OBJECTIVE. In this pictorial essay, we review the MR appearance of cranial nerve enhancement in a variety of entities including neoplastic, infectious, and idiopathic diseases.

CONCLUSION. MRI with contrast enhancement is a valuable tool for detecting and characterizing disease of the cranial nerves. Abnormal cranial nerve enhancement on MRI may sometimes be the first or only indication of an underlying disease process.

MRI is invaluable in characterizing disease of the cranial nerves. Gadolinium administration increases the ability of MRI to detect such abnormalities. We begin this pictorial essay with a description of the histologic anatomy of the cranial nerves and patterns of normal cranial nerve enhancement. After briefly discussing the pathophysiology, we review the MR appearance of abnormal cranial nerve enhancement in various diseases ranging from common neoplastic and infectious conditions to rare conditions such as ophthalmoplegic migraine and idiopathic pachymeningitis. In some cases, abnormal cranial nerve enhancement on MRI may be the only clue to the underlying disease.

Normal Cranial Nerve Enhancement

There are instances of normal cranial nerve enhancement. The geniculate, tympanic, and mastoid segments of the facial nerve possess peri- and epineural venous plexuses that may cause moderate enhancement by an increased vascular pool of contrast material [1]. The intracanalicular–labyrinthine segment does not normally enhance. The trigeminal ganglion and the proximal portions of its divisions are seen as discrete nonenhancing structures surrounded by an enhancing perineural vascular plexus. Enhancement of the trigeminal ganglion or its maxillary or mandibular divisions is infrequently seen as evidenced by their avascular appearance in cadaveric specimens [2]. When such enhancement is seen on MRI, it may be related to suboptimal imaging parameters, avid enhancement of the perivascularex, or a combination of both.

Neoplasm

Neoplastic meningitis refers to the disseminated seeding of the leptomeninges by malignant cells. This includes carcinomatous meningitis in patients with solid tumors and lymphomatous and leukemic meningitis when involvement is related to these underlying diseases. The most common cancers to involve the leptomeninges are breast (5%), lung (9–25%), and melanoma (23%) [3] (Fig. 1). MRI findings include pial enhancement and nodularity, smooth or nodular cranial nerve enhancement, hydrocephalus, and coexisting brain or bone metastases [4]. Primary diffuse leptomeningeal gliomatosis...
is a rare condition whereby a glioma arises from heterotopic cell nests in the leptomeninges. Leptomeningeal dissemination is an uncommon complication of gliomas and other primary intraaxial malignancies. The presence of a single unexplained enhancing cranial nerve in a patient with cancer raises the possibility of leptomeningeal dissemination.
Fig. 2—56-year-old woman after resection of adenoid cystic carcinoma of right hard palate.
A, Axial bone window CT image shows widening of right pterygopalatine fossa (arrow).
B and C, Contrast-enhanced axial T1-weighted MR images reveal infiltrating mass in right pterygopalatine fossa (short arrow, B) and cavernous sinus (short arrow, C).
Note abnormal signal intensity in right masticator space (long arrows, B) and right medial temporal lobe (long arrows, C).

Fig. 3—43-year-old man with acute lymphoblastic leukemia.
A, Axial FLAIR image reveals leukemic infiltrate of left pons and brachium pontis (arrow).
(Fig. 3 continues on next page)
Perineural tumor extension, a form of metastatic disease, involves the spread of primary mucosal or cutaneous tumors to noncontiguous regions along nerve sheaths. Perineural tumor spread has been shown in perineural or endoneural tissue planes along a path of least resistance. Retrograde spread is significantly more common than antegrade spread. A series by Parker and Harnsberger [5] found perineural spread occurs most commonly with squamous cell carcinoma and adenoid cystic carcinoma, with the facial nerve and second and third divisions of the trigeminal nerve most frequently involved (Fig. 2). Other neoplastic and aggressive infectious processes, such as acute lymphoblastic leukemia, non-Hodgkin’s lymphoma, malignant schwannoma, aspergillosis, mucormycosis, and actinomycosis, also show perineural extension (Fig. 3). MRI findings of perineural involvement include smooth thickening and enhancement of the nerve, concentric expansion of the skull base foramina with
obliteration of normal fatty contents, enlargement of the cavernous sinus, and neuropathic muscular atrophy [6].

**Infection**

Infectious meningitis results from viral, bacterial, fungal, or parasitic infection. Lep-tomeningitis is the most common form of intracranial tuberculosis, particularly in the pediatric population. Cranial nerve involvement
is seen in 17–70% of patients and occurs in the setting of diffuse leptomeningeal tuberculosis. Impairment has been attributed to ischemia of the nerve or entrapment of the nerve in basal exudates [7] (Fig. 4).

_Cryptococcus neoformans_ is the most common fungus to involve the CNS. Cryptococcal meningitis is one of the typical pathologic manifestations and can result in optic neuropathy in both immunocompetent and immunocompromised patients (Fig. 5).

Optic neuropathy is a rare complication of cryptococcal meningitis and usually occurs in non-AIDS patients. Necrosis of the optic nerves and infiltration of the meninges around the optic tracts, nerves, and chiasm by cryptococcal organisms have been observed [8].

Rhinocerebral mucormycosis is a potentially devastating fungal infection in diabetic and immunocompromised patients. Sinonasal disease often progresses to the orbit and cavernous sinus and may be complicated by vascular and perineural invasion and local thrombotic infarction [9] (Fig. 6).

Cranial neuroschistosomiasis occurs less commonly than the spinal variety and may arise with any of the clinical forms of this parasitic infection. Eggs within the CNS induce a cell-mediated periovular granulomatous reaction that leads to signs and symptoms of increased intracranial pressure and focal neurologic signs [10]. Although
meningeal spread of infection involving cauda equina nerve roots has been reported, rare instances of cranial nerve involvement may also be seen as in our case (Fig. 7).

Cranial neuritis in Lyme disease may involve any of cranial nerves III through VII, with the facial nerve most frequently affected and often associated with cochleovestibular nerve abnormalities. The affected segments appear thickened and enhance. Viral infections related to herpes simplex virus type 1, cytomegalovirus, and Varicella zoster organisms also manifest with cranial nerve involvement and show abnormal enhancement on MRI.

**Postinfectious and Demyelinating Disorders**

Bell’s palsy is the most common cause of unilateral peripheral facial neuropathy. In addition to normal enhancement of the facial nerve segments discussed earlier, there is pathologic enhancement of the intracanalicular–labyrinthine portion (Fig. 8). Martin-Duverneuil et al. [11] suggest three criteria for pathologic enhancement of the facial nerve: enhancement outside the facial canal, extension of enhancement to cranial nerve VIII, and intense enhancement of the labyrinthine and mastoid segments. In Ramsay Hunt syndrome, abnormal facial nerve enhancement is accompanied by enhancement of the vestibular and cochlear nerves as a result of extension of inflammation from cranial nerve VII to the intracanalicular portions of these cranial nerve VIII divisions.

Ophthalmoplegic migraine is a rare condition characterized by headache and oculomotor nerve palsy lasting days to weeks. MRI findings include reversible enhancement of the cisternal segment of the oculomotor nerve and focal thickening at the exit of the nerve in the interpeduncular cistern (Fig. 9). Involvement of cranial nerves IV, V1, and VI also occurs. Multiple cranial nerve involvement is also present in a group...
of inflammatory demyelinating polyneuropathies that include Guillain-Barré and variants, such as Miller Fisher syndrome and polynueritis cranialis.

**Granulomatosis**

Intracranial neurosarcoïdosis has a predilection for the basal leptomeninges, and involvement of every cranial nerve has been described. MRI shows a spectrum of CNS abnormalities including diffuse or nodular thickening and abnormal enhancement of the leptomeninges in the basal cisterns and hypothalamic regions [12] (Fig. 10). Perineural spread has also been reported in sarcoidosis [13]. Clinical involvement and imaging cranial nerve involvement frequently do not coincide, and clinical resolution may not imply imaging resolution [14].

Idiopathic hypertrophic cranial pachymeningitis is a rare disease characterized by inflammation and fibrosis of the dura mater. It remains a diagnosis of exclusion but may be the presenting manifestation of granulomatous diseases such as sarcoidosis, Wegener’s granulomatosis, or tuberculosis. MRI shows focal or diffuse thickening and enhancement of the dura that encase cranial nerves causing recurrent cranial neuropathies. The oculomotor, abducens, and facial nerves are more frequently involved [15].

Tolosa-Hunt syndrome consists of painful ophthalmoplegia related to a granulomatous inflammatory process in the cavernous sinus. MRI findings are nonspecific and include enhancement and abnormal soft tissue in the ipsilateral cavernous sinus and orbital apex [16] (Fig. 11).

**Postradiation Neuritis**

Radiation-induced cranial nerve injury is an uncommon, usually delayed, complication of radiation therapy or radiosurgery. Cranial nerve deficits may be permanent or resolve spontaneously. Loss of the nerve–blood barrier due to demyelination and ischemia, coagulation necrosis, or peripheral fibrosis results

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**Fig. 9—57-year-old man with ophthalmoplegic migraine.**

A–C, Unenhanced axial (A) and enhanced axial (B) and coronal (C) T1-weighted images reveal smooth enlargement and homogeneous enhancement of cisternal segment of left oculomotor nerve (arrows).
in cranial nerve enhancement. Radiation-induced optic neuropathy occurs months to years after exposure of the anterior visual pathways to ionizing radiation. MRI shows smooth enlargement and enhancement of the optic nerve and chiasm (Fig. 12).

**Primary Nerve Tumors**

Vestibular schwannomas are the most common cranial nerve schwannomas, followed by...
trigeminal and facial schwannomas and then glossopharyngeal, vagus, and spinal accessory nerve schwannomas (Fig. 13). Neurofibromatosis 2 is characterized by bilateral vestibular schwannomas. Schwannomas of the other cranial nerves occur more frequently in neurofibromatosis 2. Enhancing hemangiomas, meningiomas, or metastases may mimic the appearance of early schwannomas.

**References**

5. Parker GD, Hamsberger HR. Clinical–radiologic issues in perineural tumor spread of malignant diseases of the extracranial head and neck. *Radio-
MRI of Cranial Nerve Enhancement


APPENDIX 1: Classification of Cranial Neuropathies

**Neoplastic:** Carcinoma, lymphoma, leukemia, glioma, myeloma

**Infection:** Tuberculosis, syphilis, leprosy, mycoplasma, Lyme disease, viral infections, fungal infections, parasitic infections

**Postinfectious and demyelinating:** Bell’s palsy, Ramsay Hunt syndrome, ophthalmople-gic migraine, Miller Fisher syndrome, polyneuritides, multiple sclerosis

**Granulomatosis:** Sarcoidosis, idiopathic granulomatosis, vasculitis, inflammatory granulomatosis

**Angiopathic:** Wegener’s granulomatosis, Churg-Strauss syndrome, Behçet’s syndrome, diabetes

**Idiopathic:** Idiopathic pachymeningitis, Tolosa-Hunt syndrome

**Physical or chemical:** Radiation, trauma, surgery, toxins, drugs

**Hereditary:** Dejerine-Sottas disease, Krabbe’s disease

**Primary nerve tumors:** Schwannoma, neurofibromatosis