Magnetic Resonance Imaging of Superior Oblique Muscle Atrophy in Acquired Trochlear Nerve Palsy

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Isolated trochlear nerve palsy is the most common cause of acquired vertical strabismus in adults. The cause often remains unknown despite appropriate investigation. Most patients recover spontaneously within four months. Occasionally, patients with idiopathic trochlear nerve palsy do not regain superior oblique muscle function. In these cases, unrelenting vertical diplopia without clear cause may prompt neuroimaging studies.

A 62-year-old man reported the onset of vertical diplopia four years before an examination. To achieve fusion he assumed a left head tilt and used spectacles with vertical and base out prisms. For three months the patient noted constant diplopia while reading through his bifocal segment. Without correction the patient had 14 prism diopters of right hypertropia and a small esotropia in primary gaze. The right hypertropia increased to 20 prism diopters on downgaze. The hypertropia also increased on left gaze and right head tilt. Exotropia was noted by Maddox rod testing.

We obtained a magnetic resonance scan of the

Fig. 1 (Horton and associates). Serial 3-mm, T1-weighted, fat-saturation, gadolinium-enhanced images show selective atrophy of the right superior oblique muscle (small arrows). The left superior oblique muscle is normal in size (large arrows).
brain and orbits to search for a structural lesion involving the right troclear nerve. None was found. However, views through the orbits disclosed atrophy of the right superior oblique muscle. In T₁-weighted coronal sections from the orbital apex to the back of the globe the profile of the right superior oblique muscle appeared severely reduced in size when compared with the normal left superior oblique muscle (Fig. 1). Atrophy was also apparent in the axial plane (Fig. 2).

Usually the diagnosis of troclear nerve palsy can be made securely by clinical examination alone. Ocular myasthenia, however, can occur infrequently as a chronic, isolated extraocular muscle paresis. In myasthenia an affected muscle should appear essentially normal in caliber on neuroimaging. In acquired vertical strabismus from a supranuclear lesion extraocular muscle size will also remain normal.

Injury to the motor nerve innervating a striated muscle can result in loss of up to 80% of muscle bulk because of the wasting of individual muscle fibers. This process is characterized by shrinkage of sarcotubes, loss of myofilaments, and replacement by fatty tissue. The degree of muscle atrophy seen in our patient is compatible with denervation atrophy. Absence of the superior oblique muscle has been shown previously by computed tomographic scans in congenital superior oblique muscle palsy. Attention to muscle size within the orbit can be of diagnostic value when performing magnetic resonance scanning on a patient with persistent vertical strabismus. Atrophy of the superior oblique muscle confirms palsy of its nerve. When muscle atrophy is severe, functional recovery is improbable.

References