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Inferior Branch Oculomotor Nerve Palsy

A Case Report

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We describe a 34-year-old man with acute, nontraumatic inferior branch oculomotor nerve palsy. Complete ophthalmologic, neurologic, and systemic examinations were otherwise normal. The oculomotor nerve palsy resolved, but the patient subsequently developed bilateral upper extremity numbness and painful dysesthesias in the distribution of the median nerves. These observations suggest that inferior branch oculomotor nerve palsy, although uncommon, may occur as part of a more generalized neurologic disorder, presumed in our patient to be either vasculitic or demyelinating in nature.
Key Words: Oculomotor branch nerve palsy—MRI—CT scans.

Oculomotor nerve palsies account for up to one-third of all paretic ocular misalignment syndromes (1–4). Incomplete involvement of the oculomotor nerve is common, particularly with regard to pupillary involvement in the setting of an ischemic vasculopathy (5,6). However, pure branch palsies of the oculomotor nerve are rare (6,7).

Ten cases of isolated inferior branch oculomotor nerve paresis have been reported in the literature (6,8–10) (Table 1). Of these, six have been posttraumatic (8,9), while only four have occurred in the absence of trauma (6,10). We present here a fifth case of acute, nontraumatic inferior branch oculomotor nerve palsy. Our patient subsequently developed a second, temporally dissociated neurologic deficit, suggesting a generalized vasculitic or demyelinating etiology.

CASE REPORT

A 34-year-old man was seen in consultation at the Department of Ophthalmology of the University of California, San Francisco, for acute onset of right-sided periocular pain and horizontal diplopia. He had no history of diabetes mellitus, hypertension, systemic vasculopathy, migraine, or recent viral illness. The general physical examination was normal. Ophthalmologic examination showed vision of 20/20 in each eye. The right pupil was 6 mm and unreactive to light. The left pupil was 4 mm and reactive to light. In primary gaze he showed 30 prism diopters of right exotropia and 10 prism diopters of right hypertropia. The right eye also showed poor adduction and infraduction. Extraocular motility in the left eye was normal (Fig. 1). The remainder of the ophthalmologic, neurologic, and systemic examination was unremarkable.

Magnetic resonance imaging (MRI) of the central

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TABLE 1. *Reported cases of isolated inferior branch oculomotor nerve paresis*

Case	Age/sex	Etiology	Associated findings	Author/year (ref.)
1	—	Orbital trauma	—	Cross/1948 (8)
2	—	Orbital trauma	—	Cross/1948 (8)
3	—	Orbital trauma	—	Cross/1948 (8)
4	30/M	Undetermined	None	Susac and Hoyt/1977 (10)
5	5/F	Undetermined	None	Susac and Hoyt/1977 (10)
6	8/F	Undetermined	None	Susac and Hoyt/1977 (10); Miller/1977 (11)
7	—/M	Undetermined	None	Miller/1985 (6)
8	—	Orbital trauma	Trigeminal nerve damage	Saul et al./1986 (9)
9	—	Orbital trauma	Trigeminal nerve damage	Saul et al./1986 (9)
10	—	Occipital trauma	Optic nerve damage	Saul et al./1986 (9)
11	34/M	Undetermined	Upper extremity numbness/ dysesthesias	Present case report

nervous system (CNS), MRI-cerebral angiography, and intravenous contrast cerebral angiography revealed no localizing lesions. Urinalysis and serology, including tests of electrolytes, complete blood cell count, Westergren erythrocyte sedimentation rate, antinuclear antibody titer, and rapid plasma reagin test were normal. A lumbar puncture was performed under an opening pressure of 190 mmHg. The cerebral spinal fluid (CSF) had no xanthochromic discoloration, no red blood cells, and 6 white blood cells per microliter, all of which were lymphocytes. The CSF glucose, total protein, quantitative venereal disease research laboratories test, and cryptococcal antigen titer were normal. Aerobic, anaerobic, and fungal cultures of CSF were negative.

The patient was discharged and followed closely for 3 months, during which time he regained complete ocular realignment. More than 4 months after the onset of his oculomotor palsy, the patient experienced bilateral numbness and painful dysesthesias in the distribution of the median nerves.

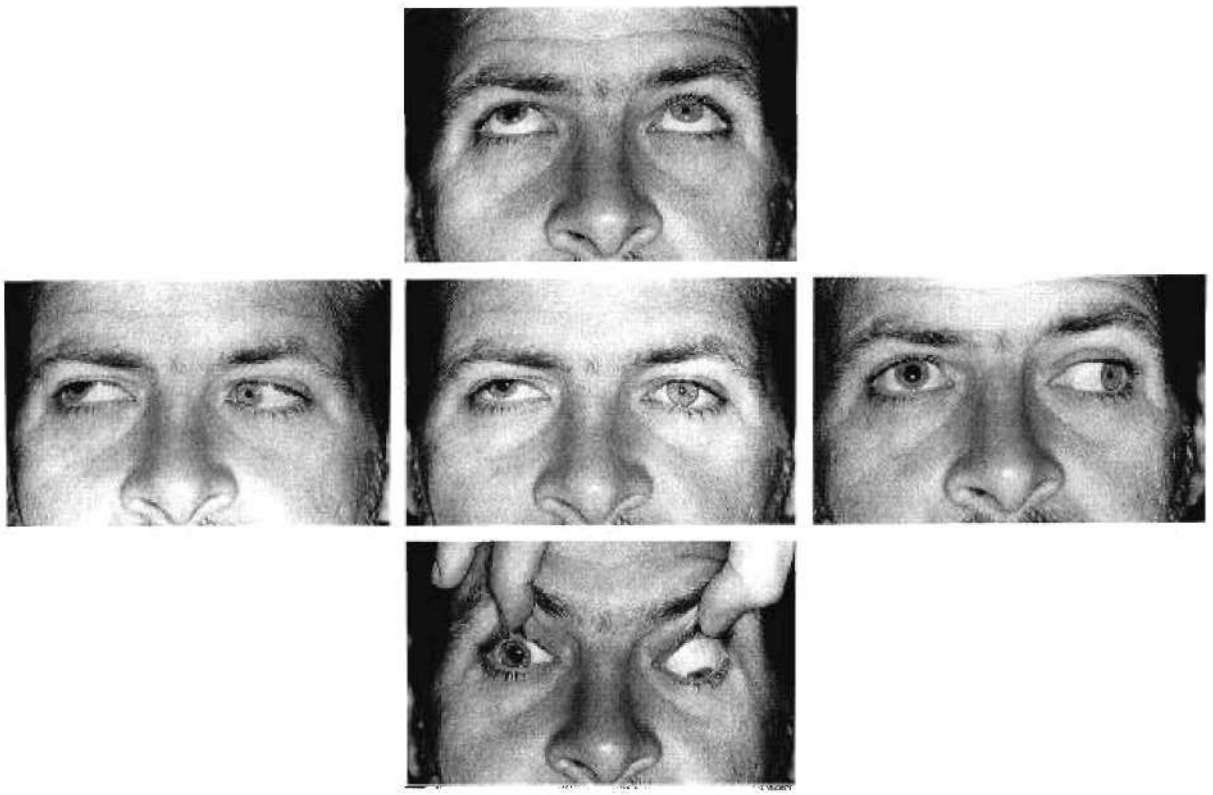

FIG. 1. Palsy of the inferior division of the oculomotor nerve. The inferior division of the oculomotor nerve provides innervation to the medial rectus (adduction), inferior rectus (depression), inferior oblique, and pupil, all of which are affected in this man's right eye. Note that he can elevate his right eye, and that there is no right ptosis, both functions subserved by the superior division of the oculomotor nerve.

Computed tomography (CT) and MRI of the vertebral column and spinal cord were performed and revealed mild lumbar disc herniation but no evidence of cervical cord or root disease.

DISCUSSION

Isolated oculomotor branch palsies are uncommon. Superior branch palsies have been reported in patients with structural disorders of the subarachnoid space in the region of the oculomotor nerve (12), intrinsic midbrain disease (13), diabetes mellitus (14,15), migraine (16), and viral upper respiratory tract infections (11,17,18). Four cases of nontraumatic inferior branch oculomotor palsy have been described (6,10,11), each with undetermined etiology.

A prodromal viral illness is often the presumed cause of idiopathic oculomotor nerve paresis (6,10,11,17–24). Our patient subsequently developed bilateral numbness and painful dysesthesias in his upper extremities. Spinal CT scans and MRI failed to disclose localizing lesions. The occurrence of two temporally dissociated neurologic deficits is highly suggestive of a generalized neurologic illness, such as occurs with vasculitic or demyelinating disease. Patients with inferior branch oculomotor nerve palsy have a good prognosis for spontaneous recovery, but they should be evaluated and followed closely for the possibility of a generalized systemic or neurologic disorder.

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