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MR NEUROGRAPHY AND BRACHIAL PLEXUS IN TRAUMA

Radio-Diagnosis

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ABSTRACT

Evaluation of the peripheral nervous system and its various pathologies previously was dependant on clinical examination and electrodiagnostic tests. But, electrodiagnostic studies are often time-consuming and provide indeterminate results. These tests can even at times be unfeasible in patients with skin diseases and in cases where deeply situated inaccessible nerves are involved. Imaging of peripheral nerves was considered difficult as only the superficial nerves could be imaged by ultrasound. With the advent of 3T MRI, things began to change. MR Neurography is not operator-dependent unlike ultrasound which is operator dependant. It helps in better depiction of soft tissue abnormalities, subtle signal changes, and offers better visualization of a nerve abnormality as well as any abnormalities in the surrounding tissues. Also, the secondary muscle denervation changes are better seen on MR Neurography examinations. In this study we aim to understand the various imaging patterns of injuries involving the peripheral nerves and brachial plexus in trauma.

KEYWORDS

MR Neurography (MRN), Neuropraxia, Axonotmesis, Neurotmesis, Brachial plexus.

INTRODUCTION

The global prevalence of traumatic peripheral nerve injury is approximately 5%, which also includes brachial plexus injuries [1]. RTA (road traffic accident) is one of the most common causes of trauma, laceration and fracture of the peripheral nerves [2]. In the early days, evaluation of peripheral nerve injury and its outcome after surgery was based on clinical examination and electrophysiologic tests [3-4]. USG (Ultrasonography) and MRI (Magnetic Resonance Imaging) are now considered the most important imaging modalities for evaluation of the nerve injury and its outcome. MR Neurography has been proposed as a useful tool for assessing the anatomical course of peripheral nerve, post-surgical outcomes and the post-operative complications [5]. The high spatial resolution has made detailed imaging of the nerves and perineural tissues possible, becoming thus an important tool in understanding the anatomy of peripheral nerves and helping in assessment of imaging characteristics such as signal intensity (SI), calibre, course, size, fascicular pattern and peri-neural changes. The precise localisation of the pathology can thus help reduce the need for exploratory surgery and allow for targeted surgery.

PERIPHERAL NERVE STRUCTURE (Fig 1& 2)

Every peripheral nerve is made up of fascicles that are grouped nerve fibres, with a surrounding connective tissue that helps in protecting the nerve and also aids in its function. The fundamental conducting unit of nerve fibre is the neuronal axon. Schwann cells envelop and provide support to each and every neuron, although they produce only a conductive lipoprotein layer around certain larger nerves. This process, called as myelination aids in faster conduction. The surrounding layers of the endoneurium, that being perineurium and epineurium organize each nerve into a fascicular structure (Figure 1) [6-7]. The interfascicular tissues and epineurium serve as the "gliding layer" which enables the nerve trunk to mechanically adapt to any deformation. The integrity of the neuron and its supportive connective tissues is very important for the normal functioning of nerve [6, 8].

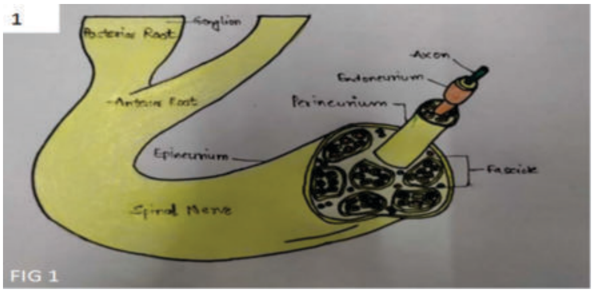

Figure 1: Diagrammatic representation of anatomy of the peripheral nerve.

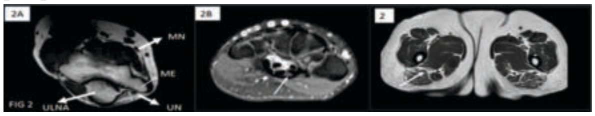

Figure-2

FIG 2A, B AND C: T1 axial image showing normal median and ulnar nerve with fascicular pattern (A) [MN-median nerve, ME-medial epicondyle, UN-ulnar nerve]. STIR axial image showing normal appearance of median nerve in carpal tunnel (B). T2 WI showing normal sciatic nerve with maintained fascicular pattern©.

PERIPHERAL NERVE INJURIES (Fig 3-Fig 5)

Nerve injuries were originally classified into three different categories by Seddon. This classification was based on the extent of damage to the axons and the connective tissues of the nerve, including demyelination [9]. The mildest form of injury being Neuropraxia is defined as a focal demyelination without any obvious damage to the axons or connective tissues. Neuropraxia results from compression or traction of the nerve and causes decreased conduction velocity. The effects can vary from mild asynchronous conduction to severe conduction block, depending on the extent or severity of demyelination. "Saturday night" radial palsy and entrapment neuropathies like carpal tunnel syndrome are some examples of neuropraxia. It is a reversible condition with nerve recovery often occurring after remyelination and sensory/motor functions are completely restored within a few days to weeks without the need for any surgical interventions in most of the cases [10-12].

The next being Axonotmesis. Here, there is direct damage to the axons in addition to focal demyelination; however continuity of the connective tissues of the nerve is maintained. Axonotmesis is commonly seen in stretch and crush injuries [13]. After injury, antero-grade Wallerian degeneration of the distal fibres is completed within a few days.

The most severe form of all is known as Neurotmesis, which is characterised by full transection of the axons and connective tissue layers resulting in complete discontinuity of the nerve. Surgical nerve repair is compulsory in such cases in order to enhance the chances for reinnervation after neurotmesis, [14]. Without surgery, uncontrolled axonal re-growth increase chances of neuroma formation.

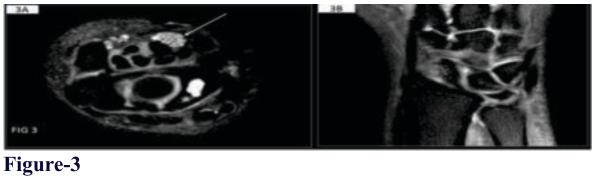

Figure-3

FIG 3A: T2 FS axial showing bulky median nerve with T2 hyperintense signal within
FIG 3B: T2 FS coronal showing associated TFCC injury.

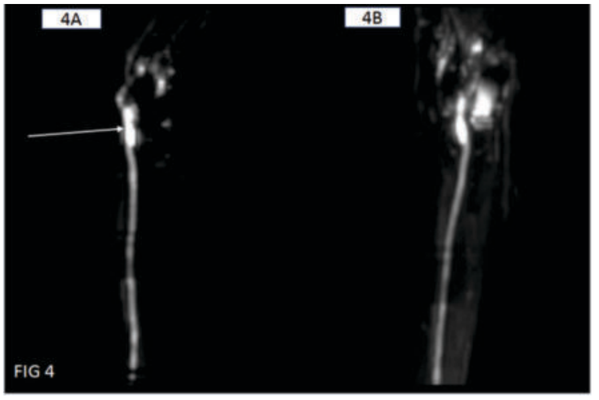

Figure-4
FIG 4A AND 4B: DWIBS coronal images from the level of elbow joint to distal forearm showing thickened hyperintense ulnar nerve with its bulbous dilatation at the elbow joint suggestive of post-traumatic neuroma formation(arrow).

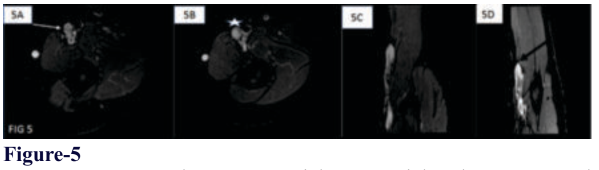

Figure-5
FIG 5A, 5B, 5C and 5D: Sequential STIR axial and STIR coronal images showing the bulky median nerve (arrow) with loss of fascicular pattern and abnormal signal within. There is bulbous dilatation of the nerve at the mid-arm level suggestive of neuroma (asterisk). The median nerve beyond this neuroma is not visualized--in keeping with transection.

Sunderland Classification of Nerve Injury with their imaging and prognosis [15-17]
Seddon classified the peripheral nerve injuries into Neuropraxia, Axonotmesis and Neurotmesis. Sunderland later expanded on this classification to distinguish the extent of damage in connective tissues [18]. In his classification, Grade I and Grade V corresponded with Seddon's Neuropraxia and Neurotmesis with grade II-IV corresponding to various forms of Axonotmesis with increasing amounts of connective tissue damage.

Sunderland Grade I (Seddon Classification-Neuropraxia):
There is increased T2/STIR nerve signal, however, the muscle shows normal signal. Prognosis is usually good with recovery within few days to 3 months.

Sunderland Grade II (Seddon Classification – Axonotmesis):
There is increased T2/STIR signal in nerve and muscle (myelin and axonal injury but the endoneurium is intact). Full recovery usually occurs at a slower rate, but the prognosis is good.

Sunderland Grade III (Seddon Classification - Axonotmesis):
There is enlargement of nerve fascicles which corresponds pathologically to myelin and axonal injury with involvement of endoneurium and intact perineurium. Prognosis depends on the amount of scarring and fascicular involvement. Recovery is usually slow and prognosis is good.

Sunderland Grade IV (Seddon Classification - Axonotmesis):
There is myelin and axonal injury with involvement of endoneurium and perineurium but with intact epineurium. Spontaneous recovery does not occur. Surgical intervention with grafting is necessary in almost all the cases.

Sunderland Grade V (Seddon Classification -Neurotmesis):
Complete disruption of the continuity of a nerve with or without haemorrhage and fibrosis in the gap. Initially, the muscle shows hyperintensity, but with time atrophies and undergoes fatty replacement. Prognosis is generally poor. Surgical intervention is required.

BRACHIAL PLEXUS ANATOMY (Fig 6)
Nerve Roots and Their Anatomic Relations: Between the anterior and middle scalene muscles, ventral rami of C5-C8 and T1 nerve roots unite to form the brachial plexus. In some cases, contributions from C4 and T2 can be seen (Prefixed when C4 is involved and Postfixed when T2 is involved). The dorsal scapular nerve (C5) and long thoracic nerve (C5-C7) to the serratus anterior muscle arise directly from the nerve roots [19].

Trunks and Their Anatomic Relations: C5 and C6 nerve roots form the upper trunk; C7 continues as the middle trunk and the lower trunk is formed by C8 and T1. All the 3 trunks are formed in the inter-scalene triangle.

Divisions, Cords, and Their Anatomic Relations: Each trunk divides into anterior and posterior division which join to form three cords which are seen distal to the lateral border of the first rib. Based on their relationship to the axillary artery, the cords are divided into posterior, lateral, and medial. The anterior divisions of the upper and middle trunks form the lateral cord which gives off the lateral pectoral nerve and contributes to the median and the musculocutaneous nerves. Posterior cord, which gives off the sub-scapular nerve is formed from the posterior divisions of all trunks. Medial cord is the continuation of inferior trunk and gives off the median pectoral nerve, the medial brachial cutaneous nerve, and the medial ante brachial cutaneous nerve.

Branches
The five terminal branches of the cords include: median, ulnar, musculocutaneous, axillary and radial nerves.

On imaging, the normal roots, trunks, divisions, cords and branches of the brachial plexus appear as linear continuous structures isointense to the muscles surrounded by fat. Roots are better visualised on axial plane while the trunks, divisions and cords brachial plexus are better seen on sagittal and coronal planes.
Roots are located in the neural foramina and trunks between scalene muscles. Divisions are located posterior to the clavicle, and cords are located inferior to it.

Various anatomical landmarks aid in identifying the different segments of the brachial plexus. Scalene triangle, formed by the anterior and middle scalene muscles is the most important being anatomical landmark. Roots are located on the medial aspect, while trunks are found immediately lateral to the triangle. The divisions are seen between the first rib and the clavicle. The cords are completely formed at the level of the medial border of coracoid process of scapula. The terminal branches are formed at the level of the lateral margin of pectoralis minor muscle, in the axilla.

Borders of the interscalene triangle are formed anteriorly, by the anterior scalene muscle, posteriorly by both the middle and posterior scalene muscles, and inferiorly by first rib. The contents of interscalene triangle include subclavian vessels, roots, and trunks of the brachial plexus.

In relation to the axillary artery the cords are seen (in clockwise order): anterosuperior (lateral cord), postero-superior (posterior cord), and postero-inferior (medial cord) in the axilla.

Delineating the terminal branches of brachial plexus is usually difficult unless the nerves are involved or concomitant imaging of the arm is performed [20,21].

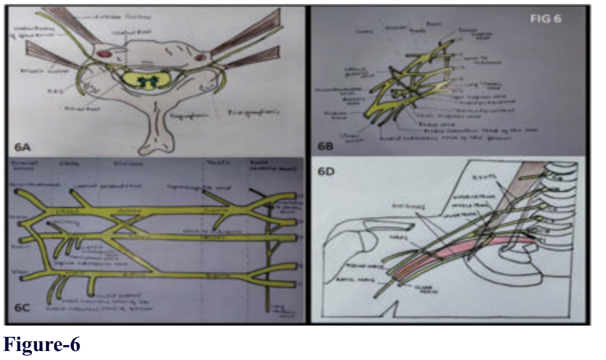

Figure-6

FIG 6A, B, C AND D: 6A: diagrammatic representation of anatomy of spinal nerve (dorsal and ventral roots and also distinguishing between the preganglionic and postganglionic parts). 6B AND 6C: diagrammatic representation of the anatomy of brachial plexus. 6D: showing relation of the roots, trunks, cords and divisions.

BRACHIAL PLEXUS INJURIES (Fig 7- Fig 10):

Motor vehicle crashes/accidents with/ without fractures/ dislocations of the cervical spine, clavicle, shoulder joint are the most common causes of injury to brachial plexus. Other aetiologies include sports injury, fall, gunshot wound and iatrogenic injuries. The injuries are classified into 3 categories from a surgical and prognostic point of view:

1. **Preganglionic**
2. **Postganglionic**
3. **Combination of both.**

The nerve roots should be carefully traced from the spinal cord to the extraforaminal location because nerve root avulsion requires a major procedure, neurotisation. Associated bony injuries/fractures should be looked for. Postganglionic injuries can be classified into- lesions in continuity (requiring rehabilitation or neurolysis) and nerve discontinuity (requiring nerve repair or grafting). The overall spectrum of injuries ranges from mild-severe, Neuropraxia (most common) to Axonotmesis, partial Neurotmesis with neuroma in continuity formation to complete nerve lacerations (Neurotmesis) or nerve root avulsions, the latter being the most severe form of injury. These injuries may simultaneously affect various upper limb nerves, such as the suprascapular, musculocutaneous, and axillary nerves.

MR Neurography plays an important role in demonstrating the location of the nerve damage. It also helps to depict the nerve continuity with or without neuroma formation, or may show a completely disrupted/avulsed nerve, thereby aiding in nerve-injury grading for preoperative planning.

An accurate indirect sign of root avulsion injury is high signal of the paraspinal muscles, on STIR images [24-24]. An abnormality of paraspinal muscles indicates that the injury is proximal to the trunks. A dural tear with formation of a pseudo-meningocele (not pathognomonic for nerve root avulsion) may be seen on conventional MR imaging. Regional denervation muscle changes which are another key finding can also be seen on MR Neurography.

The edema-like signal intensity on T2 weighted images can appear within a few days, while contrast enhancement in the abnormal muscles has been shown to appear within 24 hours of the injury.

Figure-7

FIG 7A, B AND C: T1 coronal image showing T-trunks, D-divisions C-cords with the asterisk representing scalene Medius (A). STIR coronal image showing T-trunks, D-divisions C-cords and B-branches (B).DWIBS coronal image showing C5-T1 nerve roots (C).

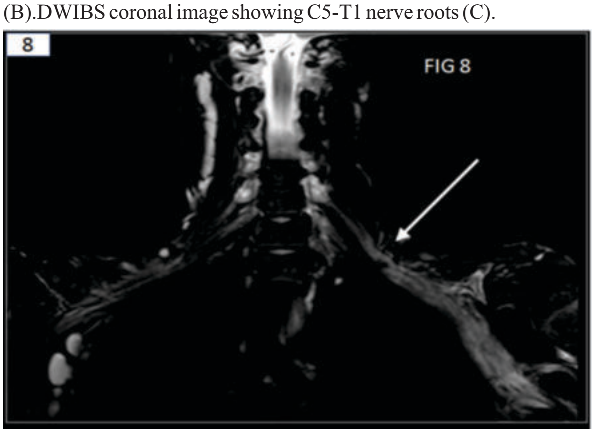

Figure-8

FIG 8: STIR coronal images showing thickening with clumping of distal trunks of C5-T1, all divisions and cords of left brachial plexus (arrow) suggestive of post-traumatic postganglionic neural injury (axonotmesis).

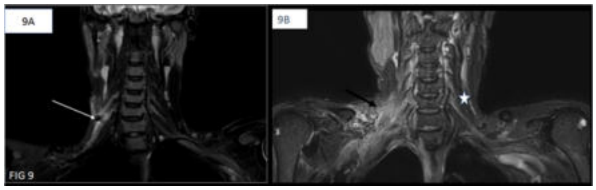

Figure-9

FIG 9A: STIR coronal image shows non-visualization of right C5 distal trunk, (arrow) with its discontinuity suggestive of avulsion with transection. There is partial transection of C6 distal trunk with hyperintense signal in the divisions. FIG 9B: STIR coronal image showing hyperintense signal involving the right pectoralis and scalene muscles (thick black arrow) suggestive of denervation edema. Note is made of normal appearing scalene muscles on the left side (asterisk).

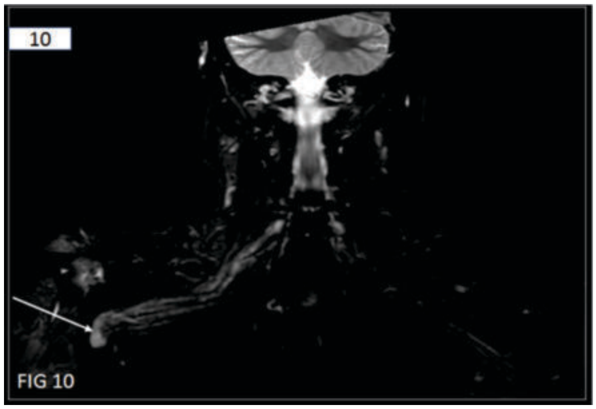

Figure-10

FIG 10: STIR coronal image showing thickening with clumping of trunks and divisions of right brachial plexus. The divisions and cords appear wavy with focal neuroma formation (arrow) seen at the infraclavicular level—post-traumatic neuroma formation with clumping.

CONCLUSION

MR Neurography helps in understanding the anatomy and different imaging patterns involving the peripheral nerves and brachial plexus in trauma and confirming the clinical examination and electrodiagnostic findings and localizing the site of involvement to assist in diagnostic and therapeutic workup and preoperative planning. MRI is arguably the best modality overall for understanding the complex anatomy of brachial plexus and assessing the different pathologies involving the brachial plexus. It is also a useful tool for assessment of postsurgical outcomes and complications in patients who have underwent surgical repair for peripheral nerve injuries.

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