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MR Imaging of Primary Trochlear Nerve Neoplasms

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We present the clinical, anatomic, and MR imaging findings in six patients with seven primary trochlear nerve neoplasms, as well as the MR and clinical criteria that serve to establish the diagnosis of these rare cranial nerve neoplasms. Three patients had a history of neurofibromatosis and five patients had clinical evidence of a trochlear nerve palsy. Six of seven neoplasms produced localized, fusiform enlargement of the proximal cisternal segments of the trochlear nerves. The lesions that were visible on noncontrast MR scans (T1-, T2-, and proton density-weighted) had signal intensities that were virtually identical to normal brain parenchyma. All lesions showed intense, homogeneous enhancement on contrast-enhanced scans. Contrast-enhanced imaging was necessary for the detection of five of seven lesions and greatly increased the value of the MR study in all six patients.

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Primary neoplasms arising from pure motor cranial nerves such as the trochlear nerve are rare. To our knowledge just nine primary trochlear nerve neoplasms have been reported in the literature [1–7], only one of which was imaged with MR [1]. Over the last 6 years, since we began to use MR as the primary imaging method for evaluating patients with cranial nerve palsies, we have noticed a striking increase in the number of primary trochlear nerve neoplasms over those encountered during the CT era. We believe this is due to a much greater sensitivity of MR in detecting these typically small lesions. We present our MR experience with the detection and characterization of these rare neoplasms.

Subjects and Methods

During the interval between July 1984 and September 1990, 250 patients studied with MR at our institution were found to have lesions that involved 281 cranial nerves. Fifteen patients in this series had neoplasms that involved the trochlear nerve, six patients had a total of seven primary trochlear nerve neoplasms, and nine patients had neoplasms that secondarily involved that nerve. Five of seven primary lesions and all nine secondary lesions were symptomatic. MR images were obtained with a 1.5-T MR system (Signa, GE Medical Systems, Milwaukee) using T1-weighted (600/20/1–2) (TR/TE/excitations), proton density-weighted (2300–3500/20–30/1), and T2-weighted (2300–3500/90–100/1) noncontrast scans as well as T1-weighted (600/20/1–2) scans after administration of gadopentetate dimeglumine (0.1 mmol/kg). Clinical and imaging findings in seven cases of trochlear nerve neoplasm are presented in Tables 1 and 2, respectively.

Representative Case Reports

Case 1

A 61-year-old man with neurofibromatosis type 2 (NF-2) had a 2-year history of progressive vertical diplopia. His medical history was significant for subtotal resection of a melanotic

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TABLE 1: Clinical Findings in Seven Cases of Trochlear Nerve Neoplasm

Case No.	Age	Sex	Trochlear Nerve Palsy	Duration of Palsy	MR Follow-up (months)	Surgical Proof	Neurofibromatosis	Other Cranial Nerve Lesions	Other Neoplasms
1	61	M							
Left			Yes	28 mo	8	No	NF-2	Bilateral III, V, VIII	Spinal/peripheral neurofibromas, spinal melanotic schwannoma
Right			No	–	8	No	NF-2	Right VII, Left XII	
2	36	M	No	–	27	No	NF-2	Bilateral VIII	Cranial/spinal meningiomas, brainstem glioma, peripheral/spinal neurofibromas
3	28	M	Yes	2 yr	–	Yes	NF-1	No	Peripheral nerve neurofibromas
4	58	M	Yes	5 yr	12	No	No	No	None
5	64	F	Yes	29 mo	12	No	No	No	None
6	48	M	Yes	9 yr	13	No	No	No	None

Note.—NF-1 = neurofibromatosis type 1, NF-2 = neurofibromatosis type 2.

TABLE 2: Imaging Findings in Seven Cases of Trochlear Nerve Neoplasm

Case No.	Lesion Size (mm^3)	CT Detection	Detection on MR Sequences				Signal Intensity Relative to Brain		
			T1	Proton Density	T2	Enhanced T1	T1	Proton Density	T2
1 Left	20	–	–	R	–	+	NS	Isointense	NS
Right	16	–	–	R	–	+	NS	Isointense	NS
2	16	–	R	R	–	+	Isointense	Isointense	NS
3	8000	+	+	+	+	+	Isointense	Isointense	Isointense
4	32	NP	–	–	–	+	NS	NS	NS
5	16	NP	–	–	–	+	NS	NS	NS
6	192	R	+	–	–	+	Isointense	NS	NS

Note.—MR enhancement pattern was intensely homogeneous in all cases; + = seen; – = not seen; R = seen only retrospectively, after a contrast-enhanced MR scan was performed; NS = not seen; NP = not performed.

schwannoma of a lumbar nerve root 4 years earlier. Neurologic evaluation disclosed a left trochlear nerve palsy, bilateral trigeminal nerve hypesthesias, and mild left facial paresis. Bilateral papilledema was present due to increased intracranial pressure related to elevated CSF protein. Enhanced T1-weighted MR images revealed small bilateral linear enhancing masses in the quadrigeminal plate cistern just posteroinferior to the inferior colliculus of the midbrain (Fig. 1). The elongated lesions were intrinsic to the cisternal segments of the trochlear nerves, just distal to their origin from the midbrain (Fig. 2). Also noted were similar bilateral lesions of the third, fifth, and eighth cranial nerves, as well as a lesion of the right facial nerve. All the cranial nerve neoplasms have remained stable in size over the past year. Surgery is not contemplated at this time because of the minimal symptoms, stable neurologic course, and stable lesion size. In view of the history of NF-2, the cranial nerve lesions most likely represent multiple schwannomas.

Case 3

A 28-year-old man with neurofibromatosis type 1 (NF-1) had a 3-month history of severe intermittent headaches and a 2-year history of vertical diplopia. A plexiform neurofibroma had been resected from his face 2 years earlier. Neurologic examination revealed right trochlear, right trigeminal (third division), and right facial nerve palsies. MR showed a 25 × 20 × 16 mm well-demarcated mass posterior to the inferior colliculus of the midbrain (Fig. 3). The lesion had a signal intensity that was similar to brain parenchyma on all noncontrast pulse sequences. The lesion was located at the point of emergence of the right trochlear nerve from the brainstem. The lesion caused mild hydrocephalus due to aqueductal compression. Contrast-enhanced T1-weighted MR scans revealed intense homogeneous enhancement of the mass (Fig. 3). At right suboccipital craniotomy, the lesion was found to be arising from the right trochlear nerve. The entire mass and involved portions of the trochlear nerve were removed. Histology revealed a schwannoma with mixed focal glial elements. Postoperatively, the patient has done well with the exception of a persistent right trochlear nerve palsy.

Case 6

This 48-year-old man was initially evaluated at another hospital 3 and 9 years earlier, respectively, for two different episodes of vertical diplopia (worse on right gaze), left retroorbital pain, and a left trochlear nerve palsy. Brain CT, MR imaging, and cerebral arteriography were reported to be normal on those occasions. Symptoms improved until 1 year later when he was first evaluated at our hospital for progression of diplopia and retroorbital pain. Neuroophthalmologic examination showed normal visual fields, visual acuity, and pupillary function. A left trochlear nerve palsy was again noted. An MR study revealed a well-defined, elongated mass in the left ambient cistern (Fig. 4). A coronal T1-weighted MR image confirmed that the 12 × 4 × 4 mm mass was intrinsic to the cisternal segment of the left trochlear nerve. The signal intensity of the lesion was similar to normal brain tissue on the T1-weighted scans but was not visible on other precontrast pulse sequences. Intense homogeneous enhancement was noted on postcontrast studies. A review of the old CT scan, obtained 3 years

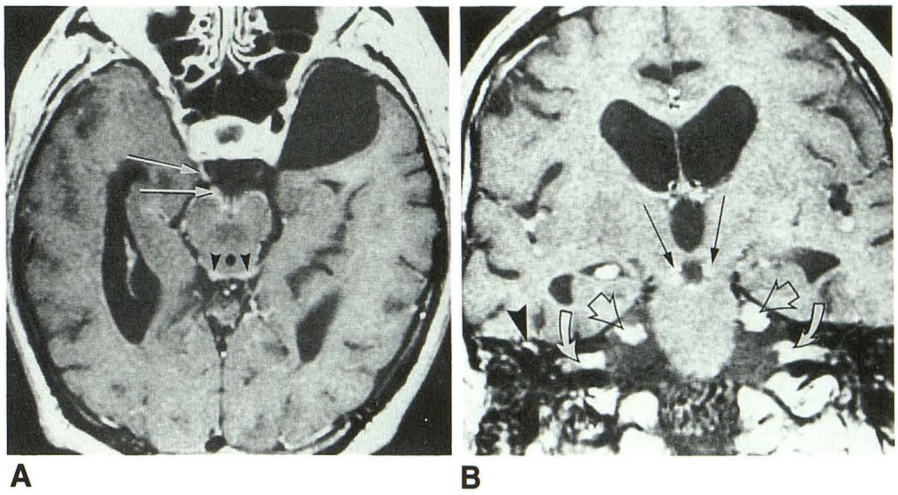

Fig. 1.—Case 1: 61-year-old man with NF-2; the lesions most likely represent multiple cranial nerve schwannomas.

***A* and *B*, Contrast-enhanced axial (*A*) and coronal (*B*) T1-weighted (600/20) MR scans reveal small bilateral homogeneously enhancing lesions of the proximal cisternal segments of both trochlear nerves (*arrowheads* in *A*). The lesions are located at the site where the trochlear nerves emerge from the midbrain, just posterior and slightly caudal to the inferior colliculus. Also noted are similar lesions of the oculomotor nerve (*long thin arrows*), trigeminal nerve (*open arrows* in *B*), vestibuloacoustic nerve (*curved arrows* in *B*), and right facial nerve (*arrowhead* in *B*).**

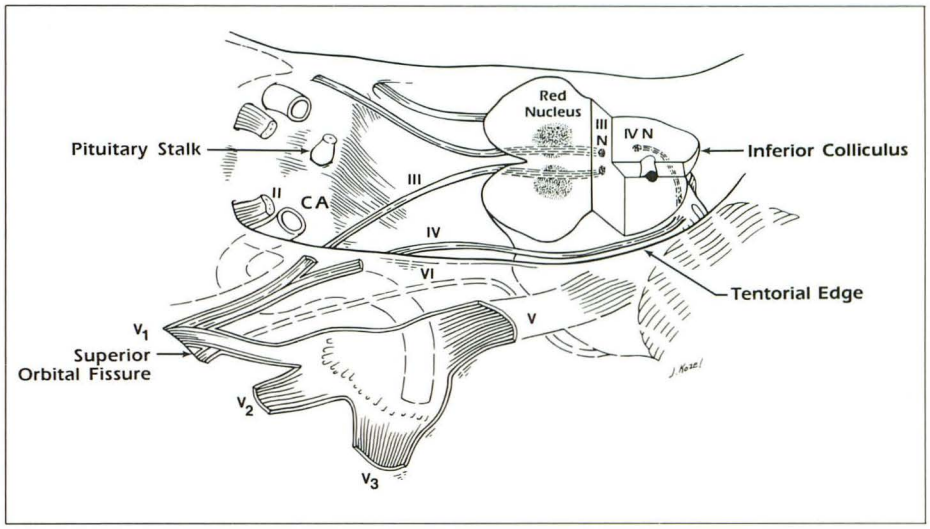

Fig. 2.—Diagram illustrating normal course of fourth cranial nerve. The nerve originates from the trochlear nerve nucleus, just ventrolateral to the cerebral aqueduct and caudad to the oculomotor nerve nucleus. The nerve fibers sweep dorsally around the aqueduct, decussate within the anterior medullary velum, and exit the dorsal surface of the midbrain just below the inferior colliculus, contralateral to the nucleus of their origin. The cisternal segment of the nerve then wraps around the brainstem, successively traversing the quadrigeminal, ambient, crural, and pontomesencephalic cisterns. The cisternal portion of the trochlear nerve is closely related to the tentorium cerebri, and usually is located a few millimeters inferolateral to its free edge. The nerve enters the cavernous sinus by piercing the dura along the lateral clivus, just below the petroclinoidal ligament and between the oculomotor and abducens nerves. The cavernous segment of the nerve runs in the lateral wall of the cavernous sinus, just inferior to the position of the oculomotor nerve. The trochlear nerve enters the orbit through the superior orbital fissure, external to the musculotendinous ring that serves as an attachment for the extraocular muscles.

earlier at another hospital, showed that the trochlear nerve lesion was visible, retrospectively, and had not changed in size. The patient's symptoms improved substantially after the placement of a prism in his eyeglasses. Surgery is not contemplated at the present time because of the minimal neurologic compromise and the significant risks of surgery.

Discussion

The trochlear nerve nucleus is located within the midbrain at the level of the inferior colliculus, just ventrolateral to the cerebral aqueduct (Fig. 2) [8]. The trochlear nerve nucleus gives rise to a group of axons that form a fascicle, which then courses posteroinferiorly around the aqueduct to decussate within the anterior medullary velum just caudad to the inferior colliculus. The trochlear nerve then emerges from the dorsal surface of the lower midbrain contralateral to the nucleus of origin and continues forward around the cerebral peduncle in the ambient cistern slightly inferior to the free edge of the tentorium (Figs. 1 and 2). The trochlear nerve leaves the posterior fossa by piercing the dura along the lateral aspect of the clivus just below the petroclinoidal ligament [8]. The parasellar segment of the trochlear nerve is located within the dura that forms the lateral wall of the cavernous sinus, positioned slightly below the oculomotor nerve [8–10]. It then enters the orbit through the superior orbital fissure, external to the tendinous ring that serves as an attachment site for the extraocular muscles [8].

Neuroophthalmologic evaluation is of great importance in clinical assessment of patients thought to have a trochlear nerve palsy. A true palsy must be differentiated from mechanical restriction of trochlear tendon movement through the trochlear sling (Brown syndrome), primary extraocular muscle abnormalities (e.g., Graves disease), orbital pseudotumor,

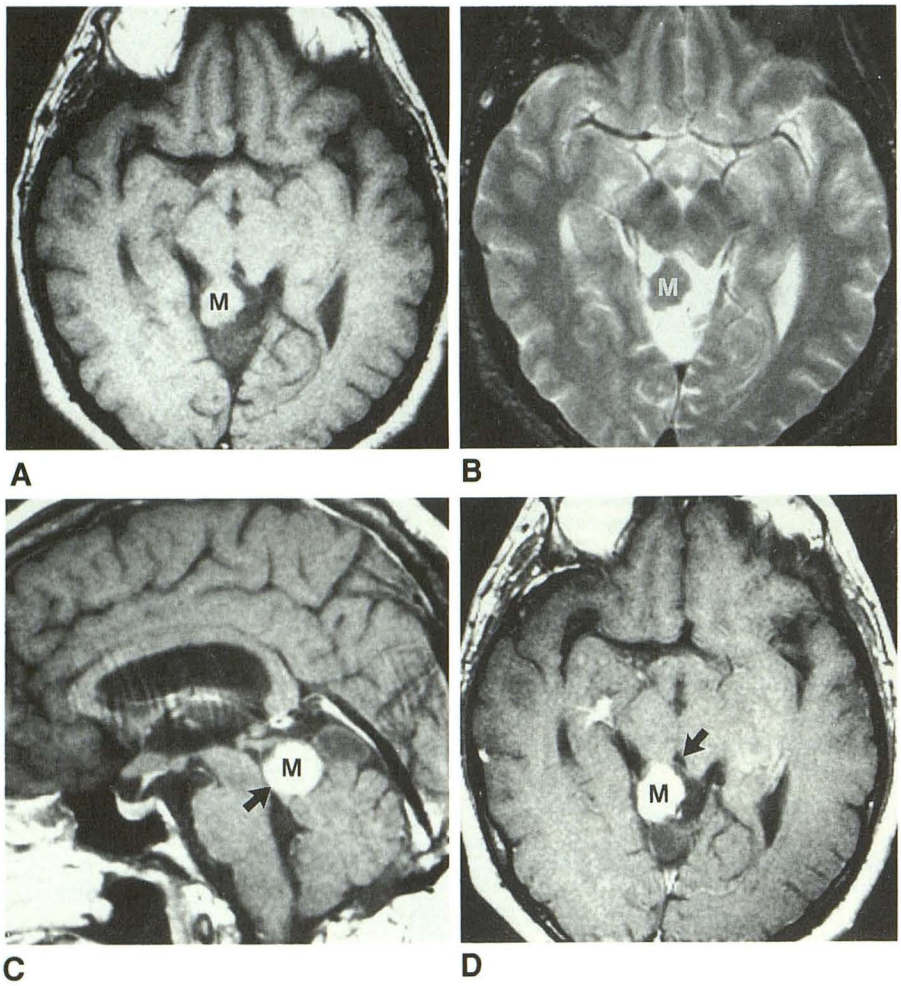

Fig. 3.—Case 3: 28-year-old man with NF-1.
***A–D*, Precontrast axial T1- (600/20) (*A*) and T2- (2300/90) (*B*) weighted MR scans, and sagittal (*C*) and axial (*D*) contrast-enhanced T1-weighted (600/20) MR scans. A large soft-tissue mass (M) arises at the site where the right trochlear nerve emerges from the inferior colliculus of the midbrain. Signal intensity of the lesion on noncontrast images is similar to that of normal brain parenchyma. Intense homogeneous enhancement of the lesion is seen on postcontrast scans. The mass lesion causes mild hydrocephalus due to midbrain distortion and aqueductal compression (*arrows*). At surgery, the lesion was noted to originate from the right trochlear nerve. Histopathologic analysis revealed a schwannoma with focal glial elements.**

and neuromuscular disease (e.g., myasthenia gravis) [11]. Imaging studies are often required to make this distinction with confidence. The type of visual field deficit and presence of associated palsies of cranial nerves III, V, and VI are often helpful in clinically localizing the site of disease. Because of considerable overlap among the clinical syndromes, however, anatomic delineation of the exact cause of trochlear nerve palsy is usually required.

MR offers considerable advantages over CT for evaluation of suspected cranial nerve abnormalities [12–14]. The superiority of MR results from a greater ability to detect abnormal tissue constituents within cranial nerves, often before there has been significant alteration of nerve morphology, and from improved anatomic depiction of lesions [12–14]. MR also provides a greater potential for increasing the conspicuity of small cranial nerve neoplasms through the use of MR contrast agents (e.g., gadopentetate dimeglumine). For these reasons, MR has become the diagnostic examination of choice at our institution for the evaluation of patients with suspected cranial nerve abnormalities.

The cisternal segment of the normal trochlear nerve is usually seen best with thin-section (3–4 mm), high-spatial-resolution (18–20-cm field of view, 256 × 256 matrix), coronal T1-weighted scans (Fig. 4). This portion of the trochlear nerve can often be traced from its emergence posterior and slightly caudad to the inferior colliculus of the midbrain to the point where it enters the cavernous sinus between the oculomotor and trigeminal nerves. The trochlear nerve is typically found just inferior and medial to the free edge of the tentorium and slightly superior to the position of the preganglionic segment of the trigeminal nerve. Occasionally, the cisternal segment can also be seen on axial images. That segment of the trochlear nerve in the lateral wall of the cavernous sinus can occasionally be identified on coronal T1-weighted images just caudad to the position of the oculomotor nerve and superior to the ophthalmic division of the trigeminal nerve [9, 10]. The orbital and superior orbital fissure segments of the trochlear nerve are not usually visible.

Review of the literature would suggest that primary neoplasms of the trochlear nerve are uncommon. To our knowledge only one case of a primary trochlear nerve neoplasm has been reported since the development of MR imaging [1]. Overall, in the medical literature, only nine cases have been reported [1–7]. Our experience, however, indicates that these lesions may be more common than previously believed. During the last 6 years, when MR was used as the primary

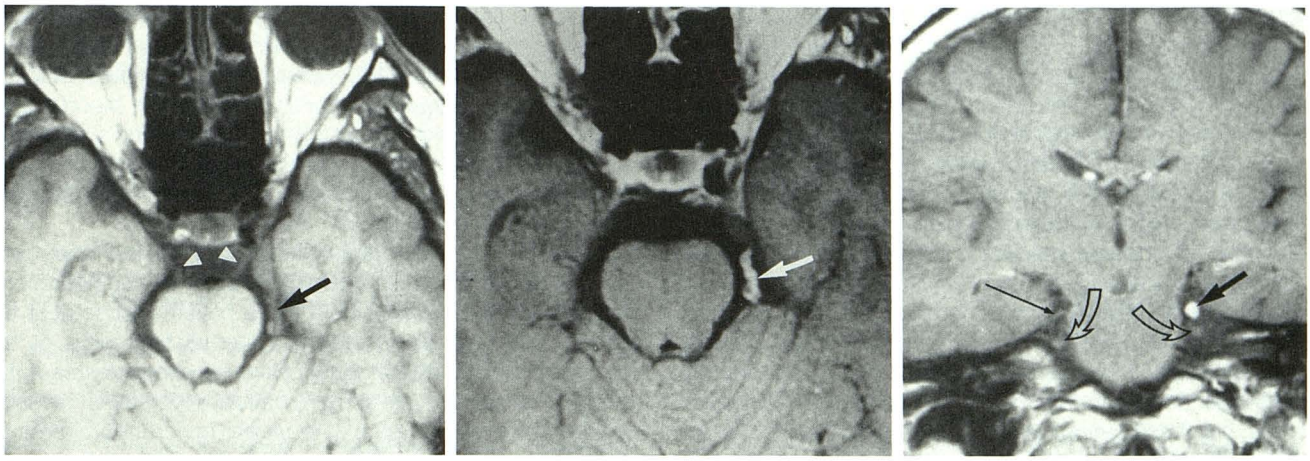

Fig. 4.—Case 6: 48-year-old man with trochlear nerve mass that has remained stable in size for 3 years, indicating that it most likely represents a benign primary neoplasm, the vast majority of which are schwannomas.

***A–C*, Unenhanced axial (*A*) and contrast-enhanced axial (*B*) and coronal (*C*) T1-weighted (600/20) MR scans. A small elongated mass (*straight arrows*) is intrinsic to cisternal segment of left trochlear nerve. The lesion is well-defined, symmetrically expands the trochlear nerve, and shows intense homogeneous enhancement. The normal contralateral trochlear nerve (*long thin arrow* in *C*), oculomotor nerve (*arrowheads* in *A*), and trigeminal nerve (*curved arrows* in *C*) are visualized.**

diagnostic study for evaluation of cranial nerve palsies at our institution, we have identified seven primary trochlear nerve neoplasms. This compares with only one lesion detected at our institution during the previous decade, when CT was used to evaluate this group of patients. We believe that the increase in the number of these lesions in the last 6 years is not merely a chance phenomenon, since four of five lesions were not initially visualized with high-quality CT scans. We believe, rather, that it is a function of the superior ability of MR to detect these typically small neoplasms.

The two main types of benign nerve sheath tumors (schwannomas and neurofibromas) are both thought to be of Schwann cell origin [15]. A comprehensive discussion of the pathologic differences between these two lesions is beyond the scope of this article, although they can easily be distinguished on morphologic and histologic grounds [15]. The schwannoma is the most common type of benign cranial nerve neoplasm [15]. It occurs in two patterns: isolated lesions and multiple schwannomas, as seen in the syndrome of neurofibromatosis [15]. Solitary schwannomas show a strong tendency to involve sensory nerves, and occur predominantly in middle-aged to elderly women [1–7, 15]. Solitary schwannomas most commonly involve the eighth cranial nerve, while other sensory cranial nerves such as the fifth, ninth, and 10th are less commonly involved [15]. Pure motor nerves, such as the trochlear nerve, are thought to be infrequently involved by solitary schwannomas [1–7, 15]. Orbital schwannomas are encountered occasionally, presumably arising from peripheral branches of the nerves to the extraocular muscles [16, 17].

The neurofibroma is the other commonly encountered nerve sheath neoplasm. This lesion usually involves peripheral and spinal nerves. Solitary cranial nerve neurofibromas are thought to rarely (if ever) occur [15]. In fact, some authors assert that there are probably no true examples of isolated cranial nerve neurofibromas in the absence of neurofibromatosis [15]. The vast majority of solitary cranial nerve neurofibromas that have previously been reported in the absence of neurofibromatosis are probably truly schwannomas [15].

Multiple benign nerve sheath tumors are a well-recognized feature of both NF-1 (peripheral neurofibromatosis, or von Recklinghausen disease) and NF-2 (central neurofibromatosis) [15, 18, 19]. With NF-1, involvement of cranial nerves is much less common than involvement of spinal and peripheral nerves; however, multiple benign neoplasms (usually schwannomas) of the cranial nerves are extremely common with NF-2 [15, 19]. These schwannomas may involve any of the cranial nerves, although, quite similar to the situation with solitary schwannomas, sensory nerves are most characteristically affected [15]. Bilateral acoustic nerve schwannomas alone, in fact, are sufficient evidence of NF-2 [15, 18]. Schwannomas of other sensory cranial nerves, such as the fifth, ninth, and 10th, occur less frequently, while pure motor nerves, such as the trochlear, are rarely involved in NF-2 [15].

It is generally believed that cranial nerve schwannomas are most likely to arise at the point where the axonal covering switches from glial elements to Schwann cells [15]. The trochlear nerve junction is usually not more than 0.6 mm away from its emergence from the midbrain [4, 15]. Five of the seven cases in our series arose from this expected location (Figs. 1 and 3). In some cases, however, schwannomas may arise at a distance of several centimeters from the brainstem, anywhere along the course of the nerve (Fig. 4) [1–7, 15]. Imaging of a patient with a trochlear nerve palsy, therefore, must always include the brainstem, ambient cistern, cavernous sinus, and orbit.

Other benign and malignant nerve sheath tumors must also be included in the differential diagnosis of trochlear nerve masses. Benign neoplasms—such as granular cell myoblastomas, neurothekeomas, localized hypertrophic neurofibrosis

(perineurinoma), neurotrophic melanomas, hamartomas, cavernous hemangiomas, and paraganglioma—may potentially affect cranial nerves, although these lesions are exceedingly rare and more commonly affect peripheral nerves [15]. Although the overwhelming majority of cranial nerve sheath tumors are benign, malignant lesions can occur [15]. De novo malignant cranial nerve schwannomas, however, are extremely rare. Benign schwannomas also have a very low propensity to undergo malignant degeneration. When this does occur, it is almost always in patients who have neurofibromatosis [15]. Malignant neurofibromas are more common, but they rarely (if ever) occur outside the setting of neurofibromatosis [15]. A number of patients (2–29%) with neurofibromatosis do eventually develop malignant nerve sheath lesions, but these usually affect peripheral and spinal nerves instead of cranial nerves [15]. Nevertheless, in patients with neurofibromatosis and documented cranial nerve sheath masses, it is important to be aware of the potential for malignant degeneration [15].

A histologic diagnosis was available in only one of our patients (case 3: schwannoma with mixed glial elements); however, the trochlear nerve abnormalities seen in the other cases almost certainly represent benign nerve sheath neoplasms (most likely schwannomas), since these patients have the clinical stigmata of NF-2. The precise pathogenesis is not available for three other lesions in this series although the clinical and radiologic observations strongly suggest that isolated schwannomas are the most likely diagnoses. Other potential causes for intrinsic trochlear nerve abnormalities, however, must also be considered in the differential diagnosis. A wide variety of nonneoplastic processes may produce intrinsic lesions of the cranial nerves for the extraocular muscles [11, 15, 17, 20–22]. The most common entities to be considered in the differential diagnosis include demyelinating diseases, granulomatous meningitis (tuberculous, fungal, sarcoid), carcinomatous meningitis, multifocal viral polyneuritis (e.g., Ramsay-Hunt syndrome) [22], recurrent benign multiple cranial neuropathies [20, 21], and Tolosa-Hunt syndrome [20, 21].

In our cases, demyelinating disease was excluded with reasonable confidence on the basis of clinical data and lack of other lesions on the MR scans. Similarly, granulomatous and neoplastic diseases were excluded on the basis of history and physical examination, clinical evaluation, CSF studies, laboratory analyses, and imaging studies. Postviral polyneuritis was considered unlikely in view of the patients' histories, prolonged symptoms, stable lesion size over time, and the MR scan appearance. Trochlear nerve involvement with the Ramsay-Hunt syndrome is quite uncommon, although cranial nerves other than the seventh and eighth (that is, cranial nerves V, VI, IX, and X) may be involved [22]. Tolosa-Hunt syndrome is a nonspecific inflammatory process of the cavernous sinus that typically affects multiple cranial nerves and presents with a steroid-responsive painful ophthalmoplegia [11, 20, 21]. It was thought to be an unlikely cause of the trochlear nerve lesions in our patients in view of one or more of the following: sparing of other intracavernous cranial nerves, location of the lesions in the cisternal segment of the nerve, lack of painful ophthalmoplegia, and long history of symptoms. Recurrent benign multiple cranial neuropathies is a poorly characterized syndrome that may affect all three ocular motor nerves [11]. Some have postulated that this represents a nonspecific arteritis involving the blood supply to the cavernous portions of the third, fourth, and sixth cranial nerves [11]. This seems to be an unlikely explanation for the isolated lesions of the cisternal segments of the trochlear nerves seen in our patients.

The seven trochlear nerve neoplasms reported here reveal several similar features. Three of six patients had neurofibromatosis and all four lesions in these patients arose near the inferior colliculus and proximal cisternal segments of the trochlear nerves. All primary trochlear nerve neoplasms in this series that were seen on precontrast MR scans revealed signal intensities that were quite similar to normal brain tissue. The precontrast appearance was identical to the more common acoustic schwannomas [12–14]. All seven fourth nerve neoplasms in this report showed intense homogeneous enhancement (Figs. 1, 3, 4), also quite similar to that seen with acoustic schwannomas.

Since primary trochlear nerve neoplasms are benign lesions and surgery is not usually advantageous unless the lesions are large, it is important to make a precise imaging diagnosis [1, 4]. Surgical removal will almost always be associated with persistence or worsening of the trochlear nerve palsy [1, 4]. Surgery, therefore, is usually to be discouraged unless other cranial nerves are in jeopardy, the lesion produces significant mass effect (hydrocephalus, brainstem compression), or there is a suspicion of underlying malignancy. Because of continuous relentless enlargement and a slight possibility of malignant degeneration, however, we believe that it is important to periodically monitor these lesions with follow-up MR scans. This is especially true in patients with the clinical stigmata of NF-1 or NF-2.

On the basis of our experience, we believe the following clinical and MR features are necessary to confidently diagnose a benign primary trochlear nerve neoplasm:

1. Location. The lesion must be intrinsic to the trochlear nerve at some point along its course. This is usually at the point where the trochlear nerve emerges from the brainstem or in the proximal cisternal segment of the fourth nerve.
2. Epicenter. The lesion epicenter must be completely within the trochlear nerve.
3. Margination. These benign lesions must be clearly demarcated from all adjacent structures. Poorly demarcated lesions that extend into adjacent structures raise the possibility of malignant schwannoma, neurofibrosarcoma, or extrinsic malignancies invading the trochlear nerve.
4. Noncontrast MR characteristics. The MR signal intensity of benign trochlear nerve sheath tumors is identical or quite similar to that of normal brain parenchyma.
5. MR contrast enhancement. The lesions typically show intense homogeneous enhancement, identical to that seen with the more common acoustic schwannomas.
6. Multiple cranial nerve lesions or the clinical stigmata of NF-1 or NF-2.
7. Lengthy history of trochlear nerve palsy. The trochlear nerve palsy is usually slowly progressive over a number

of years, since schwannomas and neurofibromas are benign, slow-growing lesions.
8. Stable lesion size over several years.

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