was begun, and 3 weeks later, he had recovered completely. Several follow-up MRIs were performed. The most recent MRI, 1.5 years after the onset of symptoms, revealed an increase in number and extent of the white matter hyperintensities (Fig. 2). Glatiramer acetate therapy was initiated.

On occasion, a one-and-a-half syndrome can be accompanied by a facial paresis if the fascicle or nucleus of the seventh cranial nerve in the lower part of the dorsal pontine tegmentum is also affected. Eggenberger (2) designated this as eight-and-a-half (1.5 + 7) syndrome. As mentioned by Connors et al (1), variations of this syndrome caused by pathology of the dorsal pontine tegmentum have since been described including a combination of a one-and-a-half syndrome and a bilateral peripheral facial paresis which Bae and Song (3) designated fifteen-and-a-half (1.5 + 7 + 7) syndrome.

Only 3 cases of isolated eight-and-a-half syndrome caused by MS have been described in the literature (4,5). In one of these cases (4), the eight-and-a-half syndrome was, as in our patient, the initial symptom of MS. A 16 syndrome caused by MS has not been reported previously.

The authors report no conflicts of interest.

REFERENCES


Optic Disc Edema and Optic Nerve Head Drusen

We are concerned about the conclusions reported by Sarac et al (1) in their article entitled “Differentiation of optic disc edema from optic nerve head drusen with spectral-domain optical coherence tomography” and the application of these conclusions to clinical practice.

Sarac et al seek to answer an old and important neuro-ophthalmic question: How can one distinguish between optic disc edema and optic nerve head drusen? Most clinicians have no trouble diagnosing advanced optic disc edema, such as Frisen Stages 3, 4, and 5 (2). Most clinicians have no trouble diagnosing optic nerve head drusen that are visible on ophthalmoscopy (“visible drusen”). Where clinicians do find themselves in a quandary is when they are asked to distinguish mild cases of optic disc edema (Frisen Stages 0, 1, and 2) from optic nerve head drusen that are not visible on ophthalmoscopy ("buried drusen"). Clinicians frequently look to new tools such as optical coherence tomography (OCT) to help sort out these difficult situations.

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Using time domain OCT, Karam and Hedges (3) concluded that OCT could not be used to differentiate individuals with congenitally crowded optic nerves from individuals with mild papilledema. Conversely, Johnson et al (4) argued that OCT could be used to differentiate optic disc edema from optic nerve head drusen, but these authors included subjects with disc edema that was “mild, moderate, and severe” and drusen that were both visible and buried. These authors also included subjects with papilledema, ischemic optic neuropathy, and optic neuritis in their study population. Lee et al (5) claimed that spectral domain OCT may be used to differentiate optic disc edema from optic nerve head drusen, but they also included subjects in whom the edema ranged from “subtle to severe,” and did not state the etiology of the disc edema in their subjects.

In the study by Sarac et al, the optic nerve head drusen group contained eyes with both visible and buried drusen. The optic disc edema group was also heterogeneous, containing subjects with “subtle to severe” optic nerve swelling. In addition, the optic disc edema group contained subjects with papilledema, nonarteritic anterior ischemic optic neuropathy, and optic neuritis. However, most clinicians would have no difficulty distinguishing a patient with optic nerve head drusen from a patient with anterior ischemic optic neuropathy or optic neuritis. We do not dispute the results reported by Sarac et al. Our concern is that clinicians reading this article will inappropriately extrapolate these conclusions to clinical care. When faced with a patient in whom the differential diagnosis includes mild optic disc edema and buried optic nerve head drusen, the guidelines proposed by Sarac et al may not hold. Their study population was not relevant to the clinical question being asked.

Currently, it appears that the conclusions reached by Karam and Hedges (3) still hold. Until a study is designed with a clinically relevant population of subjects, the question of the utility of OCT in the differential diagnosis of optic disc edema and optic nerve head drusen remains unanswered.

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REFERENCES

Subclinical Optic Neuritis in Neuromyelitis Optica

We read with great interest the review of neuromyelitis optica (NMO) by Morrow and Wingerchuk (1). Even with proposed diagnostic criteria (2), establishing the diagnosis of NMO may be difficult. We describe a patient with white matter cerebral lesions, myelitis, and subclinical optic neuritis with negative NMO-IgG at the initial presentation. The diagnosis of NMO became certain 5 months later when the patient developed overt bilateral optic neuritis and a positive NMO-IgG antibody.

A 46-year-old woman experienced the onset of dizziness, nausea, and vomiting. Neurological examination was unremarkable except for horizontal gaze evoked nystagmus and mild weakness in her right leg. Muscle strength in the right lower extremity was at 4/5, patellar deep tendon reflexes were hypoactive, and the plantar reflex was indifferent on the right side. Vision was 20/20 bilaterally with normal color vision, funduscopic, and visual evoked potentials. Automated visual fields demonstrated mild generalized depression (Fig. 1).

Brain magnetic resonance imaging (MRI) revealed enhancement of the entire length of the left optic nerve (Figs. 2A and 2B) hyperintensities in the dorsal medulla, around the fourth ventricle, in the periaqueductal gray matter, mamillary bodies, the thalamus, and in the vicinity of the third ventricle (Fig. 2C). MRI of the spine