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Contrast-Enhanced MR Imaging of Optic Nerve Lesions in Patients with Acute Optic Neuritis

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The purpose of this study was to determine if administration of gadopentetate dimeglumine aids in the MR detection of optic nerve lesions in patients with acute optic neuritis and to establish an efficient MR imaging protocol to effectively demonstrate such lesions. Patients with acutely decreased visual acuity were referred for MR imaging of the brain and orbits. Predominantly T1-weighted images were obtained in axial and coronal planes with and without contrast administration. Enhancing lesions were observed in the optic nerve (7/14 patients) and optic chiasm (2/14), and associated white matter lesions were seen elsewhere in the brain (5/19).

Our results indicate that administration of gadopentetate dimeglumine aids in the MR detection of lesions of the optic nerve and optic chiasm. Applicability of our MR imaging protocol was confirmed by the demonstration of these lesions and by the disseminated white matter lesions seen simultaneously elsewhere in the brain.

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MR imaging is the most sensitive scanning technique to date for demonstrating demyelinating lesions associated with multiple sclerosis (MS) [1]. Optic neuritis is a clinically symptomatic inflammatory disease of the optic nerve that is analogous to MS-associated active inflammatory white matter lesions elsewhere in the CNS. Optic neuritis can be the initial presenting symptom of MS, and previous studies have demonstrated progression of the symptoms of optic neuritis to clinical MS in 11.5-87% of cases [2]. It is, therefore, important to detect optic nerve lesions as early as possible. These areas of inflammation and edema may cause a transient disruption of the blood-brain barrier [3], which should permit leakage of contrast material and, thus, enhancement on MR images.

Fourteen patients were referred by a neuroophthalmologist for MR imaging of the orbits and brain after acute onset of symptoms of optic neuritis. Of these, seven patients were found to have enhancing optic nerve lesions compatible with acute demyelination. Similar results have previously been noted in smaller series of patients [4, 5]. However, these earlier studies either included contrast enhancement of optic nerves anecdotally [4] or did not limit the study to optic neuritis associated with MS [5].

Materials and Methods

Fourteen patients were referred for MR imaging after acute onset of visual disturbances suggestive of optic neuritis. The MR study was performed on a 1.5-T system (GE Signa, Milwaukee, WI). Protocol for imaging the orbits and brain consists of 5-mm-thick contiguous axial and coronal mixed and T2-weighted, 2400/30,90/1 (TR/TE/excitations), images with a 256 × 256 matrix. T1-weighted images (600/20/2) were obtained axially at 3 mm with a 256 × 256 matrix. T1-weighted images were obtained 3 min after IV bolus injection of gadopentetate dimeglumine at 0.1 mmol/kg. The axial scanning orientation was parallel to the optic nerve and chiasmal complex (10-15° below the orbitomeatal line). Selective postcontrast coronal images were added when deemed necessary.

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TABLE 1: Sites of Contrast Enhancement

Patient No.	Age (years)	Sex	Optic Nerve	Optic Chiasm	Other Sites
1	37	M	+ (R)	+	+ R periventricular
2	31	M	+ (R)	+	+ L centrum semiovale, external capsule
3	45	F	+ (R)		
4	27	M	+ (L)		
5	37	F	+ (L and R)		+ R parietal lobe
6	44	F	+ (R)		
7	37	F		+	+ L corona radiata
8	46	F			+ Bilateral deep parietal lobe

Note.—R = right, L = left.

Results

Of the 14 patients who had MR imaging, six had normal studies; seven (50%) had intraparenchymal brain lesions suggestive of MS, of which six had enhancing lesions of the optic nerve complex; one patient had enhancement of the optic nerve complex but did not have any other lesions; and one patient had enhancing cerebral lesions without any enhancement of the optic nerve complex. Specific sites of enhancing and nonenhancing cerebral lesions suggestive of MS are summarized in Table 1.

Patient 1 had T2-weighted high-signal plaque near the posterior horn of the right lateral ventricle. High-signal abnormalities on T2-weighted images were also seen in the right optic tract extending into the optic chiasm. All these areas were noted to enhance with contrast administration (Fig. 1). Patient 2 had high-signal foci that were seen on T2-weighted images in the centrum semiovale. No signal abnormalities were noted in the optic nerve or chiasm, which did show enhancement after contrast administration (Fig. 2). Patient 3 showed multiple bilateral periventricular high-signal lesions on mixed and T2-weighted images, some of which enhanced with contrast administration. There was also enhancement of the right optic nerve when compared with the left (Fig. 3). Patient 4 had a single nonenhancing demyelinating lesion seen in the brainstem. Also noted was slight enhancement,

after contrast administration, of the left optic nerve within the optic canal. Patient 5 had multiple high T2-signal foci scattered throughout the deep white matter, as well as mild enhancement of the optic nerves, left greater than right. Patient 6 had a normal unenhanced study, although there was some enhancement of the proximal portion of the extracranial right optic nerve. Patient 7 had enhancing lesions in both the cerebrum and left optic nerve. Patient 8 had several enhancing cerebral lesions suggestive of MS, but no lesions of the optic nerve complex were visualized.

Discussion

Optic neuritis is a relatively nonspecific clinical term describing inflammation of the optic nerve, which causes impaired visual acuity, pain, abnormal color vision, or afferent pupillary defects. Optic neuritis can be associated with a number of clinical diseases, including ischemia, systemic lupus erythematosus, syphilis, radiation, and sarcoid, but it is most commonly associated with MS, either in isolation or as the initial presenting symptom [2]. Therein lies the importance of the association of MS and optic neuritis.

Gadopentetate dimeglumine detects the actively demyelinating lesions of MS. Active optic neuritis in MS is associated with perivenous inflammation and destruction of myelin and disruption of the blood-brain barrier [3]. These focal areas of blood-brain barrier disruption in the optic nerve allow parenchymal leakage of contrast agent, as in the brain, and thus enable local enhancement of the involved tissue. Since the optic nerve is actually part of the CNS and possesses a blood-brain barrier, any process that causes blood-brain barrier disruption would be expected to show contrast enhancement on MR images.

Few reports of contrast enhancement on MR images of the optic nerve, per se, have appeared in the literature. Miller et al. [4] have used inversion recovery MR techniques to scan the orbits in patients with optic neuritis. Using orbital surface coils, they observed focal MR lesions in 37 of 44 symptomatic nerves. The majority of these lesions were located in the

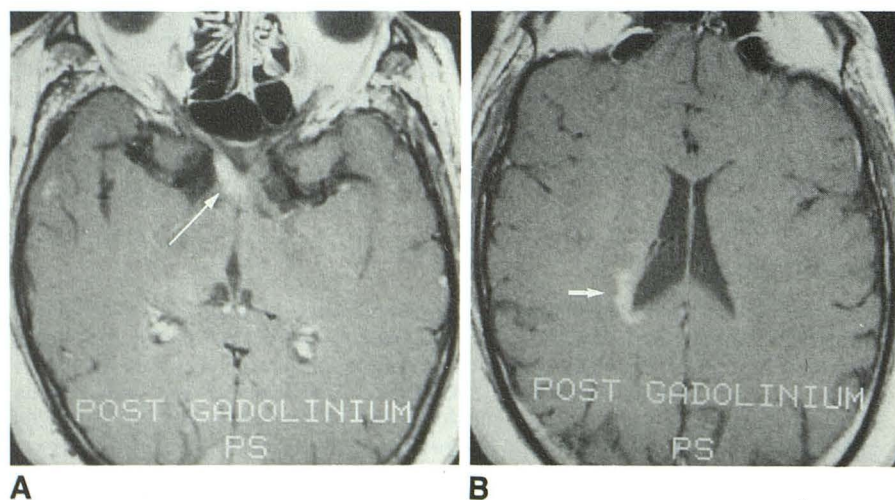


Fig. 1.—Patient 1.

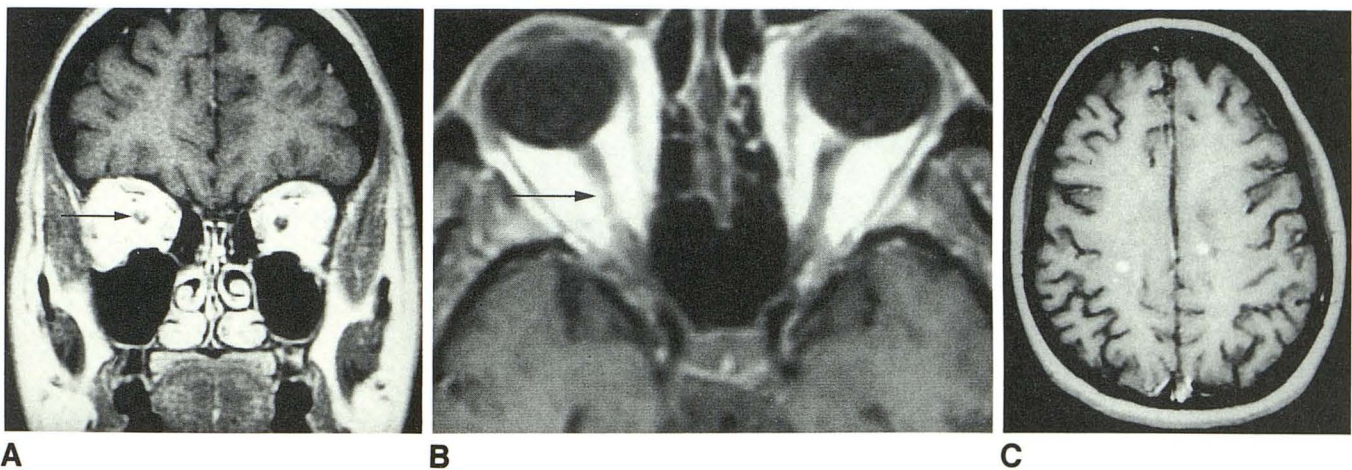
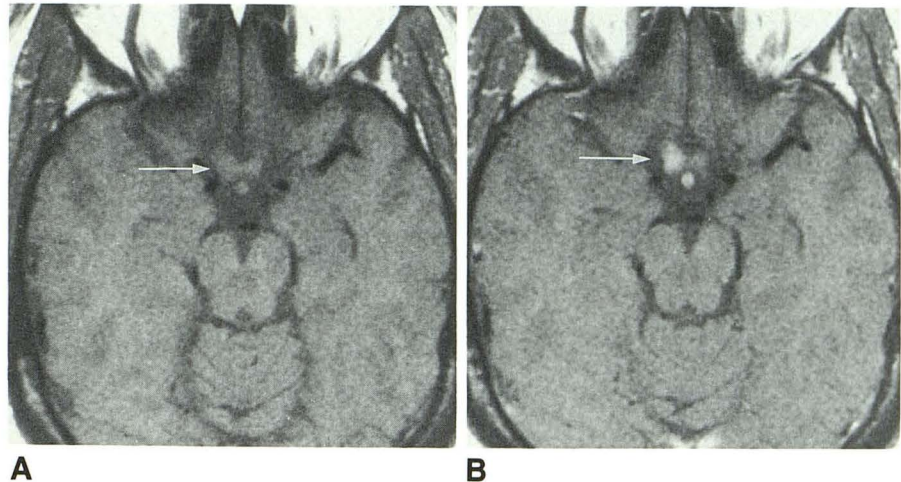
A, Axial MR image (600/20) 3 min after contrast infusion shows right optic nerve (arrow) extending to optic chiasm. Note swelling of right side of chiasm. No signal brightness was present on corresponding unenhanced scan (not shown).

B, Axial MR image (600/20) 3 min after contrast infusion shows periventricular enhancing lesion (arrow) suggestive of multiple sclerosis.

Fig. 2.—Patient 2.

A, Axial MR image (600/20) through optic nerve and chiasm shows area with mild swelling of right optic nerve (arrow).

B, Axial MR image (600/20) 3 min after contrast infusion shows enhancing lesion of optic chiasm (arrow) (same slice level as A).

**Fig. 3.—Patient 3.**

A, Postcontrast coronal MR image (600/20) shows enhancing lesion of right optic nerve (arrow).

B, Postcontrast axial MR image (600/20) shows enhancing lesion of right optic nerve (arrow).

C, Postcontrast axial MR image (600/20) shows several enhancing lesions suggestive of multiple sclerosis.

extracranial portion of the optic nerve. Only one chiasmal lesion was demonstrated, and this was seen on a T1-weighted postcontrast image.

Inversion recovery MR techniques utilizing surface coils are rather insensitive in the region of the intracranial optic nerve, chiasm, and cerebral parenchyma owing to lower signal-to-noise ratio caused by increased distance of these structures from the surface coil. Chiasm and intracranial optic nerve segments are better visualized with the contrast-enhanced technique used in the present study. Guy et al. [5] have recently reported optic nerve enhancement in seven of 13 patients, of whom only three were thought to have MS. We believe our cases are complementary and support the fact that contrast-enhanced MR studies increase the detection of optic nerve abnormalities in patients with optic neuritis.

Our results confirm that the orbits and brain can be scanned most efficiently by using T1- and T2-weighted postcontrast sequences with a head coil. A T1-weighted (600/20) spin-

echo (SE) sequence combined with a T2-weighted (2400/90) SE sequence afforded visualization of both the acute active lesions and the chronic stable lesions in the intracranial and chiasmal portions of the optic nerve, as well as in the white matter throughout the brain. Our protocol is designed to evaluate the intracranial and extracranial optic nerve, the optic chiasm, and the brain concurrently. Therefore, the patient is not subjected to separate scans of the orbits and brain. This protocol demonstrates early optic nerve and other disseminated lesions, allows better appreciation of global effects of the disease, and provides supportive evidence for a definitive diagnosis of MS.

A problem still exists with reference to differential diagnosis. As noted by Guy et al. [5], other diseases, such as sarcoidosis and radiation-induced retrobulbar neuritis, can look exactly like optic neuritis with MS. Clinical factors are, therefore, crucial in establishing the precise diagnosis. When unambiguous intracranial lesions of MS are present, the diagnosis is

simple and clear-cut. In clinical practice, however, most patients with acute optic neuritis have no additional symptoms or signs to suggest MS. Indeed, since some experts feel that optic neuritis is not necessarily an early manifestation of MS, demonstration of optic neuritis plus cerebral lesions is diagnostically helpful. Up to 50% of all patients with optic neuritis plus MS may not initially have associated plaques in the brain, and contrast enhancement may be particularly helpful in these instances (R. Beck, personal communication and unpublished data) and [6].

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