In the case reported herein, early anti-Brucella therapy and treatment with corticosteroids resulted in complete recovery and return of visual acuity.

Ronen Rabinowitz, MD
Marina Schneck, MD
Jaime Levy, MD
Tova Lifshitz, MD

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Correspondence: Dr Rabinowitz, Tabenkin 25 St, Beer-Sheva, Israel 84750 (ronenrab@netvision.net.il).


Midbrain-Thalamic Ocular Neuromyotonia

Ocular neuromyotonia is characterized by paroxysms of tonic contraction of 1 or more of the extraocular muscles, usually consequent to radiation.1,2 We describe a unique example of ocular neuromyotonia from nonirradiated, stroke-related intramedullary lesions of the midbrain and thalamus.

Report of a Case. A healthy 41-year-old man lapsed into a coma after developing sudden dizziness, sweating, and diplopia. Magnetic resonance imaging (Figure) showed high signal intensity on T2-weighted images at the left mesodiencephalic junction involving the left red nucleus and third nerve extending superiorly to the left centromedian thalamus, a punctate focus of susceptibility at the root exit zone of the left third nerve and a small area of abnormal signal at the right mesodiencephalic junction. The lesions were consistent with hemorrhage and infarction. The patient was presumed to have suffered a stroke. He improved but had residual diplopia, a "clumsy" right arm, and gait ataxia.

Three months after the onset, there was bilateral lid retraction, slow elevation and depression of both eyes, and limited elevation and depression of the left eye. His pupils were unremarkable. Two days later, the patient reported that he had developed about 30 episodes daily of vertical diplopia, each heralded by transient right periorbital tingling. His right eye would deviate down and in for up to 3 minutes. Tingling would recur, and the eye would return to a normal position. There was no oscillopsia. Observation of several episodes during re-examination several days later confirmed his description. Carbamazepine abolished the episodes. Three months later, he had full ductions, but elevation and depression of both eyes were slow. On all refixations, both eyes would make a slow, conjugate, small-amplitude clockwise...
rotation and then a corrective movement before stabilizing. At present, he is still receiving carbamazepine.

Comment. Ocular neuromyotonia affects the extraocular muscles either individually or in combination, causing episodic diplopia that develops either spontaneously or after gaze in the direction of action of the affected muscle. Electromyography suggests a neurogenic basis for the movements.3,4 The tonic contractions are thought to result from the spontaneous discharge of unstable neurons, which are transmitted to adjacent neurons by ephaptic transmission. Consistent with the theory of axonal instability, membrane-stabilizing agents such as carbamazepine are effective.

To our knowledge, this is the first report of a patient whose ocular neuromyotonia was caused by a stroke, as well as the first in which the responsible lesion was intramedullary. Our patient's lesions were predominantly contralateral to the side of his neuromyotonia, with only 1 small lesion at the ipsilateral mesodiencephalic junction. While we cannot determine which, if any, of his lesions caused the neuromyotonia of his right eye, we note that his lesions spared the nuclei and intramedullary fascicles of the right third nerve. We conclude that ocular neuromyotonia can result from purely intramedullary lesions, without the involvement of the lower motor neuron.

Michelle C. Banks, MD
Paul A. Caruso, MD
Simmons Lessell, MD

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Correspondence: Dr Lessell, Massachusetts Eye and Ear Infirmary, 243 Charles St, Boston, MA 02114 (simmons_lessell@meei.harvard.edu).

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Rosiglitazone-Induced Proptosis

Rosiglitazone maleate belongs to the class of thiazolidinedione drugs used to maintain glycemic control in patients with type 2 diabetes mellitus. The molecules in these drugs sensitize tissues to insulin activity through their ligation of the peroxisome proliferator-activated receptor-gamma (PPARγ) and by increasing expression of the glucose transporter 4 receptor. Peroxisome proliferator-activated receptor-gamma is a nuclear receptor that provokes adipocyte differentiation.1 The thiazolidinediones stimulate adipogenesis thus producing well-described weight gain by the generalized increase in subcutaneous fat volume.2

One case of reactivated thyroid associated ophthalmopathy (TAO) has been reported in a patient whose TAO had previously entered the stable phase of the disease within 3 months of instituting treatment with rosiglitazone.3 We now describe a patient without concurrent or historic thyroid disease who developed new-onset proptosis after treatment with this medication.

Report of a Case. A 53-year-old Hispanic woman had slowly progressive, painless, bilateral proptosis without periocular inflammation, change in visual acuity, or diplopia over approximately 1 year. The patient also noticed a recent 9-kg weight gain, with a 4-in increase in abdominal girth. She denied changes in diet or exercise. The patient recalled a series of evaluations for globe prominence beginning at age 14 years. Despite her assurances to the treating physicians at that time that she manifested a familial trait

Figure. Magnetic resonance images of the brain. A, Axial T2-weighted image showing high signal intensity (arrow) at the left mesodiencephalic junction. B, Axial T2-weighted image showing bilateral signal elevation (arrows) in the centromedian thalami and an incidentally noted left insular developmental venous anomaly (arrowhead). C, Axial gradient echo image showing focus of susceptibility (arrow) along the course of the left third nerve.

Michelle C. Banks, MD
Paul A. Caruso, MD
Simmons Lessell, MD


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