Reduced Apparent Diffusion Coefficient in Neuromyelitis Optica–Associated Optic Neuropathy

In their excellent review of neuromyelitis optica (NMO), Morrow and Wingerchuk (1) discuss the criteria that distinguish this condition from multiple sclerosis. Here, we describe diffusion-weighted magnetic resonance imaging (DWI) of the optic nerve in a patient with NMO-associated optic neuropathy.

A 21-year-old man developed vision loss in the right eye that was diagnosed as optic neuritis. After a delay of 5 months, he was referred for neuro-ophthalmology evaluation. Visual acuity was 20/400 in the right eye and 20/20 in the left eye. Visual field testing showed severe depression in the right eye and normal function in the left eye. The right optic disc was pale and the left optic disc appeared normal. Magnetic resonance imaging (MRI) revealed slight atrophy of the right optic nerve, but no demyelinating plaques. A year later, the patient experienced rapidly progressive vision loss in the left eye. The acuity was 20/400 in the right eye and 20/800 in the left eye, with generalized visual field loss in the left eye. The right optic disc was pale and the left optic disc was edematous. The patient was treated intravenously with methylprednisolone, followed by oral prednisone. Serum and cerebrospinal fluid were positive for anti-AQP4 immunoglobulin G, as determined by a cell-based fluorescence assay (2). Three weeks later, acuity in the left eye was 20/80, with improvement in the visual field.

MRI of the orbits 4 weeks after the onset of vision loss showed enhancement of the left optic nerve (Fig. 1A), and DWI revealed increased signal along the course of the left optic nerve (Fig. 1B). The apparent diffusion coefficient (ADC) map demonstrated hypointense signal within the left optic nerve (Fig. 1C). The ADC value along a representative 45.6 mm² segment of the left optic nerve measured 0.875 × 10⁻³ mm²/s and along a 48.4 mm² segment of the right optic nerve was 1.63 × 10⁻³ mm²/s. DWI obtained a year earlier, when visual function in the left eye was normal, yielded an ADC in the left optic nerve of 1.03 × 10⁻³ mm²/s.

In patients with NMO, immunohistochemical analysis of lesions has shown local depletion of aquaporin-4 channels (3,4). Loss of AQP4 channels in the optic nerve may contribute to the reduction of water movement. It remains to be determined if our finding of restricted diffusion will be replicated in other patients with NMO-optic neuropathy. A broader issue is whether DWI will prove useful in differentiating different types of optic neuropathy. It has been proposed that DWI may disentangle ischemic optic neuropathy from optic neuritis, which can have overlapping clinical profiles (5). In a recent study, DWI was abnormal in 5/5 patients with acute ischemic optic neuropathy, but only in 2/25 patients with acute optic neuritis (6). However, other studies have concluded that impaired diffusion is a common feature in optic neuritis (7,8). Further MR studies are needed to clarify the prevalence, time course, and correlation with severity of findings of DWI abnormalities in ischemic optic neuropathy, optic neuritis, and NMO-optic neuropathy.

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FIG. 1. A. Postcontrast T1 axial 3-T magnetic resonance imaging with fat saturation demonstrates enhancement of the left optic nerve (arrow). B. Diffusion-weighted image shows bright signal in the left optic nerve, corresponding to the area of contrast enhancement. C. Apparent diffusion coefficient map reveals dark signal (arrow) in the left optic nerve consistent with restricted diffusion.
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Supported by National Eye Institute and Research to Prevent Blindness.

The authors report no conflicts of interest.

REFERENCES