataracts and moderate nonproliferative diabetic retinopathy. Pupils were round at 3 mm and reactive to light. The result of extraocular muscle examination demonstrated bilateral adduction palsy, impaired upward and downward deviation, and bilateral ptosis. Abduction was bilaterally intact. Findings from the remainder of the neurologic examination were unremarkable.

Laboratory data indicated mild anemia (hemoglobin level, 9.6 g/dL; mean cell volume, 100 fl; red blood cell distribution width index, 14%); normal white blood cell count and differential cell count; erythrocyte sedimentation rate, 77 to 83 mm/h; and C-reactive protein, 2.2 mg/dL. (normal,
<0.8 mg/dL). The serum chemistry study results disclosed the following levels: potassium, 5.8 mmol/L (mild hyperkalemia); glucose, 266 mg/dL (14.8 mmol/L); creatinine, 6.6 to 13 mg/dL (583-1149 μmol/L); serum urea nitrogen, 35 to 64 mg/dL (12.5-22.8 mmol/L); calcium, 8.5 to 10.1 mg/dL (1.12-2.52 mmol/L); phosphate, 6.7 mg/dL (2.1 mg/dL); low-density lipoprotein cholesterol, 85 mg/dL (2.20 mmol/L); high-density lipoprotein cholesterol, 27 mg/dL (0.70 mmol/L); thyrotropin, 0.4 mIU/L; creatine phosphokinase, 74 U/L; and glycosylated hemoglobin, 7.1%. Antinuclear antibody, double-stranded DNA, antineutrophil cytoplasmic antibody, rheumatoid factor, acetylcholine receptor, and serologic evidence of abnormal enhancement, diffusion-weighted signal in the white matter without any evidence of abnormal image abnormality, or infiltrating mass of the cavernous sinus. Magnetic resonance angiography of the brain showed a small vertebral artery on the left side. Repetitive nerve stimulation did not show any abnormality in neuromuscular transmission. One week after admission a diagnosis of TA was considered, and intravenous methylprednisolone, 1g/d for 2 days, was administered, followed by oral prednisone, 100 mg/d. The day after corticosteroid administration, temporal artery biopsy tissue was obtained. The specimen revealed extensive medial calcific sclerosis, intimal hyperplasia, and multinucleated giant cells, consistent with TA. Two days later, headache and right eyelid ptosis improved significantly. The patient was discharged home receiving oral prednisone, 100 mg/d. Twenty days after starting corticosteroid therapy, the erythrocyte sedimentation rate decreased to 28 mm/h, and the prednisone dosage was reduced to 80 mg/d by an ophthalmologist. Two months later the patient had 2 episodes of new-onset generalized tonic-clonic seizures. Extraocular examination then showed slight left-sided ptosis and mild right adduction weakness. A second brain magnetic resonance image did not show any changes.

**COMMENT**

Nontraumatic, isolated, bilateral third nerve deficit may be caused by central lesions (ie, midbrain stroke, mesencephalic hematoma, Whipple disease, and neurosarcoidosis) or, more commonly, by peripheral causes (Table). Oculomotor neuropathy has been the proposed pathomechanism for peripheral causes, including cerebral infections such as mucormycosis, ruptured giant internal carotid artery aneurysm, diabetic neuropathy, neurosyphilis, lymphoma, pituitary apoplexy, and Fisher syndrome. Among the central causes of bilateral third nerve palsy are Whipple disease and basilar artery occlusion. Consideration should also be given to pseudo–third nerve palsy, such as myasthenia gravis, Grave ophthalmopathy, and chronic progressive external ophthalmoplegia.

**Table. Reported Causes of Peripheral Nontraumatic Bilateral Oculomotor Deficit**

| Microvascular: diabetes mellitus |
| Vasculitis: sarcoidosis, Wegener granulomatosis, or giant cell arteritis |
| Inflammatory: Tolosa-Hunt syndrome |
| Infectious: syphilis (meningovascular), Lyme disease, or mucormycosis |
| Tumors (mass effect): metastatic prostate cancer, lymphoma (Burkitt) to cavernous sinus, or primary oligodendroglioma |
| Polyneuropathy: Guillain-Barré syndrome, Fisher syndrome, or acute pandysautonomia |
| Pseudo-oculomotor deficit: myasthenia gravis, Grave ophthalmopathy, or chronic progressive external ophthalmoplegia |
| Subarachnoid hemorrhage |
| Intracranial hypotension |
| Congenital bilateral oculomotor palsy |

In 1 series, among 130 patients with isolated oculomotor nerve palsy only 2 patients had bilateral involvement; one had diabetes mellitus and the other had metastatic lung carcinoma. Fisher in 1959 reported the first case of bilateral simultaneous third nerve palsy in association with TA. However, the patient had a mixed partial third nerve palsy that was complete on one side and only partial on the other. In contrast, our patient had a complete pupillary sparing oculomotor nerve dysfunction. Fisher’s patient demonstrated no light reaction by either pupil, although both pupils reacted well on convergence testing.

The ophthalmoparesis in TA may be self-limited and fluctuating, frequently involve vertical excursions, display excellent corticosteroid responsiveness, and may be the sole initial symptom. Whether the primary pathologic involvement in ophthalmoparesis occurs in the nerve or muscle has not been conclusively determined. The frequent lack of brainstem findings and the fluctuating nature of the paresis suggest a common primary myogenic locus. However, the rare finding of oculomotor synkinesis in TA lends credence to the alternate occurrence of primary neurogenic dysfunction. In the lone autopsy case of ophthalmoplegia related to TA, no brainstem, oculomotor nerve, ciliary ganglion, or clinically involved left iris sphincter were affected pathologically, but necrosis of multiple extraocular muscles was noted. Even in cases of compromise of the long posterior ciliary and anterior arteries, selective anterior ocular segment ischemia can occur without ophthalmoparesis. In the aforementioned study, concluded that extraocular muscle ischemia is the mechanism by which ophthalmoplegia occurs and that arteritis of orbital branches of the internal carotid artery is the vascular substrate, to which may be added variable degrees of involvement of the external carotid artery and its branches. Although one may argue that a similar myopathic mechanism was responsible in our patient, the absence of lateral rectus muscles involvement may suggest bilateral third nerve palsy rather than multiple ischemic extraocular muscle involvement. Similarly as in our patient, pupillary sparing is commonly seen with ischemic third nerve palsy.
Ophthalmoplegia is commonly seen in diabetes mellitus and sometimes can be the initial feature. Sergott et al\(^8\) concluded that simultaneous bilateral oculomotor palsy due to the occlusive vascular disease of diabetes mellitus is a distinct clinical entity but must remain a diagnosis of exclusion. Also, in Keane's\(^{15}\) series of 125 cases of bilateral sixth nerve palsy, none were classified as diabetic in origin. At clinical neuro-ophthalmologic examination alone, it would be difficult to differentiate an arteritic cause from a diabetic cause of bilateral ophthalmoplegia. In the presence of consistent clinical findings, positive temporal artery biopsy findings, and the dramatic response to corticosteroid therapy; however, we believe that the ophthalmoplegia must be attributed to TA and not to diabetes. In light of the present case and our review of the literature, we suggest that TA must be considered a cause of bilateral oculomotor palsy, particularly in elderly persons with elevated erythrocyte sedimentation rate and new-onset headache.

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