Facial Nerve Imaging

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Imaging Study

It is important to realize that the facial nerves have a longer course through bone than any other nerve in the body; therefore, their evaluation requires imaging in many different planes and perhaps using different modalities such as CT and MRI.

For the temporal region, high-resolution temporal bone CT still plays a major role in determining the cause and extent of lesions of the seventh nerve; however, MR imaging has made significant improvement in the evaluation of certain specific disease. For example, evaluation of inflammatory lesions has become a major role of MR imaging. There typically is uniform, linear enhancement of the facial nerve, which maybe normal in size of slightly enlarged in patients with Bell' palsy and Ramsay Hunt syndrome. Thus, it is important to remember that high-resolution temporal bone CT does a superlative job of margin the bony canal of the facial nerve, but it does not primarily image the nerve itself; MR is the test of choice to image not the bony course of the facial nerve, but the nerve. Important information may be gained by both tests, however, and it is a common occurrence to use both examinations to image cranial nerve VII.

Temporal bone CT scanning is ideally performed with very thin slices(0.5－1 mm) and with an edge-enhancing algorithm to provide maximum bone detail.

MR protocols to adequately depict the facial nerve vary from institution to institution. Our current protocol consists of 5-mm axial T2 weighted images and FLAIR, 3-mm axial T1 weighted images before contrast administration, 3-mm axial and coronal T1 images with fat-suppression. In addition, strongly T2-weighted images such as 3D balanced TFE images to evaluate the cranial nerve in the cistern and the internal auditory canal.

Many cases of perineural tumor spread(PNS) are probably missed because the imaging technique is inadequate. A preferred MRI protocol for patients with suspected PNS is the application of thin(2－3 mm) section pre-contrast T1 and T2 and contrast-enhanced T1 image with and without fat suppression in the axial and coronal planes and with a field of view of 16－18cm. Pre-contrast T1 weighted imaging is important for looking at involvement of the normal fat planes. Fat-suppressed post-contrast T1 weighted imaging is important for looking at subtle enhancing lesions, which would otherwise be missed on routine post-contrast T1 weighted imaging.

Pathology

1. Neoplasms of the facial nerve

Neoplasia of the facial nerve may be primary or secondary. There are 2 rare primary neoplasm of the facial nerve, the primary facial nerve schwannoma, and a hemangioma of the facial nerve. Facial nerve schwannoma is the most common primary tumor of the facial nerve. It can occur anywhere along the facial nerve but most commonly occurs at 2 locations - in the cisternal segment and the geniculate ganglion. When small, there is generally uniform enhancement, but when large, there can be heterogeneous enhancement. Enlargement along the course of the seventh nerve highly suggests a facial nerve schwannoma. Primary facial nerve hemangiomas are unusual neoplasms occurring in the perigeniculate region, and show characteristic fine, speckled calcifications on CT scans, and are of heterogeneous signal intensity on T1 and T2 images

Secondary tumor involvement of the facial nerve is typically caused by involvement of the facial nerve by a neoplasm of the head and neck including the parotid gland. Although this pattern of tumor involvement may be seen in any primary neoplasm of the parotid, it most commonly accompanies adenoid cystic carcinoma. Although parotid disease is the most common cause, any disease process along the peripheral course of the facial nerve can result in facial neuropathy. Tumors of the parotid gland most commonly affect the peripheral segment. Most parotid gland tumors (80%) are benign and rarely cause facial palsy. A parotid tumor in the presence of facial paralysis is most likely malignant.

2. Perineural Tumor Spread(PNS)

Perineural spread（PNS） is a mechanism whereby tumor, or other pathologic conditions, spread along the tissues of the neural sheath. PNS has significant implications for treatment planning and also represents a major negative prognostic indicator. Imaging is critical in the diagnosis of PNS, particularly because it may asymptomatic. PNA may occur with any head and neck malignancy, including salivary gland tumors and mucosal and skin carcinomas. Although adenoid cystic carcinomas of the minor or major salivary glands are notorious for producing PNS, squamous cell carcinomas（SCC） of mucosal or cutaneous origin are also frequently associated with PNS

Typical imaging findings in PNS on a CT scan include foraminal enlargement, foraminal destruction, and abnormal enhancement within the foramen or canal. Loss of fat around the nerves or within the foramina should also be viewed as an indication of PNS. MRI findings suggestive of PNS are obliteration of fat planes around the nerve or within the foramen, nerve enhancement, and nerve enlargement. PNS along the facial nerve will show enhancement with or without thickening of the nerve and usually extends from the parotid segment to the intracanalicular or cisternal segment. It is important to remember that some portions of the facial nerve, namely the anterior genu and tympanic and mastoid segments, normally enhance on post-contrast MRI because of the presence of a normal arteriovenous plexus. Therefore, interpreting pathologic neural enhancement in the facial nerve can be tricky.

3. Inflammation of the facial nerve

Bell's palsy is an acute idiopathic peripheral facial palsy that may be caused by viral inflammation, polyneuropathy, or immunologic or ischemic diseases. Bell's palsy accounts for 50% to 80% of all cases of facial nerve palsy. It is usually not imaged unless there are atypical features, such as other clinical findings in addition to facial palsy, symptoms lasting longer than 3 to 6 months, or recurrent paralysis. On MR imaging, there typically is uniform, linear enhancement of facial nerve, which may be normal in size or slightly enlarged. Normally, there is some enhancement of the geniculate ganglion because of an abundant vascular epineurium. There may also be enhancement of the tympanic and mastoid segment of the facial nerve. The distinction between pathologic and normal enhancement is difficult but, generally, pathologic enhancement is usually more intense than normal enhancement.

Ramsay Hunt syndrome is a viral inflammation of the facial nerve that can involve the eighth nerve and membranous labyrinth. Imaging is usually not required because the disease is self-limited in 85% of cases. If imaged, abnormal enhancement of the seventh or eighth nerves or membranous labyrinth typically is observed. Other inflammatory or infectious lesion, such as Lyme disease, syphilis, and sarcoidosis, may also affect the petrous portions of the seventh and eighth nerves. Imaging features are nonspecific.

4. Brain and cerebellopontine angle(CPA) lesions

Upper motor neuron lesions, which involve damage to the cortex or axons above the facial nucleus, involve the contralateral muscles of facial expression but spare the muscles of the forehead. Lesions to consider for upper motor neurons include stroke, hemorrhage, arteriovenous malformations, primary or metastatic neoplasms, and multiple sclerosis（MS）.

Lower motor neuron lesions（damage to the facial nucleus of axons） involve all ipsilateral muscles of facial expression and include the muscles of the forehead. Within the brainstem, gliomas are the most common neoplasm causing cranial neuropathy. MS is the most common inflammatory lesion to affect the seventh nerve nucleus of fasciculus. Vascular lesions include pontine infarcts and vascular malformations, such as cavernous malformations. Within the cisternal segment and internal auditory canal, neoplasms make up most causes of facial neuropathy. The most common CPA mass is the vestibular schwannoma. These tumors most commonly arise from the vestibular division of the eighth nerve and account for 80% to 90% of all CPA tumors. They most commonly produce sensorineuronal hearing loss and, even when large, rarely cause facial nerve palsy. These tumors are well encapsulated and usually well demarcated from adjacent brain and CSF. The differential of CPA masses is extensive but most commonly includes meningioma and epidermoid tumors.

5. Hemifacial Spasms

Mechanical compression of the facial nerve in the CPA cistern by an ecstatic vertebral artery, anteroinferior cerebellar artery, or posteroinferior cerebellar artery is a frequent cause of hemifacial spams. MR angiography and heavily T2 weighted images that allow excellent contrast between flowing blood, CSF, and nerve are useful in evaluating vascular ectasia and aneurysms noninvasively.

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