Magnetic Resonance Neurography: Brachial Plexus

Cynthia T. Chin, MD

Professor of Radiology and Neurosurgery

Department of Neuroradiology

University of California, San Francisco

San Francisco, California

INTRODUCTION

Evaluation of peripheral nerve disorders has relied primarily on accurate clinical history, thorough physical examination and electrodiagnostic testing. This information often allows determination of the location and severity of the underlying peripheral nerve problem. However, while electrodiagnostic studies are sensitive, they do not display the anatomic detail needed for precise localization and treatment planning. Additional limitations of these electrodiagnostic studies include difficulty in performing these studies in younger children and infants; difficulty in accessing deep muscles; and having to wait several weeks after nerve injury before changes may be detected.

The radiological study of peripheral nerve disorders was initially limited to secondary skeletal changes on plain radiographs and computerized tomography (CT) myelography for demonstrating nerve root avulsion in patients with severe proximal brachial plexus injuries.

Routine CT and magnetic resonance imaging (MRI) have been useful to exclude mass lesions in the vicinity of a peripheral nerve. Recent technical improvements in MRI have resulted in improved visualization of both normal and abnormal peripheral nerves

The ability to image peripheral nerves can significantly change the diagnosis and treatment of peripheral nerve disease and can lead to an improved understanding of the pathophysiology of peripheral nerve disease.

TECHNIQUE OF MAGENTIC RESONANCE NEUROGRAPHY

MR neurography (MRN) is tissue-selective imaging directed at identifying and evaluating characteristics of nerve morphology. Visualization of the fascicular structure of the nerves is made possible by exploiting differences in the water content and connective tissue structure of the fascicles and perineurium compared with the surrounding epineurium. [1]

Standard MR imaging techniques allow the detection of nerves. However, there is low conspicuity of these structures from the surrounding tissues. The inherent problems of low signal intensity and low conspicuity are addressed by suppressing signal from the adjacent non-neural structures such as blood vessels and fat in muscle and marrow. The use of T2 fat-saturated and inversion recovery sequences allows for optimal conspicuity of peripheral nerves. Standard T1 and T2-weighted sequences can display the anatomy of the adjacent muscle, bone, vessels and nerves as they are outlined by fat planes.

Because of its small size, abnormal signal on T2-weighted sequences within a nerve is often obscured by signal in adjacent fat. Fat suppression techniques are crucial to identifying normal and abnormal nerve tissue. Frequency-selective fat resonance saturation and short-inversion-time inversion recovery (STIR) are two common techniques used for fat saturation. The STIR method provides a uniform and consistent fat saturation and maintains high T2 contrast and is therefore more reliable compared to

the frequency-selective fat-saturation method. The disadvantages of the STIR sequences include a relatively lower signal-to-noise ratio (SNR) and sensitivity to blood-flow artifacts. Flow saturation bands may be utilized to attenuate the accompanying blood-flow phase shift artifacts. [1] Iterative decomposition of water and fat with echo asymmetry and least squares estimation (IDEAL) is an MRI sequence combining the advantages of both the STIR and frequency-selective fat-saturation techniques, offering both reliable uniform fat saturation and optimal signal to noise. It has been used in musculoskeletal imaging and at our institution is being applied to imaging of the peripheral nerves as well. [2] [3]

Multiplanar reformations allow mapping of the extent of nerve involvement and can be performed with various sequences. 3D volumetric gradient sequences with T2 weighting such as 3D double echo steady state (3D DESS), or 3D water selective sequences (3D WATS) have been used for ligamentous evaluation in musculoskeletal imaging. When applied to the peripheral nervous system, these sequences allow image acquisition in the axial plane with multiplanar reformations which allow anatomic detail of adjacent structures such as muscle and bone to be demonstrated while maintaining sensitivity in detecting abnormal nerve signal. [4] Reformations with the nerves in isolation can also be displayed similar to magnetic resonance angiograms (MRA) utilizing maximum intensity projection sequences and post-processing.

Diffusion weighted imaging:

Magnetic resonance diffusion-weighted imaging (DWI) has been extremely helpful in evaluating lesions in the brain such as acute infarction, abscess and neoplasm. The image contrast it provides is dependent upon detecting random microscopic molecular water motion which can be significantly altered in various disease states. [5]Its use in spine imaging is being developed for evaluation of the spinal cord and potential etiologies of myelopathy. [6] DWI is recently being applied to the peripheral nervous system and can detect and demonstrate normal and abnormal ganglia and proximal roots. [7, 8] [9] Its potential utility is being demonstrated for evaluation of normal and abmormal nerve anatomy related to trauma, neoplasm and radiation injury.

Coils:

Using phased array coils have produced images with improved signal-to-noise and improved resolution, improving the ability to visualize both normal and abnormal peripheral nerves. Compared with MR imaging with standard coils, phased-array coils allows higher resolution, thinner sections, and faster acquisition of images. Phased-array coils integrate data from multiple small coils to produce a single image which has a high signal-to-noise ratio (SNR) and a composite field of view (FOV) similar to that of a larger surface coil. The images produced from phased-array coils is improved compared to larger surface or whole-body coils. [10] However, it is necessary to target the area of clinical interest accurately as only a relatively small FOV can be imaged with high spatial resolution. [1]

MRN has diagnostic utility because it is effective for demonstrating continuity, morphology and response to injury (edema) associated with many kinds of pathology. MRN is useful to evaluate segments of peripheral nerve that are routinely difficult to evaluate electrophysiologically. First, nerve segments involving the brachial or lumbosacral plexuses can be difficult to evaluate with conventional nerve conduction studies. Second, concomitant peripheral polyneuropathy can make interpretation of the nerve conduction studies difficult. Third, abnormalities detectable on clinical motor and sensory exam following acute nerve injury may not be detectable on EMG and nerve conduction studies for 2–5 weeks, but anatomic changes in the nerve may be seen much earlier.

ANATOMY OF THE PERIPHERAL NERVE

Peripheral nerves can be subdivided into three components: 1) conducting axons; 2) insulating Schwann cells; and 3) a surrounding connective tissue matrix which can support axonal regeneration.

Nerve fibers are ensheathed by Schwann cells individually or in groups. A basal lamina layer envelops Schwann cells and is important in supporting axonal regeneration.

The nerve fibers are embedded within a connective tissue compartment called the endoneurium. Nerve fibers and surrounding endoneurium are grouped together into

fascicles and encircled by compact concentrically arranged elongated perineural cells composing the perineurium.

These fascicles are grouped together to form the peripheral nerve and are embedded within a connective tissue compartment called the epineurium which contain fibroblasts, macrophages, mast cells and blood vessels and fat.

IMAGING APPEARANCE

Normal Nerves:

T1-weighted images show the size and location of the nerve. Normal nerve on cross-sectional imaging is oval or round. The size of a particular nerve varies along its course and from person to person. The fascicles within the nerve are visible on high-resolution, cross-sectional images. On T2 sequences, the nerves appear uniform in size, intermediate in signal, generally slightly hyperintense compared to adjacent muscle.

Depiction of the nerve fascicular pattern is based on differences in MR signal of the fascicles within the perineurium as compared to the interfascicular epineurium. The fascicular signal is dominated by endoneurial fluid and axoplasmic water. Interfascicular image signal is dominated by fibrofatty connective tissue which is susceptible to fat suppression. [1]

Abnormal Nerves:

Compressive or infiltrative lesions of the peripheral nerve, may produce three abnormalities; distort the normal nerve course and configuration, disrupt the fascicular pattern described above, or produce homogenous prolongation of the T2 relaxation time (increased T2 signal intensity) in the nerve. In the latter circumstance, increased signal is probably related to increased water content or breakdown of myelin [11]

Diffuse or focal enlargement of a nerve and diffuse or focal hyperintense T2 signal within the nerve are abnormal findings. Altered fascicular pattern is another abnormal finding which may manifest as marked enlargement or abnormal increased signal of individual fascicles within the nerve in a nonuniform pattern.

The pathogenesis of focal signal abnormality is not known but may represent localized edema or increased fluid accumulation within the endoneurial spaces. Possible mechanisms for the abnormal increased T2 signal within the nerve include interruption of normal axoplasmic flow resulting in increased axoplasm proximal and distal to the site of injury; increased endoneurial fluid as a result of venous obstruction allowing accumulation within the fascicles; and demyelination. [12] [13] [14]

The characteristic temporal nature of MR signal abnormalities may be used to evaluate the extent of nerve injury and provide valuable information that correlates with improvement of clinical function. In animal studies involving lab-induced crush injuries to the sciatic nerve, increased T2 signal changes were seen as early as 7 days before

normalizing by 10 days. In severed sciatic nerves that were prevented from regenerating, there were prolonged T2 signal abnormalities that were persistent for up to 42 days. These abnormalities normalized by 60 days, despite an absence of axonal regeneration [15]. There are numerous case reports, following traumatic nerve injury where axonal loss distal to the site of injury correlates with high signal MRI abnormalities that slowly normalize over many months. The evolution of MR signal correlates with regeneration of axons and functional recovery. [16] [13]. It can be useful to clinically follow nerve injury and recovery with MRN, given that MR signal changes in nerves normalize rapidly with regeneration.

MECHANISMS OF INJURY

Both direct mechanical distortion and microvascular compromise contribute to the pathophysiology underlying peripheral nerve dysfunction. Usual mechanisms consist of compression, ischemia, and traction. Nerves often course in neurovascular bundles, vascular trauma can occur concomitantly with neural injury and cause further damage through ischemia. [17] Compared to the central nervous system (CNS), the peripheral nervous system is relatively resistant to ischemia. Compared to the CNS, an important feature of the peripheral nervous system is its ability to recover via both remyelination and axonal regeneration.

Entrapment syndromes represent the most common type of chronic nerve injury. Mass lesions can cause nerve injury through direct compression or actual infiltration. Nerves

may also be affected by systemic diseases such as diabetes mellitus, gout, systemic amyloidosis, hypothyroidism, renal failure, genetic or environmental factors including alcoholism, malnutrition, and toxin exposure.

Susceptibility of peripheral nerves to acute or chronic compressive and tensile forces is a function of internal anatomy and location. The tough perineural layer surrounding each fascicle is composed of elastin and collagen. This layer is normally under a certain amount of resting tension longitudinally, as demonstrated by nerve shortening which occurs after transection. Nerves can stretch 10 to 20% before structural damage occurs. [18] Certain nerves are vulnerable at specific anatomic locations being superficial, fixed in position, or coursing across a joint. The nerve fibers within fascicles have an undulating course, allowing for accommodation of levels of tension produced by normal changes in body position. With increasing tension, the nerve slides then takes up internal slack provided by the undulating course and then with further tension can disrupt axons and myelin sheaths.

Connective tissue damage may occur with stretch injuries which can lead to intraneural and extraneural scar formation. Extraneural scar may distort or compress peripheral nerves along their course or tether a nerve interfering with its normal physiologic gliding. [19]

INDICATIONS FOR IMAGING OF PERIPHERAL NERVES

Indications are evolving in response to improvements in MRI techniques and methods for treatment of peripheral nerve disease. The localization, extent, and severity of nerve injury are some important characteristics which may be demonstrated on MRN. [12] [13] [14]

TRAUMA

Traumatic peripheral nerve injury ranges from disruption of axonal conduction with preservation of anatomic continuity of the nerve connective tissue sheaths (neurapraxic injury) to transection with complete loss of nerve continuity (neurotmetic injury). The majority of serious peripheral nerve injuries do not lead to actual transection of the nerve but rather leave the nerve in continuity. Clinically it may be difficult to distinguish closed nerve injuries that will recover on their own from those that do not and therefore require surgical repair. Serial clinical and electrodiagnostic evaluations over a period of months have traditionally been the mainstay of decision-making in the management of closed traumatic peripheral nerve injuries. MRN may be used to noninvasively localize traumatic peripheral nerve injury and perhaps help to determine whether surgery would be of benefit in a more expeditious manner. MRN complements electrophysiologic data in determining the exact site and type of nerve injury and can show the relationship of the intact nerve to post-traumatic lesions such as neuromas as well as focal or diffuse perineural fibrosis.

Brachial Plexus injuries:

Post-traumatic plexopathy may be the sequela of laceration, compression, stretching, perineural fibrosis or nerve root avulsion. A useful classification for surgical management divides the injured region into the more common supraclavicular level and infraclavicular (including retroclavicular) levels. Traumatic meningocele, or pseudomeningocele, may occur with or without avulsion. Pseudomeningoceles and fusiform retraction of the distal plexus may suggest avulsion injury. However, simple dural tears or partial avulsion injuries can also result in the presence of pseudomeningoceles. [20]

Adult Brachial Plexus Trauma:

In the adult, brachial plexus injuries can be caused by various mechanisms including penetrating injuries, falls, and most commonly motor vehicle trauma (up to 70% involving motorcycles or bicycles). Often the diagnosis can be delayed or ignored as the clinician may wait for signs of clinical recovery.

3D volumetric acquired sequences allow reformations which can demonstrate peripheral nerve continuity or disruption which is important in determining whether surgical treatment is required. 3D volumetric acquired sequences and diffusion sequences can also aid in identifying pre and post-ganglionic injury which is also important for prognosis and appropriateness of surgical repair. Post-ganglionic injuries are located distal to the dorsal root ganglion (DRG) compared to pre-ganglionic lesions which are located proximal to the DRG. In pre-ganglionic injuries, the nerve has been avulsed from the spinal cord and repair would require a neurotization procedure compared to post-

ganglionic injuries which may be surgically repaired or grafted. MR diffusion-weighted imaging is a method for detecting random microscopic motion (diffusion) of water protons and has recently been applied in evaluation of peripheral nerves. The spinal cord, ganglia and proximal peripheral nerves can be demonstrated and may prove to be useful for distinguishing preganglionic from postganglionic avulsion. [7] [8]

Birth-Related Trauma:

An infrequent injury to the brachial plexus can occur in newborn infants manifest by inability to actively move one upper extremity. When the C5 and C6 cervical roots are involved, it is called Erb's palsy and there is an associated flaccid upper arm with a lower arm that is extended and internally rotated.

An important concept in MR neurography is to detect not only abnormal increased signal on T2 sequences within the nerve, but also to look for an abnormal course of the nerves which may be present as a sequela of traction injury. The loss of the normal oblique orientation of the cervical plexus in the absence of a pseudomeningocele is an important indicator of severe traction injury without avulsion and can result in a relatively lax appearance of the cervical roots. [21]

Post-traumatic neuroma formation may interfere with nerve recovery and its presence can be suggested on MR neurography by focal expansion, increased signal and the presence of enhancement with gadolinium. Normal ganglion enhance with gadolinium. Normal nerve-blood barrier results in no appreciable enhancement of the peripheral nerves.

Disruption of this normal barrier allows gadolinium to leak into the epineural tissues with resultant enhancement that can be homogeneous, and in the correct clinical setting, suggest the presence of neuroma formation which is important for surgical planning in patients who no longer demonstrate clinical recovery.

RADICULOPATHY

Radiculopathy is usually caused by compression of the proximal portion of a spinal nerve or roots by disc or osteophyte. MRI has been used extensively in evaluation of cervical and lumbar radiculopathy because of its sensitivity in visualizing degenerative changes. However, its specificity is limited as changes may be found in a large percentage of asymptomatic patients. On MRN, abnormal increased signal on T2 and STIR sequences are observed in symptomatic spinal nerves. This increased signal in symptomatic patients may be seen associated with or without electrodiagnostic abnormalities and may therefore be a highly sensitive technique in increasing the specificity of selecting patients who might benefit most from surgical decompression.

MRN is a sensitive technique for detecting signal change in proximal lumbar and cervical nerve roots compared with standard spine MRI. Among patients with clinical evidence of cervical radiculopathy [22], significant signal change was observed 2-3 cm distal to the site of nerve root compression on STIR sequences in the affected nerve roots. MRN may be particularly helpful in assessing the structural integrity of specific nerve roots in patients with diffuse anatomic change in the spine (i.e., multi-level degenerative disk or

spondylotic changes). Abnormal nerve root signal in this setting draws further diagnostic attention to a specific segment of the spine (i.e., lateral recess syndrome detected by CT myelography).

ENTRAPMENT SYNDROMES

MRN can localize the site of nerve entrapment by demonstrating abnormal signal at the site of entrapment. Common locations include the median nerve within the carpal tunnel, the ulnar nerve within the cubital tunnel, the lower trunk of the brachial plexus within the thoracic outlet, and the sciatic nerve at the greater sciatic foramen. Abnormal nerve morphology and signal abnormalities may be detected and identified along with denervation and atrophy [23]

Brachial Plexus Entrapment:

There are three potential sites of compression along the course of the brachial plexus or the subclavian /axillary artery or vein; the interscalene triangle, the costoclavicular space between the first thoracic rib and the clavicle, and the retropectoralis minor space posterior to the pectoralis minor muscle. Clinical symptoms of entrapment or compression ("thoracic outlet syndromes") may be due to venous or arterial compression, brachial plexus compression, or a combined neurovascular compression.

Classic neurologic thoracic outlet syndrome usually manifests as a chronic lower trunk plexopathy with paresthesias and atrophy affecting arm, forearm and hand. Compression or entrapment is usually the result of a congenital fibrous band extending from an

elongated transverse process or rudimentary cervical rib to the first thoracic rib with resultant stretching, angulation and distortion of the usual course and orientation of the brachial plexus. Routine MR coronal T1 sequences may demonstrate the osseous and fibrous anomalies. [27] MR neurography is useful in demonstrating the distortion of the course of the brachial plexus, and in cases of significant compression, the associated intraneural edema.

Traumatic thoracic outlet syndrome results from clavicular injury, usually a midshaft fracture with resultant injury to the subjacent blood vessels and brachial plexus. There may be compression by displaced fracture fragments, hematoma or pseudoaneurysm formation. Symptoms may frequently present in a relatively delayed fashion, days to even years after the initial injury due to hypertrophic callus and fracture non-union. The cords of the brachial plexus and portions of the subclavian artery and vein may be damaged alone or in various combinations. [28]

TUMOR AND RADIATION PLEXITIS

MRN is useful in localizing the relationship of the nerve fascicles to intraneural and extraneural masses and preoperatively identifies, localizes and assesses surgical resectability. In addition, MRN may be helpful in distinguishing recurrent tumor versus radiation-induced plexitis. Tumor results in focal and irregular enlargement of the nerve with increased signal on T2, short tau inversion recovery (STIR) images, and contrast enhancement. Radiation-induced plexitis results in more uniform enlargement and diffuse abnormal signal of the nerve without contrast enhancement. Our preliminary data

utilizing diffusion weighted imaging of nerves suggest that intraneural tumor tends towards diffusion characteristics which are reduced or similar to normal nerve compared to radiation changes which tend towards increased diffusion relative to normal nerve.

MUSCLE DENERVATION

Increased T2 signal change on STIR sequences has been demonstrated in denervated muscles. The signal change correlates with the degree of denervation found on needle EMG and with the clinical weakness observed on neurologic examination. [18] The signal changes in the denervated muscle can appear as early as 4 days after severe nerve injury, and are accompanied by gadolinium contrast enhancement. [29] Signal change is found in muscles supplied by nerves sustaining axonal injury only, and is not seen with demyelinative lesions. The pathophysiology of increased muscle signal is not completely understood, but is thought to be related to an acute increase in blood flow associated with a loss of sympathetic vasoconstriction, and a shift in protons (water) from the intracellular to the extracellular space. It appears that MRI of muscle can provide valuable information regarding the type of nerve injury (i.e. axonal versus demyelinative) that in turn can help to predict clinical prognosis. Chronic denervation shows decreased volume and fatty atrophy manifest as high T1 signal intensity.

SUMMARY

Treatment of spinal and peripheral nerve lesions relies on localization of the pathology. Current evaluation of such pathology mainly involves the use of conventional MRI and

electromyography. MRN is a novel technique used for direct imaging of spinal and peripheral nerves. Features of intraneural topography can be displayed and morphology and signal intensity characteristics which distinguish normal from abnormal nerve can be demonstrated and is a valuable complement in the evaluation of spinal and peripheral nerve pathologies.

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