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Resolution of Saccadic Palsy After Treatment of Brain-Stem Metastasis

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• A 65-year-old man was unable to generate normal horizontal saccadic eye movements. Smooth pursuit of sinusoidal target motion was normal. The saccadic palsy resolved rapidly, twice, after treatment with intravenous corticosteroids. Computed tomography showed a lesion in the pons, and seven months later he was found to have metastatic adenocarcinoma.

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Horizontal gaze palsy is characterized by a slowing or loss of conjugate fast eye movements.¹ It has been reported in a variety of lesions of the pontine tegmentum,¹ including metastasis.² Herein we report the case of a patient who had metastatic adenocarcinoma and rapid resolution of a saccadic gaze palsy after treatment.

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REPORT OF A CASE

A 65-year-old man was admitted for neurologic examination. For several months he had noticed an inability to "focus" his eyes when shifting gaze, unsteadiness when walking, and a dull headache.

The visual acuity, visual fields, and pupils were normal. Horizontal saccades were slow; and attempts to make a single,

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Fig 1.—Axial computed tomographic sections showing contrast enhancing round lesion in pons. Sections (1.0 cm) from rostral (left) to middle (center) to caudal (right) pons.

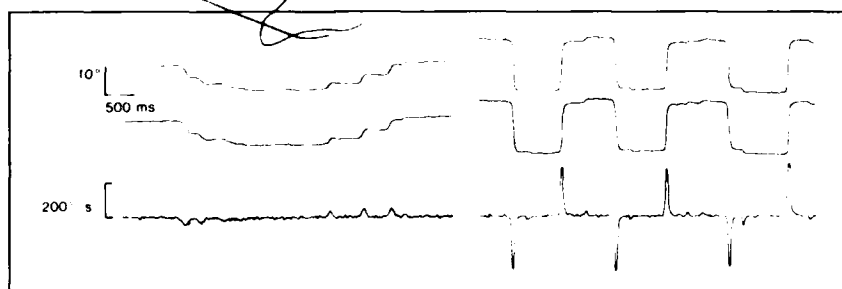


Fig 2.—Horizontal saccadic eye movements before (left), and 48 hours after (right), treatment with dexamethasone. Upper lines are right and left eye position, respectively; and lower line is eye velocity. Horizontal saccades were hypometric and slow before treatment, but of normal size and velocity after treatment.

large saccade produced a sequence of short saccades. The fast component of the vestibulo-ocular reflex was deficient when the patient was rotated in a chair. Vertical saccades were preserved as was convergence, smooth pursuit, and the slow component of the vestibulo-ocular reflex. He was mildly dysarthric and his gait was unsteady.

A computed tomographic (CT) scan with contrast enhancement (Fig 1) showed a 1.5-cm round lesion in the pontine tegmentum. Lucent areas suggesting edema were evident within the pons adjacent to the lesion. A vertebral arteriogram showed no vessel displacement or abnormal vessels.

The patient's dysarthria worsened, and he developed a right hemiparesis. Dexamethasone acetate, 4 mg administered intravenously every six hours, was started. Within 48 hours, the patient's eye movements were clinically normal, and his hemiparesis and dysarthria had resolved. Repeated CT scans showed shrinkage of the areas of contrast enhancement in the pons. Although the lesion was thought to be a possible metastasis, extensive work-up failed to reveal a primary source. The dexamethasone was gradually reduced and the patient was discharged.

He returned in one month with complaints identical to those of his initial

presentation. Electro-oculographic eye movement recordings showed slow, grossly hypometric saccades in the horizontal meridian (Fig 2, top). Smooth pursuit of sinusoidal target motion was normal (Fig 3). Forty-eight hours after starting treatment with intravenous dexamethasone, the saccades were normal (Figure 2 bottom). The dexamethasone was gradually reduced, and radiation therapy to the brain stem was initiated. He remained free of symptoms for the next seven months.

His headaches and hemiparesis returned. A fourth CT scan showed no pontine lesion, but multiple densities scattered throughout both cerebral hemispheres. A frontal craniotomy and biopsy specimen disclosed adenocarcinoma. In the weeks that followed, the patient's mental status deteriorated. He had two generalized seizures, lapsed into a coma, developed pneumonia, and died. The family refused permission for an autopsy.

COMMENT

The biopsy result, CT scan appearance, and clinical course of this patient are compatible with adenocarcinoma metastatic to the pons. The areas of decreased attenuation around the pontine lesion suggested tumor edema. Corticosteroids are known to

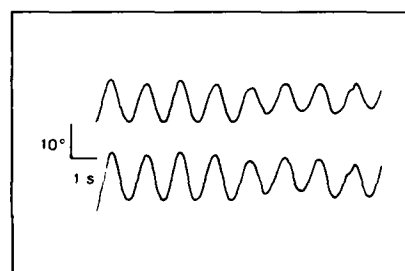


Fig 3.—Horizontal smooth pursuit eye movements were normal before treatment (compare with left panel of Fig 2). Patient pursued hand-held target that was moved at about 15 degrees per second.

be effective in reducing vasogenic edema due to brain tumor,¹ and this appears to have been the critical factor in reversing the saccadic palsy in our patient.

That pursuit eye movements and the slow-phase of the vestibulo-ocular reflex were preserved in this patient is unusual. Extracellular recordings from the pons of behaving monkeys indicate that slow eye movements are associated with lower rates of neuron firing, than are saccadic eye movements.⁴ Palsy of saccades, but preservation of pursuit, implies that this patient had a greater impairment of ocular motor neurons that fire at high rates.

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