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Gd-DTPA Enhancement of the Cisternal Portion of the Oculomotor Nerve on MR Imaging

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PURPOSE: To describe a radiographic finding—enhancement of the cisternal portion of the third cranial nerve on postcontrast MR—and to correlate it with patients' clinical symptoms and ultimate diagnosis. **MATERIALS AND METHODS:** Thirteen consecutive patients with enhancement of the cisternal portion of the third cranial nerve on postcontrast MR were retrospectively identified; 50 control patients referred for pituitary microadenomas were also retrospectively reviewed. **FINDINGS:** The enhancement was bilateral in six patients and unilateral in seven patients. Four of the six patients with bilateral enhancement had unilateral oculomotor nerve palsies; none had bilateral third cranial nerve palsy. Five of the seven patients with unilateral enhancement had ipsilateral third nerve palsies. Of the nine patients with third nerve palsies, the pupil was involved in four patients. Follow-up studies were available in six patients, four of whom had third nerve palsy. Resolution of the enhancement correlated with resolution of the symptoms in two patients. The patients' underlying diagnoses were lymphoma (four), leukemia (one), viral meningitis (one), neurofibromatosis (two), inflammatory polyneuropathy-HIV related (one), ophthalmoplegic migraine (one), Tolosa-Hunt syndrome (one), coccidioidomycosis (one), and diabetes (one). No enhancement was seen in any of the controls. **CONCLUSION:** Enhancement of the cisternal segment of the third cranial nerve is always abnormal, revealing an underlying inflammatory or neoplastic process. However, it is not always associated with clinically apparent oculomotor nerve dysfunction.

Index terms: Nerves, oculomotor (III); Contrast media, paramagnetic; Nerves, anatomy; Migraine

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The clear depiction of the anatomic course of many of the cranial nerves has become routine on clinical magnetic resonance (MR) imaging. Although lesions of the cranial nerves have been identified by computed tomography (CT) (1), it is now generally recognized that the diagnostic work-up for suspected cranial nerve pathology must include MR. More recently, contrast enhancement of the second (2, 3), fifth (4), and seventh (5, 6) cranial nerves on MR has been described in patients with clinically apparent cranioneuropathies. Incidental enhancement of the seventh nerve has also been observed in asymptomatic patients (6). In this report, we describe gadopentetate dimeglumine diethylenetriamine pentaacetic acid (Gd-DTPA) enhancement of the cisternal segment of the third cranial nerve in 13 patients and correlate it with the patients' final diagnoses and clinical findings; 50 normal controls were also studied. The purpose of the paper is to answer two questions: 1) Is the enhancement of the third cranial nerve always abnormal or can it be seen in normal subjects? 2) Is the enhancement of the oculomotor nerve always associated with a clinically apparent third nerve dysfunction?

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Subjects and Methods

Our study included 13 consecutive positive studies (ie, studies that demonstrated enhancement of the cisternal segment of the third cranial nerve) collected from four institutions over a period of 3 years.

A 1.5-T system was used for imaging all patients. All patients underwent precontrast axial and/or coronal T1-weighted images and immediate postcontrast axial and/or coronal T1-weighted images (600-800/20-25/2), 3-mm thick sections with 0- to 1-mm gaps, 256 × 192–256 matrix, 20- to 22-cm field of view. All patients also underwent long TR images (2300/30-90/1), with 5-mm thick sections and 2.5-mm gap and 256 × 196 matrix. Gd-DTPA (Berlex Laboratories, Wayne, NJ) 0.1 mmol/kg was administered intravenously. The medical records of each patient were reviewed with particular attention to the neuroophthalmologic examination and to the patients' final diagnosis. Thickening of the third cranial nerve was diagnosed when one of the nerves appeared larger on the precontrast coronal images. No specific measurements were used. Enhancement of the third cranial nerve was diagnosed when an increase in the intensity of the nerve relative to the precontrast study occurred after contrast administration. In cases of unilateral enhancement, the enhancing nerve was brighter than the contralateral one.

For comparison, 50 consecutive adult patients with normal third cranial nerve function referred for suspected pituitary adenomas in one institution (Washington Hospital Center) were evaluated with pre- and postcontrast coronal T1-weighted images using a similar technique. These images were retrospectively evaluated by two neuroradiologists (A.S.M. and D.B.) with particular attention to the morphology and enhancement characteristics of the third cranial nerve.

Results

Our results describing the patients' age, sex, final diagnosis, presence or absence of bilateral or unilateral third cranial nerve palsy, involvement of the pupils, the presence of bilateral or unilateral enhancement, third nerve morphology, other associated symptoms, and associated MR findings, as well as the proof of diagnosis, are listed in Table 1. Of the 13 patients with third cranial nerve enhancement, six patients had bilateral enhancement (Figs. 1–3) and seven patients had unilateral enhancement (Figs. 4–8). Four of the six patients with bilateral enhancement had unilateral third cranial nerve palsy. None had bilateral third cranial nerve palsy. Five of the seven patients with unilateral enhancement had ipsilateral third cranial nerve palsies. Two patients with unilateral enhancement had neurofibromatosis and had normal third cranial nerve function. One patient had a cavernous sinus syndrome, including a third cranial nerve palsy. Of the nine patients with third cranial nerve palsies, the pupil was involved in four patients.

Unilateral thickening of the third cranial nerve was noted in four patients on a pre- and postgadolinium studies. Two patients had neurofibromatosis and a presumed schwannoma of the third cranial nerve. The other two had an inflammatory process and lymphoma, respectively, involving the third cranial nerve (patients 8 and 10). One of the patients with a thickened nerve had bilateral enhancement. The patient's symptoms were ipsilateral to the side of oculomotor nerve enlargement.

Follow-up studies were available in six patients (four symptomatic, two asymptomatic), some of whom had interval treatment (see Table 1). In four symptomatic patients, repeat MR studies demonstrated resolution of the enhancement correlating with resolution of the symptoms in three patients (who had lymphoma, Tolosa-Hunt, and ophthalmoplegic migraine, respectively); and persistence of symptoms in one patient with idiopathic (? diabetic, ? viral) oculomotor nerve palsy.

In the first asymptomatic patient who had leukemia, repeat MR demonstrated persistent but decreased bilateral enhancement of the oculomotor nerves following intrathecal chemotherapy. In the other asymptomatic patient who was HIV positive, the enhancement of the oculomotor nerve resolved following zidovudine (AZT, Burroughs-Wellcome Co., Research Triangle Park, NC) treatment.

No enhancement of the cisternal segment of the third cranial nerve was encountered on short TR/short TE images in any of the 50 patients referred for evaluation of pituitary microadenoma, all of whom had normal third cranial nerve function. The cisternal segment of the third cranial nerve could not be seen on the long TR images in the normal and abnormal patients because of their thickness (5 mm) and interslice gap (2.5 mm).

Discussion

The imaging of cranial neuropathies has been dramatically improved with the refinement of high resolution MR. Although morphologic alterations of the cranial nerves can sometimes be seen, many reports suggest that intravenous contrast plays an important role in the diagnosis of cranial nerve pathology (2–6). Enhancement of

TABLE 1: Summary of findings in 13 patients with enhancement of the cisternal segment of cranial nerve III

Case No.	Age	Sex	Diagnosis	CN III Palsy	CN III Enhancement/ Thickening	Other Symptoms	Associated MR Findings	Proof/Follow-up
1	5	M	Viral meningitis	Yes, unilateral pupil spared	Bilateral/no	None	Meningeal enhancement	Lymphocytosis, negative cultures
2	67	F	Leukemia	No	Bilateral/no	Headaches	Enhancing V,VII/VIII, meningeal enhancement	CSF cystology +
3	74	F	Lymphoma	Yes, unilateral pupil spared	Unilateral/no	Right hemiparesis	Enhancing left basal ganglia mass	Biopsy of brain mass
4	34	M	HIV infection, lymphoma	Yes, unilateral pupil spared	Bilateral/no	Back pain	Low-intensity bone marrow on lumbar spine MR	Bone biopsy
5	39	M	Neurofibromatosis	No	Unilateral/yes	Bilateral sensorineural hearing loss	Multiple other cranial nerve neurofibromas (CN V, VIII, IX, X, XI)	Excised acoustic neuroma
6	49	M	Inflammatory polyneuropathy HIV infection	No	Bilateral/no	Diffuse weakness	Enhancement of right CN V and VII	CSF lymphocytosis, with elevated protein and negative cultures and cytology. Enhancement resolved post-AZT
7	7	F	Ophthalmoplegic migraine	Yes, unilateral pupil involved	Unilateral/no	Headache	None	Clinical: similar episode 3 years ago spontaneous resolution of enhancement and symptoms
8	24	F	Tolosa-Hunt	Yes, unilateral pupil involved	Unilateral/yes	Headache	Enhancement of the posterior cavernous sinus	Clinical: symptoms and enhancement resolved on steroids
9	35	M	Neurofibromatosis	No	Unilateral/yes	None	Multiple other schwannomas	Clinical diagnosis
10	40	F	Lymphoma	Yes, unilateral pupil involved	Bilateral/yes	None	None	Enhancement resolved spontaneously. New left-sided palsy. Positive peripheral biopsy
11	64	M	? inflammatory diabetic	Yes, unilateral pupil spared	Bilateral/no	None	None	1 year later persistent symptoms resolved enhancement
12	56	M	Coccidiodiomycosis	Yes, unilateral pupil involved	Unilateral/no	Hemiparesis	Right basal ganglia infarct	CSF analysis
13	40	M	Lymphoma	Yes, unilateral pupil spared	Unilateral/no	Left cavernous sinus syndrome proptosis	Left cavernous sinus mass infiltrating orbital apex	Biopsy

Note.—CN III = third cranial nerve; CSF = cerebrospinal fluid.

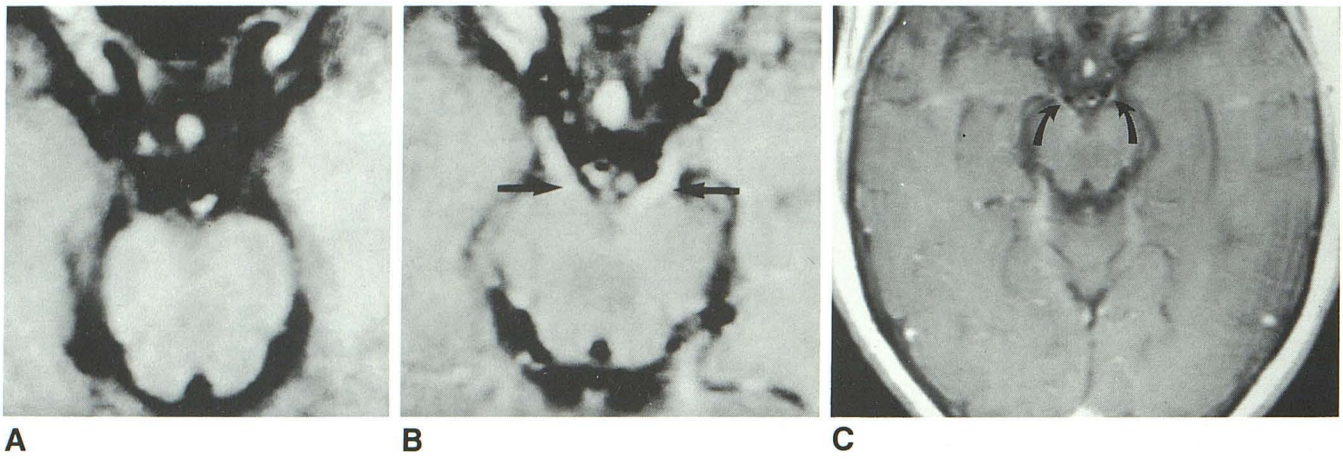


Fig. 1. Patient 2; 67-year-old woman with chronic lymphocytic leukemia; no third cranial nerve palsy. Axial pre- (*A*) and post-gadolinium (*B*) T1-weighted (600/20) images demonstrate bilateral enhancement of the third nerves (*arrows*). Axial T1-weighted image (*C*) postintrathecal chemotherapy shows decreased but persistent enhancement (*curved arrows*).

Fig. 2. Case 4; 34-year-old man—HIV positive and lymphoma proven by bone marrow biopsy. Right third nerve palsy. Pre- (*A*) and postcontrast (*B*) contrast T1-weighted (600/20) axial images demonstrate enhancement of the third cranial nerves.

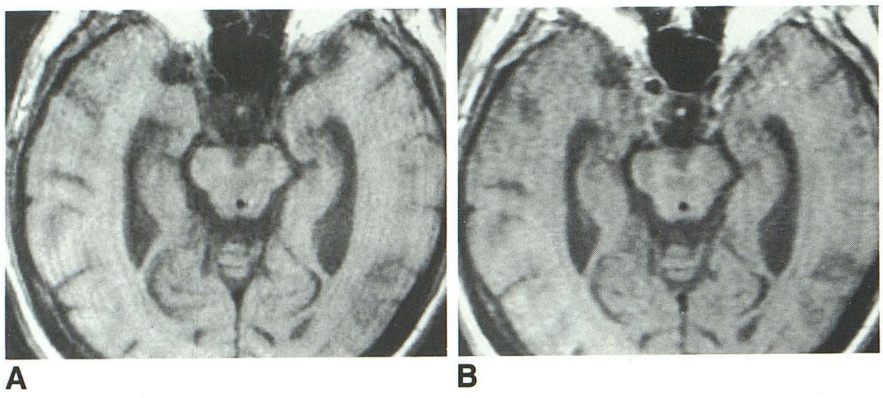
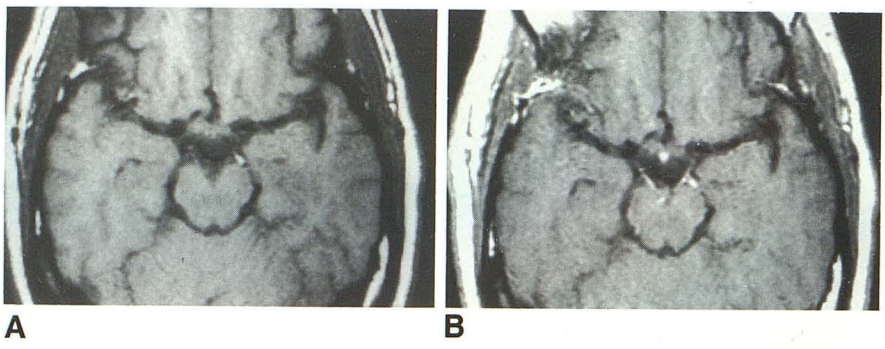


Fig. 3. Case 6; 49-year-old HIV positive man with an inflammatory polyneuropathy resulting in diffuse arm and leg weakness and a right facial weakness. No diplopia. Pre- (*A*) and postcontrast (*B*) T1-weighted (600/20) axial MR demonstrates enhancement of the third cranial nerves. Enhancement of the right seventh cranial nerve in the temporal bone was also demonstrated on the lower sections.



the second, fifth, and seventh cranial nerves on contrast-enhanced MR has been reported in patients with neuropathies of these nerves secondary to a variety of inflammatory or neoplastic processes (2–6). The enhancement has been associated with viral neuropathies, in particular herpes (4), Bell's palsy (5, 6), syphilis (7), as well as demyelinating optic neuritis and post-radiation optic neuritis (2, 3). However, we, as well as other authors (6), have occasionally encountered enhancement of the seventh cranial nerve in asymptomatic patients with no apparent underlying pathology.

Since we have not observed enhancement of the third cranial nerve in any of our controls and since all 13 patients had an underlying disease

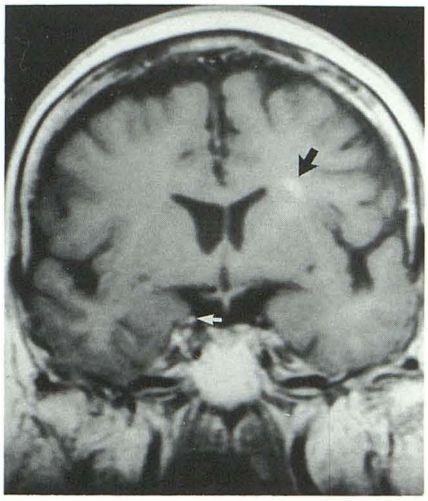


Fig. 4. Patient 3; 74-year-old woman with left parenchymal lymphoma (*black arrow*) and a right third cranial nerve palsy. Coronal post-gadolinium T1-weighted (600/20) images. Notice enhancement of the right oculomotor nerve (*white arrow*).

and/or a third cranial nerve palsy, our study suggests that enhancement of the third cranial nerve is always abnormal, indicating an underlying inflammatory or neoplastic pathology.

Enhancement of the oculomotor nerve, however, is not always associated with a clinically apparent third nerve palsy. Furthermore, while resolution of the enhancement was associated with resolution of the third cranial nerve palsy in some patients, in two patients the symptoms persisted and/or recurred while the enhancement resolved (patients 10 and 11). Four of the nine patients with third cranial nerve palsy had involvement of the pupil, whereas the other five had normal pupillary function. The parasympathetic fibers travel on the superficial aspect of the oculomotor nerve in the cisternal portion and are most susceptible to extrinsic compression by extraneural masses such as posterior communicating artery aneurysms. Conversely, in 68% to 86% of cases due to infarction of the microvasculature located centrally in the nerve, the pupillary fibers are spared (8). These clinical findings are not absolute. In 3% to 5% of aneurysms, the pupil may be spared (8).

In our series, only one patient was diabetic (patient 11). The persistence of the palsy 1 year after the initial presentation is unusual since most such patients recover after several months (8). Persistence of the palsy beyond this time suggests a different cause for the third cranial nerve palsy. The low incidence of diabetic microvascular infarcts in our series may be explained by the fact that most diabetic patients with pupil-sparing third cranial nerve palsies do not undergo MR. However, we have studied three diabetic patients with acute pupil-sparing third cranial nerve palsies using a similar MR technique and did not observe any enhancement of the third cranial nerve. Thus, it is unlikely the palsy in patient 11 is diabetic in origin. Additional studies are necessary to determine the incidence of oculomotor nerve enhancement in diabetic microvascular infarct third cranial nerve palsies.

Third cranial nerve palsy in patients with AIDS has been previously reported (9, 10). The palsy may be due to an intraaxial mass lesion such as parenchymal toxoplasmosis or lymphoma affecting the midbrain in the region of the third cranial nerve nucleus, or as demonstrated by patient 6, direct involvement of the third cranial nerve by HIV as suggested by the resolution of the enhancement on the post-AZT study. In patients with CNS lymphoma, the enhancement of the third cranial nerve probably reflects coating and/or infiltration of the nerve by lymphomatous cells.

The two patients with neurofibromatosis and presumed third cranial nerve schwannomas were both asymptomatic with respect to oculomotor nerve function. They both had many other schwannomas diagnosed by gadolinium-enhanced MR. The diagnosis is often clinically obvious and enhancement of the third cranial nerve may be only one of many findings. In such cases, the depiction of a third cranial nerve-enhancing lesion would be without a great deal of clinical significance. However, isolated oculomotor nerve schwannomas may be symptomatic, presenting with third cranial nerve palsy (11).

In the past, a number of nondiabetic and nonmyasthenic patients with third cranial nerve palsies and negative arteriograms and CT scans were categorized as idiopathic, and an inflammatory or "vascular" process was suspected. These conditions are nevertheless important since the pupil is often involved, suggesting a compressive lesion (8). Our study suggests that such inflammatory processes may now be imaged by gadolinium-enhanced MR.

Ophthalmoplegic migraine is a rare cause of third cranial nerve palsy (12). Miller, in a review of 3 million admissions at Johns Hopkins Hospital, found 30 cases of isolated third cranial nerve palsy in children, two of which were diagnosed as ophthalmoplegic migraines (13). It is a diagnosis of exclusion, traditionally requiring a typical

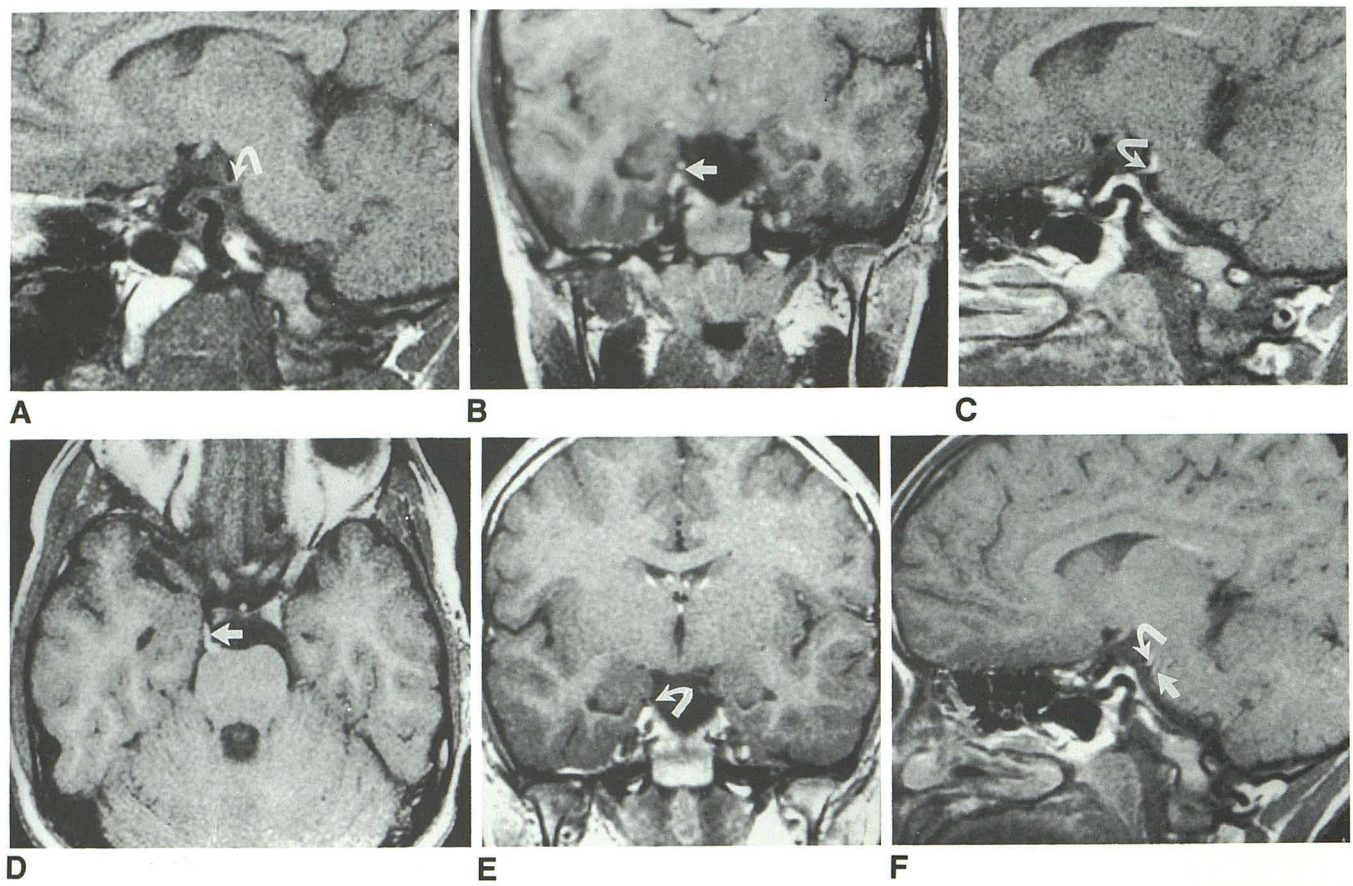


Fig. 5. Patient 7; 7-year-old girl with severe headache, nausea, vomiting, and a right third cranial nerve palsy. Clinical diagnosis: ophthalmoplegic migraine. Precontrast parasaggital (*A*) T1-weighted image through the right third nerve (*curved arrow*). Postcontrast coronal (*B*), parasagittal (*C*), and axial (*D*) T1-weighted (600/20) images demonstrate enhancement of the third cranial nerve (*arrows*) and of the pia in the interpeduncular cistern. Follow-up coronal (*E*) 3 weeks later demonstrates resolution of the enhancement of the anterior aspect of the third cranial nerve (*curved arrow*); minimal residual enhancement of the posterior aspect of the nerve and pia in the interpeduncular cistern is still present (*curved arrow*) on the parasagittal image (*F*). A phase encoding artifact is seen just below the third cranial nerve (*straight arrow*). The patient's symptoms resolved spontaneously.

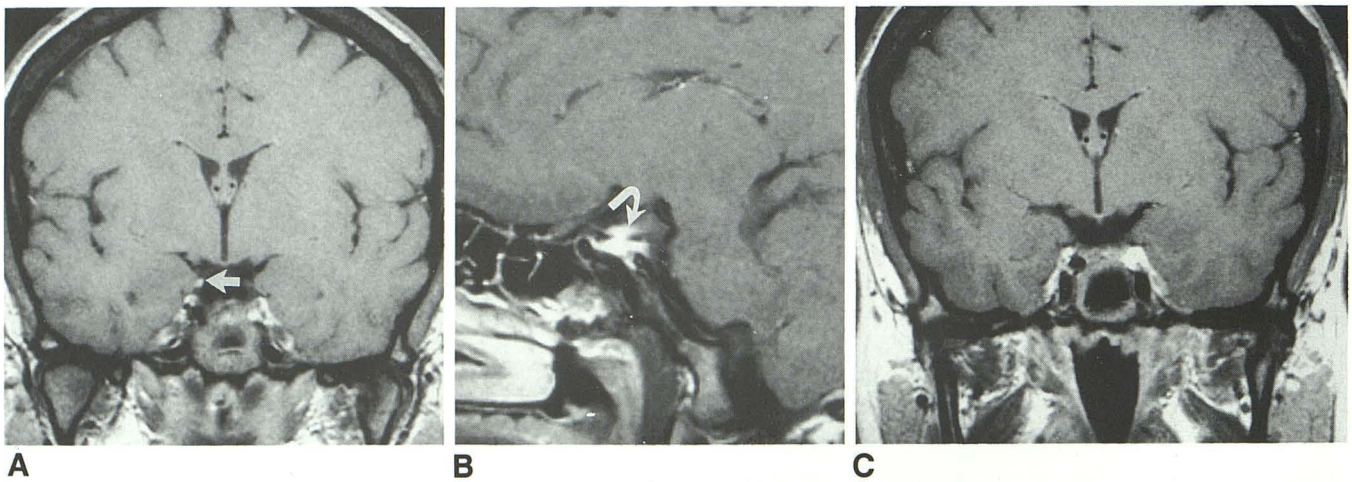


Fig. 6. Case 8; 24-year-old woman with unilateral headache and third cranial nerve palsy. Coronal (*A*) and parasagittal (*B*) contrast-enhanced T1-weighted (600/20) images demonstrate enhancement of the right third cranial nerve (*arrows*). Follow-up study, coronal (*C*) 1 month later after steroid treatment demonstrates resolution of the third cranial nerve enhancement. The symptoms resolved. Clinical diagnosis: Tolosa-Hunt syndrome.

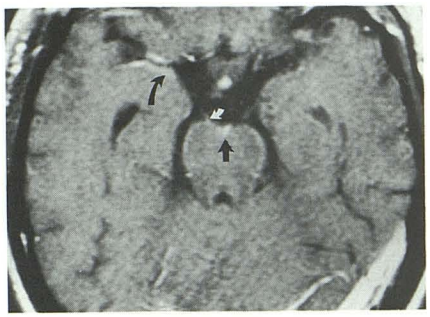


Fig. 7. Case 12; 56-year-old man with right-sided third nerve palsy involving the pupil. Arteriography was negative. Axial T1-weighted image (600/20) demonstrates enhancement of the cisternal segment of the right third cranial nerve (*white curved arrow*). Notice the enhancement along the pia of the right temporal lobe (*black curved arrow*), and interpeduncular cistern (*straight black arrow*). Cerebrospinal fluid studies confirm the diagnosis of coccidioidomycosis.

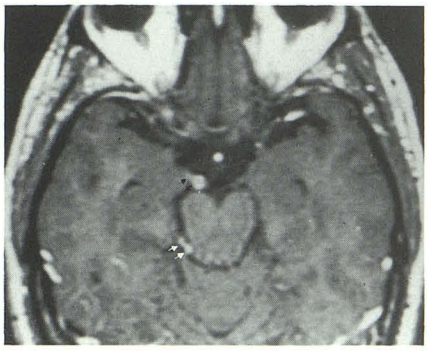


Fig. 8. Case 9; 35-year-old man with neurofibromatosis and oculomotor nerve palsy. Axial short TR/TE (600/20) MR shows enhancing right third nerve mass (*black arrows*) arising from interpeduncular cistern in patient with neurofibromatosis. Also note enhancing right fourth nerve mass (*white arrows*) coursing around midbrain from dorsal aspect of perimesencephalic cistern.

history of migraine and a normal arteriogram to exclude an aneurysm. Atypical forms without the accompanying headaches have been described as a variant of ophthalmoplegic migraine (14). The etiology of this condition remains obscure. Certain authors suggested that narrowing of the carotid artery in the cavernous sinus produced edema that may compress the third cranial nerve (15). Other authors believe it is due to delayed ischemic neuropathy (16). The location of the enhancement in patient 6 at the origin of the third cranial nerve in the interpeduncular cistern is clearly not consistent with this hypothesis. Additional studies will be necessary to elucidate the nature of this clinical syndrome.

Tolosa-Hunt syndrome is a clinical condition characterized by retro-orbital pain and variable degrees of ophthalmoplegia with or without decrease in vision (17). Although the characteristic clinical presentation in these patients is a cavernous sinus or an orbital apex syndrome, an isolated third cranial nerve palsy (as in patient 8) can occasionally be seen. Until the advent of high-resolution of MR, Tolosa-Hunt syndrome was also often a diagnosis of exclusion after arteriography confirmed the absence of an aneurysm. Recently, the MR appearance of Tolosa-Hunt syndrome has been reported (17). Enhancement and abnormal soft tissue in the ipsilateral cavernous sinus is usually noted. This appearance is nonspecific since lymphoma, sarcoidosis, and other neoplastic conditions can have a similar radiographic appearance. In these patients, the cavernous sinus usually returns to normal radiographically either spontaneously or after steroid treatment. Enhancement of the cisternal segment of the oculomotor nerve in Tolosa-Hunt syndrome has not been previously reported and the significance of this finding is unclear. One can speculate that there may be some overlap clinically between Tolosa Hunt syndrome and ophthalmoplegic migraine, especially the "variant" form.

Viral meningitis, as in patient 1, may also produce a third cranial nerve palsy. By demonstrating meningeal enhancement, MR suggested the correct diagnosis, differentiating this condition from other inflammatory processes affecting the oculomotor nerve primarily.

From our observations, it is apparent that the role of MR in the evaluation of patients with third nerve palsies is rapidly evolving. As with any other cranial neuropathy, when imaging these patients it is important to evaluate the entire course of the nerve from its nucleus through the cisternal portion, cavernous sinus, and to the orbital apex. MR is uniquely suited for this task (18). The most serious potential cause for a third nerve palsy is an aneurysm originating from the origin of the posterior communicating artery. At the present time, the sensitivity of MR angiography for the detection of these aneurysms is not known. Because of the potentially devastating consequence of missing such an aneurysm, we believe that in a patient with a third cranial nerve palsy involving the pupil, arteriography is the initial modality of choice to exclude an aneurysm. This approach may change if MR angiography proves itself a reliable diagnostic tool for aneurysm detection. If an aneurysm is excluded in a patient with pupillary involving oculomotor palsy, MR with contrast should be the next imaging

study. Likewise, in patients with pupillary-sparing third cranial nerve palsy who are neither diabetic nor myasthenic, MR with contrast may be extremely useful in detecting neoplastic or inflammatory processes in the oculomotor nerve and direct further investigation.

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