Teaching NeuroImages:

Ocular neuromyotonia

An underrecognized cause of transient diplopia

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Figure 1 Tonic left inferior rectus contraction causing diplopia in ocular neuromyotonia

Right hypertropia in primary and eccentric gaze (aside from downgaze), maximal in right and upgaze with a left supraduction deficit.

A 63-year-old man presented with several months of intermittent vertical diplopia mainly while reading. He consistently developed a transient right hypertropia (with left supraduction deficit) after looking down for approximately 10 seconds and then back to primary (figure 1). Neurovascular contact between the left third nerve and posterior cerebral artery was demonstrated with magnetic resonance constructive interference in steady state imaging (figure e-1 on the *Neurology*[®] Web site at Neurology.org), and episodes were resolved with carbamazepine. Aside from presumed transient tonic contraction of the left inferior rectus, there was no evidence that other third-innervated muscles were involved.

When vascular compression of an ocular motor nerve causes ocular neuromyotonia (ONM), it is thought that ephaptic transmission is responsible. Axonal “cross-talk” causes irritability and abnormal firing independent of the synapse.¹ Without thin, heavily T2-weighted constructive interference in steady state or FIESTA (fast imaging employing

steady-state acquisition) sequences, neurovascular contact can be missed.² ONM has also been associated with radiation therapy, thyroid eye disease, mass lesions, or superior oblique myokymia.¹ If diplopia manifests or worsens *after* prolonged eccentric gaze, ONM should be considered.

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DISCLOSURE

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Supplemental data
at Neurology.org

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