

Abducens Neuropathy Detected by Three-Dimensional Magnetic Resonance Imaging Using a Multiplanar Reconstruction Technique With Gadolinium-DTPA Enhancement

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Abstract: Three-dimensional magnetic resonance imaging (3D MRI) using a multiplanar reconstruction technique with gadolinium diethylenetriamine pentaacetic acid (DTPA) enhancement was used for assessment of a 20-year-old woman who had sudden complete restriction of abduction in the right eye. The 3D MRI revealed abducens nerve enhancement in the cisternal portion. An enhanced lesion was also observed in the right lower pons at the pontomedullary junction. These clinical and 3D-MRI findings were diagnosed as right abducens palsy due to abducens neuropathy. Observation of the entire pathway of the cisternal portion of the cranial nerves can be extremely useful in patients with ophthalmoplegia. **Jpn J Ophthalmol 1997;41:59-61** © 1997 Japanese Ophthalmological Society

Key Words: Abducens nerve, abducens palsy, gadolinium-DTPA-enhanced magnetic resonance imaging.

Introduction

Three-dimensional magnetic resonance imaging (3D MRI) using a multiplanar reconstruction technique with gadolinium (Gd)-diethylenetriamine pentaacetic acid (DTPA) enhancement, which gives high-quality images for delineating the cisternal portion of the cranial nerves, has recently been developed.¹ This technique is useful for contrast enhancement of the cranial nerves (optic,^{2,3} oculomotor,⁴ trigeminal,⁵ and facial^{6,7}). We studied Gd-DTPA enhancement of the abducens nerve in a patient with abducens palsy. We also studied the correlation of the abducens nerve enhancement and the clinical course of abducens palsy.

Case Report

A 20-year-old woman complained of right retro-ocular pain, dull headache, and sudden double vision

on September 30, 1992. Family history and medical history were unremarkable. Ophthalmologic examination revealed a complete restriction of abduction in the right eye, consistent with right abducens palsy. The right oculomotor and trochlear nerves were intact. The left eye was abducted slightly beyond the midline, but movement was normal. The pupillary reflexes were normal. No nystagmus was observed. Visual acuity was 0.1 ($1.0 \times -2.00D$) in the right eye and 0.1 ($1.0 \times -2.25D$) in the left eye. Visual field examination with Goldmann perimetry was normal in both eyes; intraocular pressure measured with Goldmann tonometry was 18 mm Hg in both eyes. The anterior segment and the lens were normal on slit-lamp examination. Ophthalmoscopic examination found no abnormality in the fundi.

Physical examination results were normal except for slight hypesthesia in the trigeminal region. Computed tomography (CT) of the brain and cranial angiography were normal. Hematology was normal except for a mild increase in white blood cells (12,900 μ L). Cerebrospinal fluid (CSF) pressure was normal with normal mononuclear cell counts (14/3), glucose

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**A****B**

Figure 1. First contrast-enhanced 3D-MR images. Axial (**A**) and sagittal (**B**) reformatted images through the right abducens nerve show obvious contrast enhancement in both the right abducens nerve (small arrow) and the right lower pons at the pontomedullary junction (large arrow).

(71 mg/dL) and protein (27 mg/dL). An oligoclonal IgG band was seen in the CSF, but there were no other findings to suggest a diagnosis of multiple sclerosis.

The first MRI examination was done on October 9, 1992 with the 1.5 T SIGNA system (General Electric, USA). Contrast-enhanced (CE) 3D MRI by spoiled gradient recalled acquisition (SPGR) in a

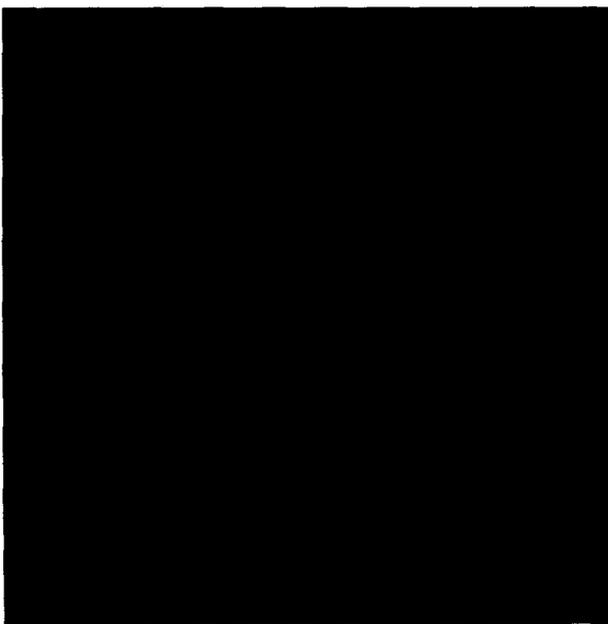
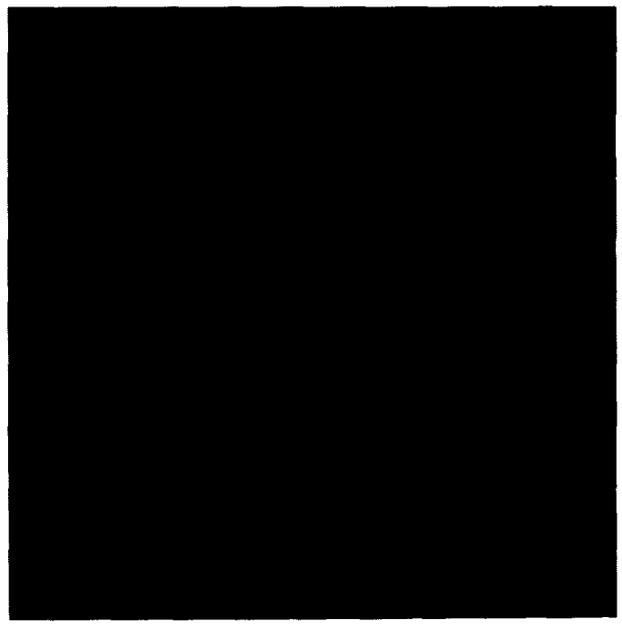
**A****B**

Figure 2. Fourth contrast-enhanced 3D-MR images. Axial (**A**) and sagittal (**B**) reformatted images show low intensity at the pontomedullary junction (large arrow) or in the abducens nerve (small arrow).

steady state with a contrast medium (0.1–0.15 mmol/kg gadolinium-DPTA) was done after T1-weighted axial images with spin echo sequences were completed. Proton density (PD)-weighted and T2-weighted axial images with fast spin echo sequences were taken on each examination. SPGR sequence specifications were: 24–26 msec repetition time; 4.5 msec echo time; 35° flip angle; 16 cm field of view; 1.0 mm section thickness; 60 mm slab thickness with two excitations; 256/192 matrices with flow compensation. Total time was approximately 10 minutes. The multiplanar reconstruction technique was used to delineate the entire pathway of the cisternal portion of the abducens nerve. A precontrast study showed only a faint high-intensity area at the right lower pons on T2-weighted images. Contrast-enhanced 3D-MRI images clearly showed the right abducens enhancement of the entire path of the cisternal portion as well as the enhanced lesion in the right lower pons (Figure 1).

The patient was treated with methylprednisolone (8 mg t.i.d. × 16 days) during the following 2 weeks, with improvement of the right abduction. Three follow-up MRI examinations were done with the same parameters as the first. Nineteen days after the initial visit, there was a 50% improvement in right abduction; enhancement of the right abducens nerve and lower pons was diminished but still evident. Twenty-eight days after the first visit, right abduction was normal, right abducens enhancement was still apparent, but the lower pons lesion was less distinct. On January 31, 1993, the abducens enhancement had completely disappeared and the right lower pons lesion was a low-intensity lesion without enhancement (Figure 2). There was no change in the faint high-intensity lesion seen on the T2-weighted images in all follow-up examinations.

Discussion

This is the first description of Gd-DTPA enhancement of the cisternal portion of the abducens nerve using contrast enhanced 3D MRI for high-quality images. This imaging, used in conjunction with clinical observations, allowed us to diagnose the right abducens palsy due to abducens neuropathy. Mark et al⁴ reported that enhancement of the third cranial

nerve was always abnormal, indicating an underlying or a neoplastic pathology. We agree with this statement, and stress that enhancement of the cisternal portion of the cranial nerve is always abnormal, based on our observation by 3D MRI of over 2000 patients without ophthalmoplegia. In no case was there enhancement of the cisternal portion of the cranial nerve.

The mechanism of enhancement is uncertain, but may involve hypervascularity in the cranial nerve structure. The blood-nerve barrier can easily be disrupted by neoplastic or inflammatory processes as well as by cerebral infarction, brain contusion, or multiple sclerosis.¹⁻⁷ In some patients with unilateral ophthalmoplegia, contralateral or other cranial nerve enhancement was seen in the nerve without clinically apparent palsy; the blood-nerve barrier may have been disrupted, but not enough to cause dysfunction. In our patient, the blood-nerve barrier disruption in the abducens nerve may be the cause of the abducens neuropathy.

Observation of the entire pathway of the cisternal portion of the cranial nerves, using this multiplanar reconstruction technique with contrast enhanced 3D MRI should be extremely useful in evaluating patients with ophthalmoplegia.

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