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Diffusion-weighted imaging hyperintensity and low apparent diffusion coefficient of the optic nerve in myelin oligodendrocyte glycoprotein–IgG optic neuritis

Myelin oligodendrocyte glycoprotein (MOG)–IgG has emerged as a reproducible marker for patients with optic neuritis or those with an aquaporin-4 (AQP4)–IgG–negative neuromyelitis optica spectrum disorders (NMOSD) phenotype.[1] MOG-IgG optic neuritis tends to have a more favourable prognosis compared with those with AQP4-IgG, but poor visual outcomes may occur in a minority of patients with this condition.[2] Certain magnetic resonance imaging (MRI) features may suggest MOG-IgG disease—especially perineural enhancement and longitudinal involvement.[2] We describe a case of MOG-IgG optic neuritis with diffusion-weighted imaging (DWI) hyperintensity and low apparent diffusion coefficient (ADC) of the optic nerve and incomplete visual recovery, suggesting that these imaging features may confer a poorer visual outcome.

A 73-year-old woman presented with a 1-month history of gradually declining vision in her left eye with significant impairment in colour vision and pain with eye movements. Her medical history was remarkable for an episode of right optic neuritis 11 years prior, which led to a decline in vision to counting fingers and pain with eye movements. AQP4-IgG was undetectable and treatment with high-dose corticosteroids resulted in significant improvement in visual acuity. The patient also had a history of dyslipidemia, hypertension, deep vein thrombosis, and breast cancer, and was taking candesartan 8 mg, rivaroxaban 20 mg, and atorvastatin 20 mg daily.

On examination, visual acuity was 20/30 in the right eye and 20/200 in the left eye. There was a left relative afferent pupillary defect and decreased colour vision in the left eye. Dilated fundus examination revealed left greater than right temporal pallor of the optic nerves (Fig. 1A). No additional abnormalities were identified on neurological examination. Humphrey 24-2 SITA-Fast visual field testing was normal in the right eye and demonstrated generalized depression in the left eye, with a mean deviation of −21.47 dB (Fig. 1B). Optical coherence tomography of the retinal nerve fibre layer showed an average thickness of 79 μm on the right and 77 μm on the left with temporal thinning in both eyes. The left intraorbital optic nerve exhibited increased T2-signal, contrast enhancement on T1 weighted-imaging, and hyperintensity on DWI with a corresponding decrease in ADC signal (Fig. 2). The patient was seropositive for MOG-IgG (moderate positivity) and seronegative for AQP4-IgG antibodies. Treatment with intravenous methylprednisolone 1g daily for 5 days resulted in minimal improvement. The patient underwent 7 complete sessions of plasmapheresis, which led to only partial restoration of visual function. At the 3-month follow-up, she had a visual acuity of 20/70 and a constricted visual field with a mean deviation of −14.94 dB. The left optic nerve continued to demonstrate temporal pallor, and there was further reduction in the thickness of the retinal nerve fibre layer on optical coherence tomography to 66 μm in the left eye.

DWI provides functional information about the diffusion characteristics of tissues and is useful for identifying conditions that cause the redistribution of water between intracellular and extracellular compartments.[3] Although DWI has traditionally been applied in the setting of acute brain ischemia, several studies have assessed its role in the evaluation of

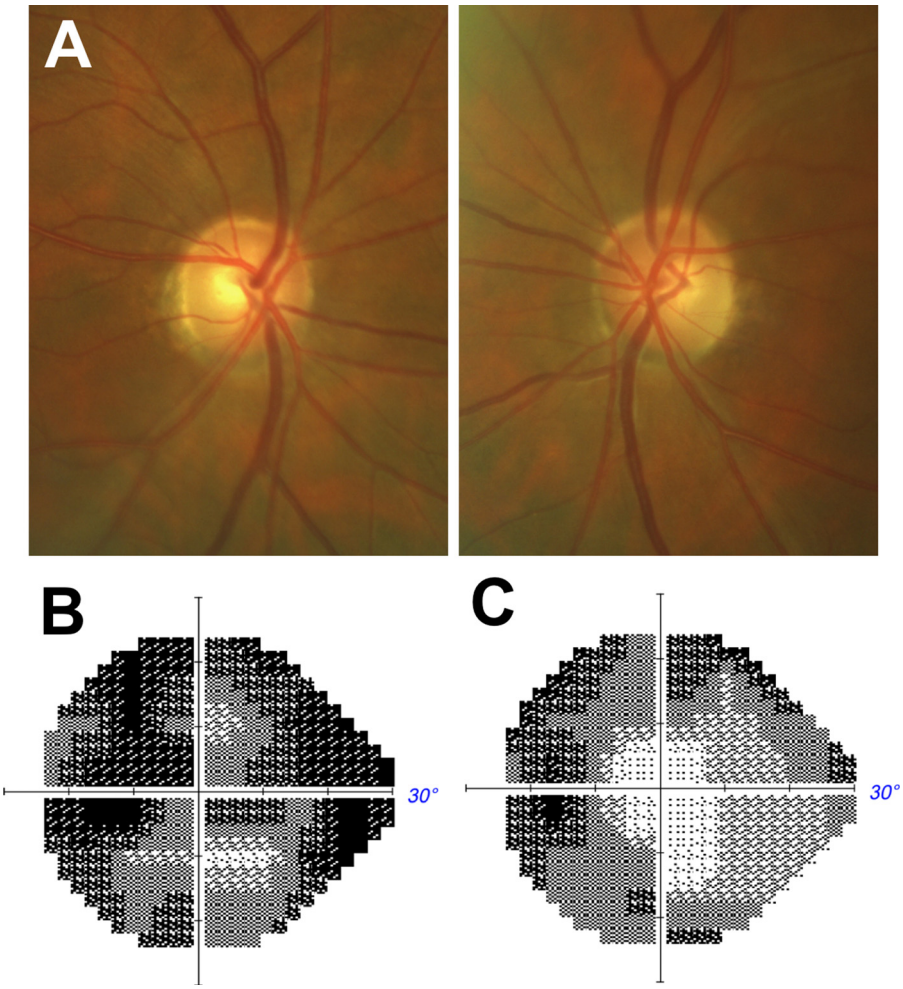

Fig. 1—Fundus photographs of both optic nerves at presentation (A) demonstrating mild left greater than right temporal pallor of the optic nerves. Humphrey 24-2 SITA-Fast visual field of the left eye at presentation (B) demonstrating generalized depression with a mean deviation of −21.47 dB, and at 3-month follow-up (C) demonstrating a constricted visual field with a mean deviation of −14.94 dB.

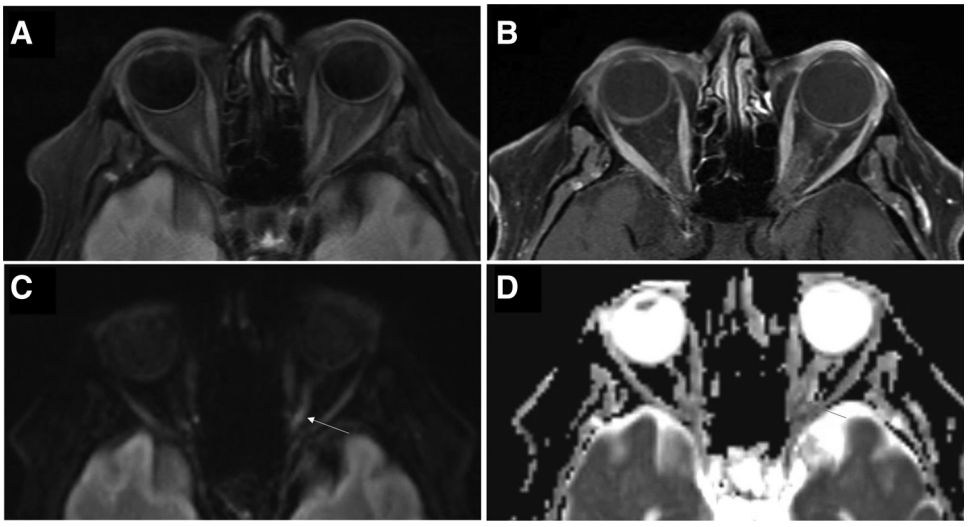

Fig. 2—Magnetic resonance imaging of the orbits with Fluid-attenuated inversion recovery (A) T1-postcontrast with fat suppression (B) diffusion-weighted imaging (DWI) (C) and apparent diffusion coefficient (ADC) (D) demonstrating high DWI signal (yellow arrow) and low ADC signal (red arrow) in the left optic nerve.

optic neuritis. Acute optic neuritis has been reported to cause DWI hyperintensity and various signal changes on ADC maps of the optic nerve.[4–7] DWI abnormalities and corresponding ADC values may have prognostic implications, as a lower ADC value during acute optic neuritis was associated with worse visual outcomes and more severe thinning of the retinal nerve fibre layer and macular ganglion cell complex 6 months after initial presentation.[5] ADC values may also vary with the chronicity of disease with more chronic cases having higher values.[7] DWI hyperintensities and postcontrast enhancement exhibit similar localizations within inflamed optic nerves,[4] suggesting that DWI may be an appropriate alternative for identifying acute optic neuritis when MRI with contrast is contraindicated. DWI has a sensitivity of 77%−83%, specificity of 80%−84%, and diagnostic accuracy of 80%−83% when used to evaluate optic neuritis.[6,8]

DWI abnormalities have recently been shown to aid in the differentiation of optic nerve pathologies. Diffusion restriction of the intraorbital optic nerve was predictive of acute optic neuritis over nonarteritic anterior ischemic optic neuropathy (NAION), whereas postcontrast enhancement of the optic disc in the absence of DWI findings was predictive of NAION.[4] The presence of postcontrast enhancement of the optic chiasm and lower ADC values were found to be the most important imaging characteristics for differentiating between NMOSD and optic neuritis caused by multiple sclerosis.[5,9] When applied as diagnostic criteria for NMOSD, the presence of low ADC values or optic chiasm involvement had a specificity of 78.1%, sensitivity of 77.8%, and diagnostic accuracy of 77.9%.[9] The ADC value was still low 4 weeks after symptom onset in our patient, and this was similar to a previous case of AQP4-optic neuritis, which was presumably due to lasting depletion of water channels.[10]

The clinical utility of DWI has yet to be directly explored in patients with MOG-optic neuritis. Here we report the first case of confirmed acute MOG-IgG optic neuritis resulting in DWI hyperintensity and corresponding low ADC signal of the intraorbital optic nerve with a similar localization to that of postcontrast enhancement on T1-weighted MRI. Although MOG-IgG optic neuritis is typically associated with good visual outcomes, in the present case, intravenous corticosteroids and aggressive treatment with plasmapheresis led to only modest improvement in visual function at 3 months. This case suggests that DWI may be an appropriate alternative imaging modality in patients with suspected MOG-IgG optic neuritis and contraindications to the use of gadolinium contrast agents. It also highlights the possibility that DWI hyperintensity and low ADC signal of the optic nerve may indicate poor visual prognosis in MOG-IgG optic neuritis as suggested in AQP4-IgG optic neuritis.[5] Further studies are required to characterize the DWI abnormalities seen in MOG-IgG optic neuritis and evaluate the diagnostic and prognostic capabilities of this imaging modality.

Footnotes and Disclosure:

The patient consented to the publishing of this information and signed a form verifying her consent.

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