ROLE OF MAGNETIC RESONANCE NEUROGRAPHY IN BRACHIAL PLEXUS LESIONS

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Magnetic resonance neurography (MRN) is a relatively new technique that combines magnetic resonance imaging (MRI) with specially designed phased-array surface coils that allows for visualization of the peripheral nerves.1,6,8,10,11 The surface coil technology is combined with excellent fat-suppression highly T2-weighted magnetic resonance (MR) pulse sequences to best visualize the nerves amidst the other soft tissues. Compared to conventional MRI techniques, MRN has faster image acquisition and higher signal-to-noise ratio with resultant decreased motion artifact and higher resolution capable of showing fascicular organization of nerves to detect both extraneural and intraneural lesions. It has been used increasingly in assessing lesions affecting peripheral nerves, plexus, and spinal nerve roots.4,5,10,12,13 We report six cases in which MRN played an important role in confirming the presence of lesions in the brachial plexus.

CASE REPORTS

Six patients, four with brachial plexitis (two postsurgical and two without precipitating factors) and two with multifocal demyelinating disorders affecting the brachial plexus, were studied with MRN. All patients underwent clinical evaluation, appropriate laboratory testing, nerve conduction studies (NCS), and electromyography (EMG). The study was approved by our Institutional Review Board.

The imaging protocol utilized phased-array surface coils as well as body coil, and the MR protocol included axial and coronal T1-weighted scans, axial and coronal inversion recovery fast spin echo T2-weighted scans, and postcontrast axial and coronal images. Coronal scans were oblique parallel to the brachial plexus. Axial scans were performed from the C5 to T2 levels.

Case 1. A 46-year-old woman developed pain over the left arm after abdominal surgery for pseudomyxoma peritonei. Three days later, she was referred to our neuro...
muscular clinic because the weakness had not improved. Examination showed weakness limited to the left flexor pollicis longus (grade 0 on the Medical Research Council (MRC) scale), pronator quadratus (4), and flexor digitorum profundus to digits 2 and 3 (4). Sensory examination and deep tendon reflexes were normal. Median and ulnar sensory nerve action potential (SNAP) amplitudes and latency values were normal. Needle EMG examination showed abundant fibrillation potentials and positive sharp waves, and a few high-amplitude and long-duration voluntary motor units only in the muscles supplied by the left anterior interosseous nerve (AIN), i.e., flexor pollicis longus, flexor digitorum profundus of the second and third digit, and pronator quadratus. MRN of the left forearm was normal. MRN of the left brachial plexus showed edema, with increased T2 signal of the plexus at the level of trunks and divisions, consistent with left brachial plexitis (Fig. 1). Evaluation for other etiologies, including for vasculitis, was negative. In particular, erythrocyte sedimentation rate, antinuclear antibody, and antineutrophil cytoplasmic antibody were normal. There were no risk factors for brachial plexus injury related to the surgery, such as use of shoulder braces, head-down position, or malposition of arms. Ten months after the onset, the patient reported significant improvement of her weakness.

**Case 2.** A 17-year-old woman developed acute right upper-extremity weakness 1 day after spine fusion surgery for scoliosis. During the surgery, right median somatosensory evoked potentials were intact. Immediately after the surgery, she was able to move her right arm. She denied sensory symptoms although she was on narcotics for postoperative pain. Examination showed profound weakness (MRC grade 0–1) of the right deltoid, biceps, triceps, wrist extensors, and wrist flexors, and moderately severe weakness (2–3) of the interossei and thumb abductor. Patchy sensory loss in the right upper arm was present. Deep tendon reflexes were absent in the right arm. NCS of the right upper extremity, 2 days after the onset of symptoms, showed severe motor conduction block across the Erb’s point to axillary segments of the right median and ulnar nerves and absent F-waves in these two nerves. Right median and ulnar SNAPs were normal. Needle EMG showed scattered fibrillation potentials and positive sharp waves, lack of recruitment of any motor units in the proximal muscles, and only a few motor units in the distal muscles of the right arm. Follow-up study 6 weeks after the onset of symptoms showed absent SNAPs and severely reduced compound muscle action potential (CMAP) amplitudes of the right median and ulnar nerves. Profuse abnormal spontaneous activity (fibrillation potentials and positive waves) was now present in the right deltoid, biceps, triceps, extensor digitorum communis, pronator teres, abductor pollicis brevis, and first dorsal interosseous muscles. No motor units were recruited in the right deltoid and biceps, and motor unit recruitment was severely reduced in the other noted muscles. MRN, 2 days after the onset of right arm weakness, showed mild edema and thickening along the course of the fibers of the right brachial plexus, consistent with right brachial plexitis. At 1-year follow-up, marked improvement to near-normal strength had occurred in the right arm.

**Case 3.** A 23-year-old woman developed acute right shoulder pain lasting for 3 days followed by weakness that evolved over a period of 4 weeks. There was no antecedent infection, trauma, or surgery. Four months after the onset of symptoms, when there was no improvement, she was referred to our neuromuscular clinic. Examination showed right-sided scapular winging, mild weakness (MRC grade 4 or 4+) in the right rhomboid, supraspinatus, infraspinatus, deltoid, biceps, and brachioradialis, and moderately severe weakness in the wrist extensors (1) and finger extensors (3). There was patchy sensory loss to pinprick in the lateral aspect of the right upper extremity. NCS showed reduced right radial and musculocutaneous SNAP amplitudes; the median and ulnar SNAPs were normal. Needle EMG showed some abnormal spontaneous activity (fibrillation potentials and positive waves) and high-amplitude long-duration voluntary motor units in the right biceps, brachioradialis, extensor digitorum communis, extensor indicis, and serratus anterior muscles. MRN...
showed thickening with increased T2 signal throughout the course of the right brachial plexus, consistent with right brachial plexitis. The patient improved over the course of 2 years.

**Case 4.** A 37-year-old man developed acute pain in both shoulders, followed 3 days later by weakness in both arms. There was no history of antecedent infection, trauma, or surgery. He was seen by us 3 months later. At that point, he had already noted spontaneous improvement of his weakness. Examination showed left scapular winging and weakness in both deltoids (MRC grade 4) and infraspinatus (4– on the left and 4 on the right). Sensory examination was normal. NCS was normal and EMG showed only a few fibrillation potentials, positive waves, and large voluntary motor units in the C5–6 myotomes bilaterally, including deltoid, infraspinatus, and serratus anterior muscles. Cervical spine MRI was normal. MRN showed increased T2 signal in both brachial plexuses, especially the left, consistent with bilateral brachial plexitis. The patient had improved close to his normal state 4 months after the onset of symptoms.

**Case 5.** A 40-year-old man was evaluated for 2 years of progressive right hand weakness. He had noticed difficulty with his grip and was dropping objects. No sensory symptoms were present except for mild numbness in the right fourth and fifth digits. Examination showed weakness in the right rhomboid (MRC grade 4), spinati (4), deltoid (4), triceps (4–), wrist extensors (1), finger extensors (2), interossei (4–), and thumb abductor (4–) muscles. Sensory examination was normal. Deep tendon reflexes were absent at the right triceps and brachioradialis, and normal elsewhere. NCS showed normal distal CMAP amplitudes in median, ulnar, and radial nerves bilaterally. Normal CMAP amplitude of the extensor indicis despite severe weakness of this muscle suggested the presence of a proximal conduction block in the radial nerve. Bilateral ulnar SNAP amplitudes were reduced and ulnar motor conduction velocities were also reduced across the elbow segments. Needle EMG showed fasciculation potentials and fast-firing units in the extensor indicis muscle. A few positive waves were present in the triceps and deltoid muscles, where a neurogenic recruitment pattern was seen. The cervical paraspinal muscles were normal. MRN showed marked enlargement of the upper and middle trunks of the right brachial plexus with increased T2 signal and no extrinsic mass lesions. Given the progressive nature of this plexopathy in the absence of a mass lesion and with an indirect suggestion of a conduction block on NCS, multifocal motor neuropathy presenting as weakness confined to the right brachial plexus distribution was diagnosed. After intravenous infusion of immunoglobulin (IVig), the patient had dramatic improvement in strength, most notably of the wrist and finger extensors (MRC grade 4).

**Case 6.** A 40-year-old chef with a history of type 1 neurofibromatosis (NF-1) was seen with progressive right arm weakness for 3 years. Examination showed severe weakness in the right upper trunk distribution involving the deltoid (MRC grade 3), biceps (3), brachioradialis (4), rhomboid (4–), spinati (3), and serratus anterior (4+) muscles. Sensation was slightly reduced in the radial aspect of the right hand and forearm. He was areflexic. Cervical spine MRI showed mild disc bulging at C6–7. Conventional MRI of the right brachial plexus showed an enlarged plexus suggestive of a neurofibroma or Schwannoma. Surgical intervention was considered. MRN showed diffuse enlargement of the upper and middle trunks and the lateral cord of the right brachial plexus measuring over 4–5 cm in length but without contrast enhancement (Fig. 2). NCS showed partial motor conduction block in the forearm segments of both median and the right ulnar nerves, with markedly prolonged F-wave latencies. Median and ulnar sensory conduction velocities were reduced and sural SNAP amplitudes were asymmetrically reduced. Needle EMG examination showed no abnormal spontaneous activity and decreased recruitment with a few high-amplitude and long-duration voluntary motor units in the above-named weak muscles. These findings were suggestive of multifocal chronic inflammatory demyelinating polyneuropathy. A right sural nerve biopsy showed many thinly myelinated fibers confirming a demyelinating and remyelinating process. The patient received IVIg with near-total recovery of the severe weakness. He continues to receive IVIg every 2 months and has also been placed on mycophenolate mofetil with stabilization of his symptoms.

**DISCUSSION**

MRN has been used to image the peripheral nervous system directly because of the improved signal-to-noise ratio from dedicated surface coils, which allows higher resolution scanning. The principle of this technique has been elucidated in great detail elsewhere.\(^1,2\) The peripheral nerves are isointense on T1-weighted spin echo and slightly hyperintense on fat-saturated T2-weighted fast spin echo due to the
FIGURE 2. Marked enlargement of the brachial plexus without enhancement: The inversion recovery, fast spin echo, T2-weighted scan shows very high signal and enlargement of the right brachial plexus affecting the C6 root [(A) arrowhead], the upper and middle trunks and the lateral cord [(A) arrows]. There is no enhancement of the right brachial plexus on the postcontrast T1-weighted study [(B), arrows].

presence of endoneurial fluid. Peripheral nerves can be easily distinguished from surrounding tissue because muscle is very hypointense on T2-weighted scans, the inversion recovery technique suppresses signal from adipose tissue, and vessels usually contain signal void. Peripheral nerve disorders from various etiologies, including trauma, compression, and inflammation, usually result in increased T2 signal with increased nerve/muscle signal intensity ratio due to increased water content in the nerves. As with conventional MRI, the T1-weighted sequence is useful for evaluating anatomy and the T2-weighted sequence for demonstrating pathology.

MRN appears to be a highly sensitive new imaging technique to localize and delineate root, plexus, and peripheral nerve lesions. It has been shown to successfully detect cervical and lumbosacral radiculopathies, lumbar plexopathy, and sciatic and median mononeuropathies related to entrapment, trauma, ischemia, neoplastic infiltration, nerve sheath tumor, cyst, and granulomatous infiltration. It is useful in presurgical evaluation to guide operative intervention.

In our study, MRN detected six brachial plexus disorders with inflammatory and demyelinating etiologies. Even though a previous study has shown that the sensitivity of conventional MRI in brachial plexus disorders is 63%, MRI has been utilized primarily for detecting neoplastic and traumatic lesions but not inflammatory brachial plexus lesions. As noted by our study, inflammatory brachial plexus lesions show abnormalities on MRN. MRN has higher sensitivity than conventional MRI in the evaluation of inflammatory brachial plexus lesions. In addition, the presentation of brachial plexitis as predominantly an AIN disorder, although previously reported, has never been verified by imaging studies to be localized to the brachial plexus. Our study of case 1 provides confirmatory radiological evidence for this disease entity with MRN. Although MRN changes can be mild, side-to-side comparisons facilitate the detection of abnormalities. Case 6 is complicated because brachial plexopathy was present in a setting of NF-1 and demyelinating changes on NCS. The management of these two causes differs; one requiring surgery and the other immunomodulation. MRN showed a nonenhancing plexus lesion consistent with demyelination but not neurofibroma, which resulted in effective treatment.

Brachial plexus lesions shown on MRN can be more diffuse and proximal than suggested by NCS/EMG and clinical findings, as exemplified by our case 1. Although clinical presentation and EMG findings suggested a pure anterior interosseous nerve lesion, MRN showed signal changes at the level of trunks and divisions of the brachial plexus. There has been some debate as to why a brachial plexus lesion gives rise to a pattern of predominantly motor involvement of peripheral nerves. Although localization to the brachial plexus has been problematic in many cases, intraneural topography studies have shown that some nerve branches retain a significant localization for considerable distances above the sites of branching. Fiber bundles destined for the
AIN can be identified as high as the medial and lateral roots of the median nerve. Therefore, a selective AIN palsy can occur from a lesion within the brachial plexus. MRN is helpful in detecting this involvement.

MRN is a useful imaging technique in diagnosing peripheral nerve and plexus disorders. It not only helps surgical decision-making, but also facilitates accurate diagnoses and thus appropriate therapy for nonsurgical cases. It is particularly useful to confirm the diagnosis of idiopathic brachial plexitis, which hitherto has largely been a diagnosis of exclusion.

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REFERENCES