

Original Research Article

IMPORTANCE OF MRI IN THE EVALUATION OF BRACHIAL PLEXUS INJURIES

Abstract

There is a global increase in the incidence of brachial plexus injuries (BPI) due to the increasing participation in extreme sports and the increased number of motor vehicle accidents survivors. Detailed clinical examination is followed by MR imaging. The study was conducted on 30 patients of BPI. The patients with flail upper limbs and suspected root avulsions were subjected to MRI immediately while others underwent MRI after 4-6 weeks. MRI helps to classify an injury as pre- and post-ganglionic or mixed¹, specially in patients where there are both pre- and postganglionic injuries which are difficult to assess. This is important as timing and type of surgery depends on whether the injury is pre- or postganglionic. We recommend that full upper limb should be imaged (only T2W/STIR sequence can be done) to look for muscle edema pattern and movements of diaphragm must be assessed. Contrast studies can also be added if not contraindicated.

Keywords : brachial plexus injuries, MRI imaging, Motor vehicle accidents, atrophy

Introduction:

The upper extremities derive their motor as well as sensory innervation through brachial plexus, a major neural structure. Motor vehicle accidents are major source of injury though sports, radiation injury etc also contribute. There is global increase in the incidence of brachial plexus injuries (BPI) due to the increasing participation in extreme sports and the increased number of motor vehicle accidents survivors. Detailed clinical examination is followed by MR imaging. The major role of MRI is to distinguish pre- from postganglionic injuries, to grade the injuries (Sunderland classification – table 1) and pick up obscure injury with the help of indirect signs like extent and pattern of muscle edema and/or atrophy.

Methods:

The study was conducted on 30 patients of BPI. The patients with flail upper limbs and suspected root avulsions were subjected to MRI immediately while others underwent MRI after 4-6 weeks. MRI was done on 3 Tesla system (GE Healthcare Discovery 750W with GEM Suite, Milwaukee, WI, USA) using head-neck forty coil. Axial T2W (TE=114ms, TR=4800ms, FOV=22cm, slice thickness=5mm, spacing=1mm, frequency=512, band width=50), coronal 3D/STIR (TE=102ms, TR=7000ms, FOV=40cm, slice thickness=2mm, spacing=0, frequency=256, band width=35.71), coronal T1W (TE=7ms, TR=456ms, FOV=22cm, slice thickness=4mm, spacing=0, frequency=352, band width=62.5), STIR neurography (TE=90.9ms, TR=16081.3ms, FOV=40cm, slice thickness=3mm, spacing=0, frequency=100, band width=250), and DW neurography (TE=73.8ms, TR=7000ms, FOV=30cm, slice thickness=4mm, spacing=0.5mm, frequency=100, band width=190) sequences were done. In addition, oblique sagittal T2 fat sat (TE=110ms, TR=5885ms, FOV=25cm, slice thickness=4mm, spacing=1mm, frequency=288, band width=31.25) sequence was done on shoulder ipsilateral to injured brachial plexus, axial STIR (TE=42ms, TR=5465ms, FOV=35cm, slice thickness=4mm, spacing=0.5mm, frequency=352, band width=41.67) of ipsilateral arm, sagittal T2W (TE=76ms, TR=3105ms, FOV=16cm, slice thickness=2.5mm, spacing=0.2mm, frequency=288, band width=35.7) of cervical spine and axial cube T2 (TE=90mm, TR=1360mm, FOV=24cm, slice thickness=1.6mm, spacing=0, frequency=288, band width=83.33) of cervical spine were done.

Location of injury was identified, whether the injury was at root, trunk, division, cord or terminal branch level and whether it was pre- and/or postganglionic. Further, injury was graded according to Sunderland classification (Table 1). MRI findings were correlated with clinical examination at the time of injury/MRI and on follow-up at three months.

Results and Discussion:

The patients ranged from 11 days to 58 years with mean age of 29.4 ± 12.09 years with 26 males and 4 females. 14 patients had left sided, 12 right sided and 4 bilateral injury. Nerve roots were involved in 18, trunks in 17, divisions in 5 and no direct injury of brachial plexus was seen in one patient. Ten patients had preganglionic injury, 17 had postganglionic injury while three had both pre- and postganglionic injuries. C5 nerve root was involved in 21 patients, C6 in 27, C7 in 18, C8 in 19 and T1 in 14 patients. Grade I injury was seen in three patients, grade III in nine, grade V in eight and multiple grades of injuries were seen in 10

patients. No isolated grade II and IV injuries were seen. Figure 1 to 8 show some selected cases. There was excellent correlation between MRI and clinical findings. 23 cases showed total correlation, seven showed partial correlation. Six patients showed complete recovery, six showed partial recovery while three showed minimal recovery and 15 no recovery. Patients having lower grades of injury (grade I/II) showed complete recovery and those with higher grades and extensive injury showed minimal or no recovery.

MRI helps to classify an injury as pre- and post-ganglionic or mixed¹, specially in patients where there are both pre- and postganglionic injuries which are difficult to assess. This is important as timing and type of surgery depends on whether the injury is pre- or postganglionic. In our study preganglionic involvement was seen in 10 patients and postganglionic in 17 while both pre- and post-ganglionic injuries were seen in 3 patients.

Each BPI is different. Brachial plexus injuries must be assessed in terms of extent of injury, site of injury (nerve roots, root/trunk/division/cord/terminal branches involved, pre- or postganglionic), grade of injury, associated injuries and status of others surrounding structures

like muscles and nerves to decide treatment (conservative or surgical, type of surgical treatment, timing of surgery) and prognosis. Status of surrounding muscles and nerves helps in deciding their utility for neurotization. Nerve roots with pre-ganglionic injury cannot be used as a nerve source for reconstruction, whereas in postganglionic injuries, connection to CNS is intact and the nerve can be used as a source of axons².

Detailed examination is a must to assess motor and sensory functions, presence or absence of Horner's syndrome, to know the dermatomes and myotomes involved and whether the injury

is pre- or postganglionic. Some signs of preganglionic injury are Horner's syndrome, injury to very proximal nerves like the long thoracic nerve (winging of scapula), dorsal scapular nerve (rhomboid paralysis) and phrenic nerve (paralysis of ipsilateral hemidiaphragm), severe pain in an anaesthetic extremity, constant burning background pain and periodic sharp paroxysms of shooting pain and absence of Tinel's sign in supraclavicular fossa³. This gives idea about the extent of injury and whether early imaging and treatment are required.

EMG at rest and with activity helps diagnose and localize the level of the lesion. Denervation

changes can be seen in proximal muscles as early as 10 to 14 days after injury and 3 to 6 weeks post injury in distal muscles⁴. It can estimate the severity of axon loss, and completeness of the lesion which is difficult on MRI. EMG can also distinguish preganglionic from postganglionic lesions. EMG can evaluate muscles that are difficult to test clinically and can quantify the extent of denervation. Because of Wallerian degeneration, the EMG signs of denervation are not reliably demonstrated until 3 to 4 weeks after injury⁵, and hence these examinations should not be done earlier. EMG is also used in serial evaluations of the injury, to search for signs of reinnervation, which are seen several weeks before the onset of detectable voluntary muscle contraction³. EMG recovery does not always equate with clinically relevant recovery, either in terms of quality of regenerate or extent of recovery. Conversely, EMG evidence of reinnervation may not be detected in complete lesions despite ongoing regeneration, when target end organs are further distal⁴. Complex muscle action potentials (CMAPs) are not useful in complete distal lesions because of the necessary time for regeneration to occur into distal muscles, but are useful in partial lesions where their size is proportional to the number of functioning axons⁴. Nerve conduction velocity studies can also differentiate pre- from postganglionic injury. Spinal or cortical potential (SEPs) cannot be elicited in preganglionic lesions, despite their normal peripheral sensory NCV. Both NCV and SEPs are absent in postganglionic lesions as well as in combined pre and postganglionic lesions³. Negative SEP examination is clinically more useful than a positive one³. Pre or post ganglionic localization of lesion can be done with the help of sensory nerve action potentials (SNAPs). In a preganglionic lesion, in spite of the extremity having no sensation in the distribution of the affected nerve root, there will be SNAPs. The SNAPs are not present in postganglionic or combined pre- and postganglionic lesions⁶. Electrodiagnostic studies reveal subclinical injuries and recognize recovery⁶. The presence of NAP indicates the viability of thousands of axons rather than hundreds as seen with other techniques like SEP where integrity of few hundred fibers is sufficient for positive response. Hence NAP more strongly correlates with recovery than SEP. More than 90% of patients with a preserved NAP gain clinically useful recovery⁴. The presence of NAP bodes well for recovery after neurolysis alone, without the need for additional treatment like neuroma resection and grafting. However, there is no adequate peripheral stimulation site for C5 root³ and hence NCV studies cannot help in C5 root injuries. MRI is the imaging investigation of choice. It can directly show preganglionic injury.

Expansion of extravascular bed and extracellular space can lead to enhancement of paraspinal muscles as early as 24 hours after injury. This can help in identifying functionally impaired nerve root even when their anatomy is preserved and guide surgeon in avoiding abortive reconstructive procedures. Contrast enhancement is an accurate indirect sign of root avulsion injury [6]. However, timing of MRI is very important. MRI should be deferred for 3 to 4 weeks as clear picture of injury appears once nerve injury is demarcated and edema, hematomas and inflammation of acute phase subside, but there are some situations where urgent surgical treatment is recommended like major vascular injuries, root avulsions, flail upper limb, sharp open injuries, high velocity gunshot wounds⁶. In such situations, MRI should be done as early as the patient stabilizes. It is very good at directly showing brachial plexus as well as showing indirect signs of nerve injury like muscle denervation edema. In our case number 7, no direct injury to brachial plexus could be seen, but edema of serratus anterior muscle suggested injury to long thoracic nerve. Many of our patients showed edema of trapezius, a muscle supplied by spinal accessory nerve. Though this is not related to brachial plexus, but likely injury to spinal accessory rules out possibility to use this nerve for nerve grafting or neurotization and hence helps surgeon in deciding which nerve to harvest for this purpose. MRI, in conjunction with EPS, can grade injuries which helps in deciding the type of treatment. Grade I and II injuries mostly require conservative treatment, while grade III injuries may be treated conservatively or surgically. These patients should be treated conservatively to allow time for natural reinnervation and if there is no improvement after 3 months, surgical treatment should be planned. Grade V injuries, where there is gap in the nerve, can be treated with nerve grafting (sural nerve is one of the best donor nerves which can provide graft up to a length of 30cm). Our case number 2, 6, 8, 12, 19, 20, 25 and 28 had grade V injury and case number 6, 13, 14, 16 and 28 showed measurable nerve gap on MRI, making them ideal candidates for nerve grafting. By giving the size of gap, MRI helps in planning the source and length of graft needed.

Grade IV injuries (neuroma in continuity) should be assessed intraoperatively with EPS. If there is nerve action potential (NAP) across the neuroma, it means that reinnervation is taking place and only neurolysis may be sufficient, but if there is no NAP across neuroma, the neuroma has to be excised and nerve grafting done³. Preganglionic injuries are always surgical candidates and require early surgery. Nerve grafting is not possible in

preganglionic injuries due to absence of proximal stump. Neurotization or nerve transfers is the treatment of choice in such cases. The status of surrounding nerves, muscles, etc. shown by MRI helps in deciding which nerve to harvest for neurotization. Even fracture ribs are important because in cases of fracture ribs, adjacent intercostal nerves are mostly injured making them unlikely candidates for use in nerve transfers. Nerve root repair and re-implantation of ventral roots into spinal cord are emerging techniques⁷. The end-to-side neurorrhaphy with removal of the epineurial sheath is an up-to-date surgical technique⁸. Assessment of muscle atrophy and fatty degeneration using quantitative three-point Dixon MRI sequence can reflect active flexion and muscle force after BPI⁹.

MRI examination can add considerable value to the evaluation of these patients, since the clinical examination is often fraught with false negatives and positives. There were three patients during our study who came for MRI brachial plexus with clinical diagnosis of brachial plexus injury, but who showed normal brachial plexus on MRI, but had tears of surrounding muscles. They could not move their upper arms properly due to muscle tear and also had pain giving false impression of brachial plexus injury. These cases were excluded from the study. Here, MRI did great value addition by ruling out brachial plexus injury and changing treatment decision. One patient had a fall and then has symptoms of brachial plexopathy, but imaging showed brachial plexus involved by carcinoma (Fig. 9) and there was no sign of injury to brachial plexus. Another patient had brachial plexopathy due to cervical rib (Fig. 10). In the latter two patients, minor trauma just drew attention towards the pathology which was diagnosed only on MRI examination. Cervical spondylosis is another mimicker of brachial plexopathy. MRI examination mostly establishes the cause of symptoms in such cases. Cervical spine should always be imaged along with brachial plexus. MRI not only changed diagnosis in these patients, it also added value to the cases where clinical diagnosis was correct. In case 1, MRI pinpointed the injury to C5-C6 level, while clinical examination suggested pan-brachial plexus injury. In case 6, MRI showed medial cord injury which was missed clinically, re-examination showed weakness of thumb and index finger consistent with medial cord injury. In case 12, MRI correctly localised injury to postganglionic segment while clinical examination suggested preganglionic injury. In case 15, MRI picked up right lateral cord neuroma and left brachial plexus injuries which were missed on

clinical examination. Subtle opposite plexus injury were picked in addition to the other side injury in case 25 and 30. In cases of pan-brachial plexus injury (case 17), MRI could grade different injuries, thus helping in treatment planning.

Moreover, in cases where it was difficult to make out exact nerve structures injured due to edema, hematoma and inflammation, muscle edema pattern identified on MRI helped reach a decision as in case 20.

The judicious and complementary use of all these techniques ultimately helps in deciding type of treatment, timing of treatment and guiding and monitoring response. If electromyography suggests that the damage is non-degenerative, conservative management is indicated⁶ which includes slings, splints, physical therapy, passive range of movements, electrical stimulation and therapeutic massage for edema and scar management and adequate pain relief. An advancing Tinel's sign suggests a recovering lesion⁴. Persistent pain (lasting for more than six months) is a bad prognostic sign for neurological recovery, no matter where the lesion is located³. If there is no recovery in few months, reconstruction should be planned⁶. Surgery is also recommended in patients with clinical and EMG signs of recovery of distal branches instead of proximal axons⁸. Spontaneous reinnervations require some time, but more than 6 to 18 months' denervation can lead to motor end plate failure¹⁰. In general, the optimal time of surgical intervention is regarded as 3 to 6 months after injury¹¹. Nerve reconstruction should not be attempted more than 9 months after the accident. There is complete disorganization and muscle is replaced by fatty tissue two years after injury⁸. Recovery of elbow flexion is most important followed by shoulder abduction. Restoration of not only motor function, but also sensation should be aimed for prevention of injuries, and modulation of the pathogenesis of the deafferentation pathway of pain⁶. Arthrodesis, tendon transfer, and functional free muscle transplantation are other treatment options⁸ where nerve grafting/neurotization is not possible.

Outcome depends on patient's age, interval between injury and surgery (denervation period), the two coaptation sites, long nerve grafts, scar tissue, ischemia and the degree of root lesion³.

The ultimate aim should be to make patient at least self-reliant in daily activities.

Diagnostic accuracy of MRI has been widely reported in literature. MRI was helpful in making the diagnosis in presence of nondiagnostic EMG & NCV1. There are limited studies on the impact of MRI on treatment and outcome. Chhabra et al found moderate to major impact of MRI in the evaluation of the majority of upper extremity neuropathies (84%)¹². In addition, negative examination results allowed the patients to forego further expensive work-ups, and surgery was avoided in such cases.

Our study had some limitations too. The overall sample size was comparatively small owing to time constraint. Forearm and hand muscles were not imaged for edema. Diaphragm movements were not assessed and contrast enhanced MRI was not done which could assist in refining diagnosis of preganglionic injury. Contrast studies can specially be helpful where there is no pseudomeningocele formation and suspicion of preganglionic injury is high. Based on our observations, we recommend that full upper limb should be imaged (only T2W/STIR sequence can be done) to look for muscle edema pattern and movements of diaphragm must be assessed. Contrast studies can also be added if not contraindicated.

Conclusion:

MRI is very useful in proper diagnosis and assessment of extent of brachial plexus injury helping in deciding treatment (conservative or surgical) and timing of surgery. MRI also assesses surrounding normal muscles and nerves in terms of their suitability for nerve transfer.

Compliance with ethical requirements:

All procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and with the Helsinki Declaration of 1975, as revised in 2008 (5). Informed consent was obtained from all patients for being included in the study. This article does not contain any studies with animal subjects.

Consent :

Written informed consent was obtained from the patients for publication and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

References:

1. Mallouhi A, Marik W, Prayer D, Kainberger F, Bodner G, Kasprian G. 3 T MRI

tomography of the brachial plexus: structural and microstructural evaluation. *Eur J Radiol* 2012; 81:2231-2245.

2. Yoshikawa T, Hayashi N, Yamamoto S, Tajiri Y, Yoshioka N, Masumoto T, et al. Brachial plexus injury: clinical manifestations, conventional imaging findings, and the latest imaging techniques. *Radiographics* 2006;26(1):S133-S43

3. Siqueira MG, Martins RS. Surgical treatment of adult traumatic brachial plexus injuries. *Arq Neuropsiquiatr* 2011;69(3):528-35

4. Moran SL, Steinmann SP, Shin AY. Adult brachial plexus injuries: Mechanism, patterns of injury, and physical diagnosis. *Hand Clin* 2005;21:13-24

5. Warren J, Gutmann L, Figuerca AS Jr, Bloor BM. Electromyographic changes of brachial plexus root avulsion. *J Neurosurg* 1969;31:137-40.

6. Park HR, Lee GS, Kim S, Chang JC. Brachial plexus injury in adults. *The Nerve* 2017;3(1):1-11

7. Carlstedt T, Anand P, Hallin R, Misra PV, Noren G, Seferlis T. Spinal nerve root repair and reimplantation of avulsed ventral roots into the spinal cord after brachial plexus injury. *J Neurosurg* 2000;93:237-47

8. Sakellariou VL, Badilas NK, Stavropoulos NA, Mazis G, Kotoulas HK, Kyriakopoulos S, et al. Treatment options for brachial plexus injuries. *ISNR Orthopedics* 2014;2014:1-10

9. Duijnisveld BJ, Henseler JF, Reijnierse M, Fiocco M, Kan HE, Nelissen RGHH. Quantitative Dixon MRI sequences to relate muscle atrophy and fatty degeneration with range of motion and muscle force in brachial plexus injury. *Magn Reson Imaging* 2017;36:98-104

10. Viguie CA, Lu DX, Huang SK, Rengen H, Carlson BM. Quantitative study of the effects of long term denervation on the extensor digitorum longus muscle of the rat. *Anat Rec* 1997;248:346-54

11. Kato N, Htut M, Taggart M, Carlstedt T, Birch R. The effects of operative delay on the relief of neuropathic pain after injury to the brachial plexus: a review of 148 cases. *J Bone Joint Surg Br* 2006;88:256-9

12. Chhabra A, Thakkar RS, Andreisek G, Chalian M, Belzberg AJ, Blakeley J, et al. Anatomic MR imaging and functional diffusion tensor imaging of peripheral nerve tumors and tumorlike conditions. *AJNR Am J Neuroradiol* 2013; 34(4): 802-7.

Degree of nerve injury	MRN (signal intensity)	Recovery potential	Surgery
I Neurapraxia	Nerve-increased T2 Signal intensity Muscle-Normal	Full	None
II Axonotemesis	Nerve-increased T2 signal intensity and diffusely enlarged	Full	None
III	Fascicles-enlarged or effaced due to edema Muscles-denervation	Usually slow, incomplete	None or Neurolysis
IV NIC-neuroma in continuity	Nerve-focally enlarged with heterogeneous signal intensity. Underlying diffuse abnormality ± fascicles disrupted with heterogeneous SI-NIC Muscles-denervation	Poor to none	Nerve repair, graft or transfer

V Neurotmesis	Complete nerve discontinuity ±hemorrhage and fibrosis in the nerve gap and end-bulb neuroma proximally. Epineurial thickening Muscles-denervation	None	Nerve repair, graft or transfer
Degree of nerve injury	MRN (signal intensity)	Recovery potential	Surgery
VI Mixed injury (I to V)	Variable findings along the circumferential segment of the nerve (I-V) with heterogeneous signal intensity due to fibrosis	Variable, can be poor to none	Neurolysis, nerve repair, graft or transfer

Table 1: Table showing degrees of postganglionic nerve injuries (Sunderland classification)

Figure Legends:

Fig 1

(a) 3D T2W/STIR coronal images showing slight thickening and hyperintensity of left C5 & C6 postganglionic roots and upper trunk

(b) T2W/STIR coronal image showing edema in left supraspinatus and infraspinatus muscles

Fig 2

(a) 3D T2W/STIR coronal image showing pseudomeningocele at right C6 root (white arrow)

(b & c) Coronal diffusion weighted image & negative image showing kinking of posterior cord laterally (there was healed fracture rib at this site) (blue arrow) and discontinuity and end neuroma at proximal fragment (red arrow) at medial

cord

(d) T1W axial image showing atrophy of infraspinatus, subscapularis, pectoralis major and minor

Fig 3

(a) Coronal diffusion neurography showing normal brachial plexuses

(b) T2W/SPIR axial image showing edema and atrophy of right serratus anterior muscle (arrow)

Fig 4

(a, b & c) 3D T2W/STIR coronal, T2W sagittal cervical spine, coronal diffusion neurography show pseudomeningocele s/o preganglionic injury to left C6, C7, C8 & T1 nerve roots, thickening and irregularity of left postganglionic C6, C7, C8 and T1 nerve roots and left middle and lower trunks

(d) T2W/STIR coronal image showing edema of supraspinatus, infraspinatus, fracture left upper ribs and left pleural effusion

Fig 5

(a) T2W/STIR coronal image showing pseudomeningocele at right C7 nerve root level (C6-C7 neural foramen)

(b) T2W/STIR coronal image showing discontinuity of postganglionic C5 (red arrow) and C6 (yellow arrow) nerve roots

(c) T2W/STIR sagittal image showing thickening, hyperintensity and blurred fascicles of lateral, posterior and medial cords

(d) T2W/STIR oblique sagittal image showing edema of supraspinatus, infraspinatus, subscapularis and teres minor muscles

(e) T2W axial image showing atrophy of pectoralis major, minor and also infraspinatus and subscapularis muscles

Fig 6

(a & b) 3D T2W/STIR coronal image and its negatives showing hyperintensity and thickening of B/L C5, C6, C7 & C8 postganglionic roots and neuroma in continuity in right lateral cord distally (arrow)

(c) T2W/STIR oblique sagittal image showing slight denervation edema in right supraspinatus, infraspinatus, teres minor and deltoid muscles

(d) T2W/STIR axial image showing edema of biceps brachii muscle (arrow)

Fig 7

(a) T2W axial image showing pseudomeningocele at left D1-D2 neural foramen (arrow)

(preganglionic injury D1)

(b& c) 3D T2W/STIR and diffusion neurography coronal images showing thickening, irregularity and hyperintensity of left C5 nerve root and neuroma in continuity

(arrow) in left C7 postganglionic root

(d) Negative image of diffusion neurography coronal image showing normal cords on right side (red arrow) and discontinuous cords on left side (black arrow)

(e) T2W/STIR coronal image showing edema in left supraspinatus and subscapularis muscles

Fig 8

(a& b) 3D T2W/STIR coronal image and its negative showing entanglement and probable discontinuity of left upper and lower trunk with a small (5mm) gap in lower trunk

(arrow)

(c) T2W/STIR oblique sagittal image showing edema in supraspinatus, infraspinatus with tear in superior aspect of subscapularis muscle

Fig 9

T1W coronal image showing involvement of left brachial plexus by carcinoma larynx

Fig 10

(a& b) Coronal diffusion neurography and its negative image showing indentation of right brachial plexus by cervical rib

(c) T1W oblique coronal image showing gross atrophy of supraspinatus, infraspinatus and subscapularis

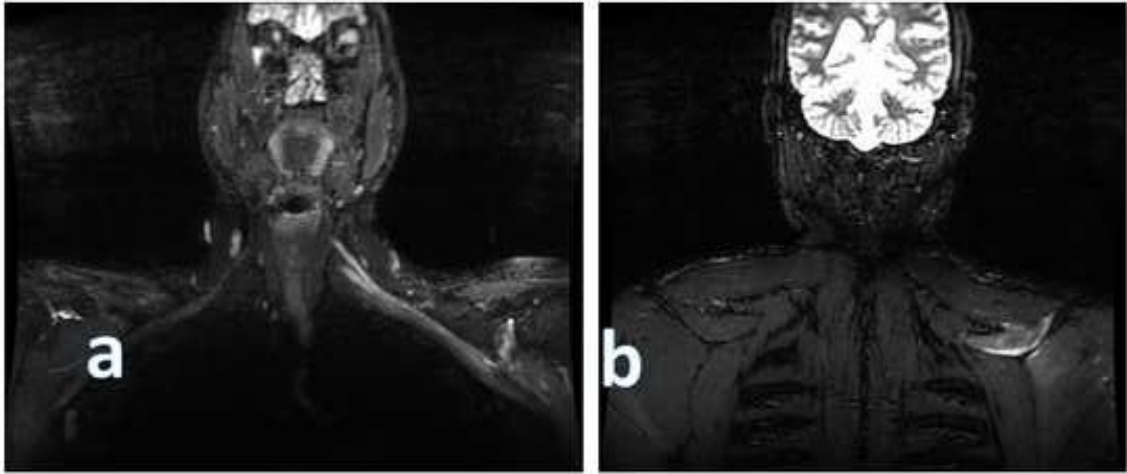


Fig.1

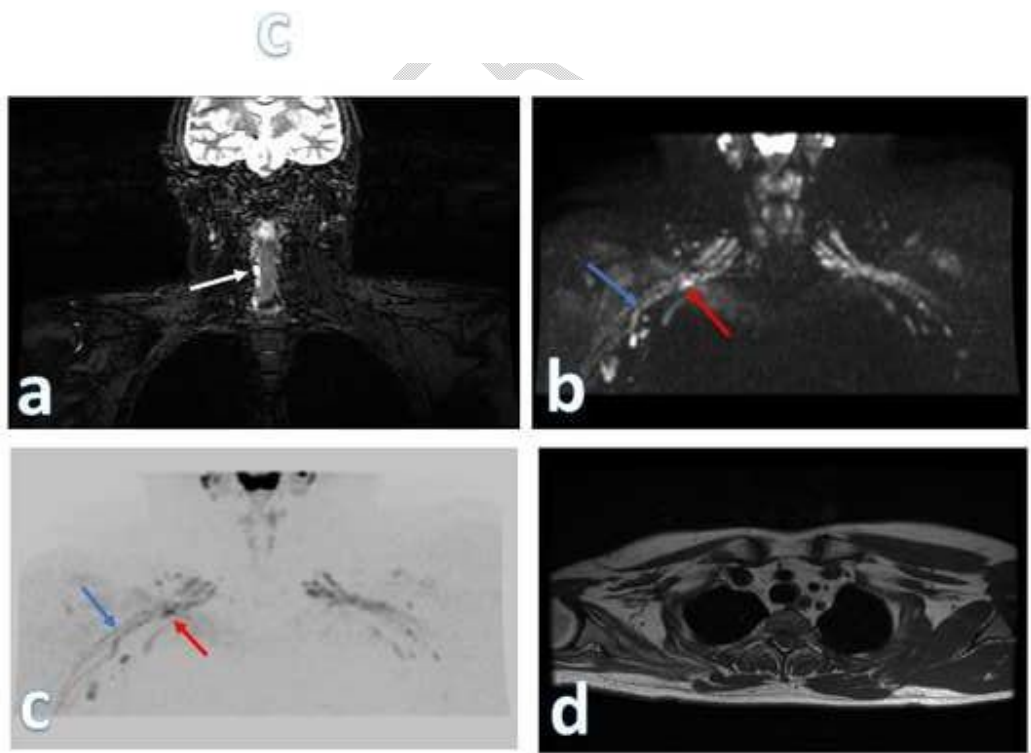


Fig.2

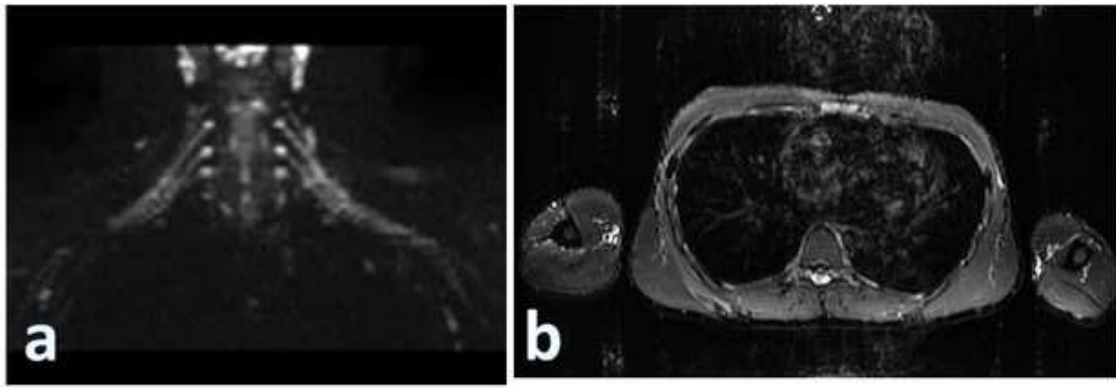


Fig.3

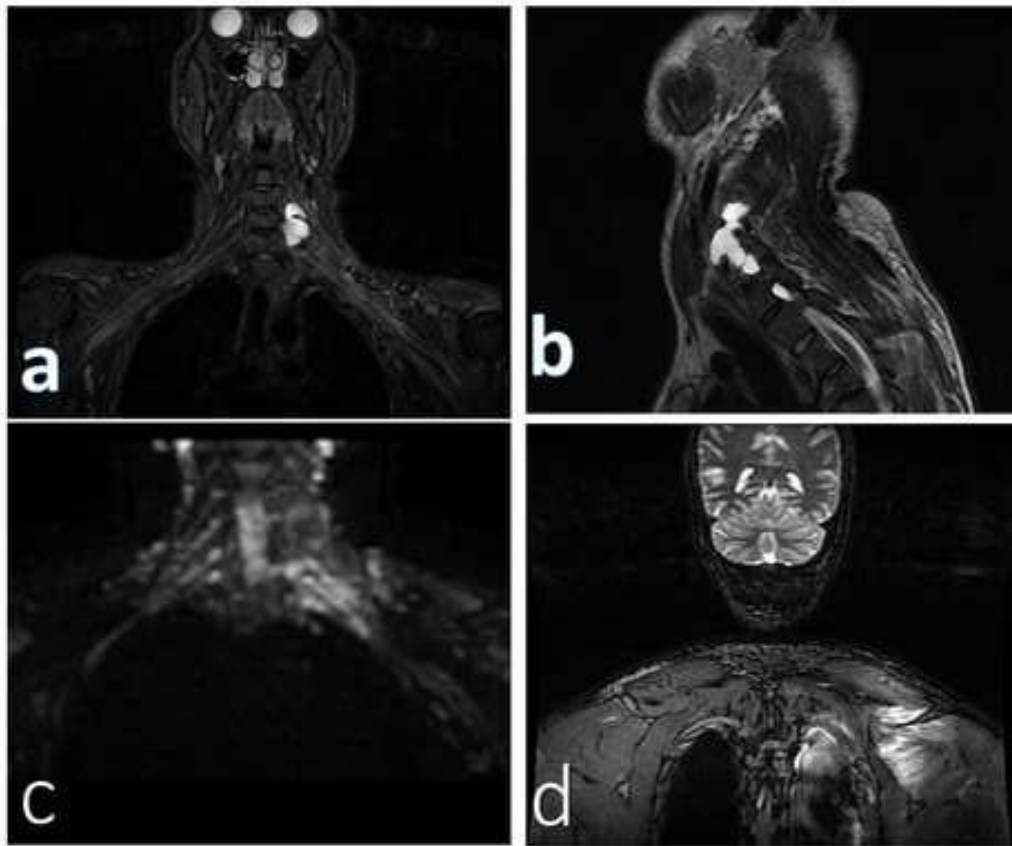


Fig.4

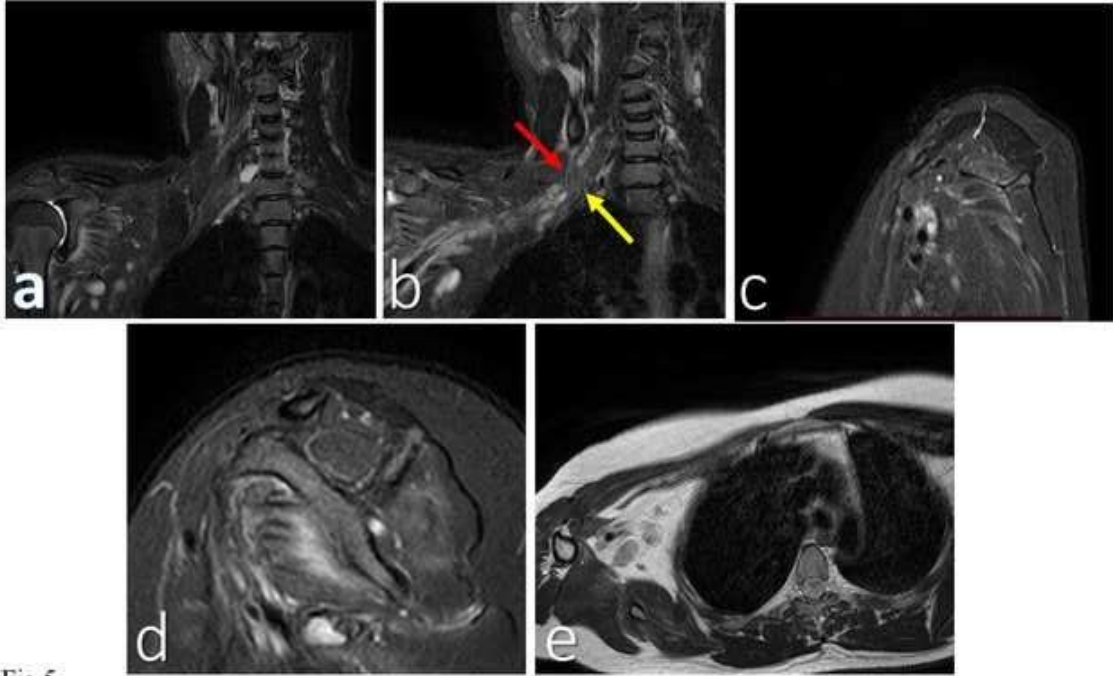


Fig.5

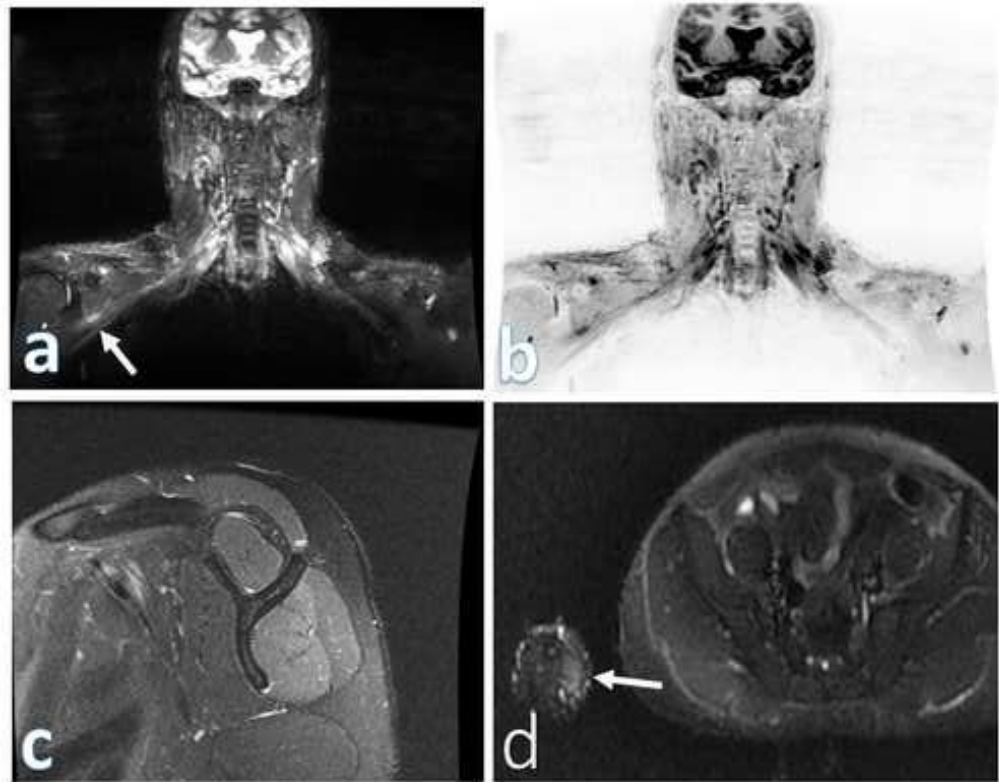


Fig.6

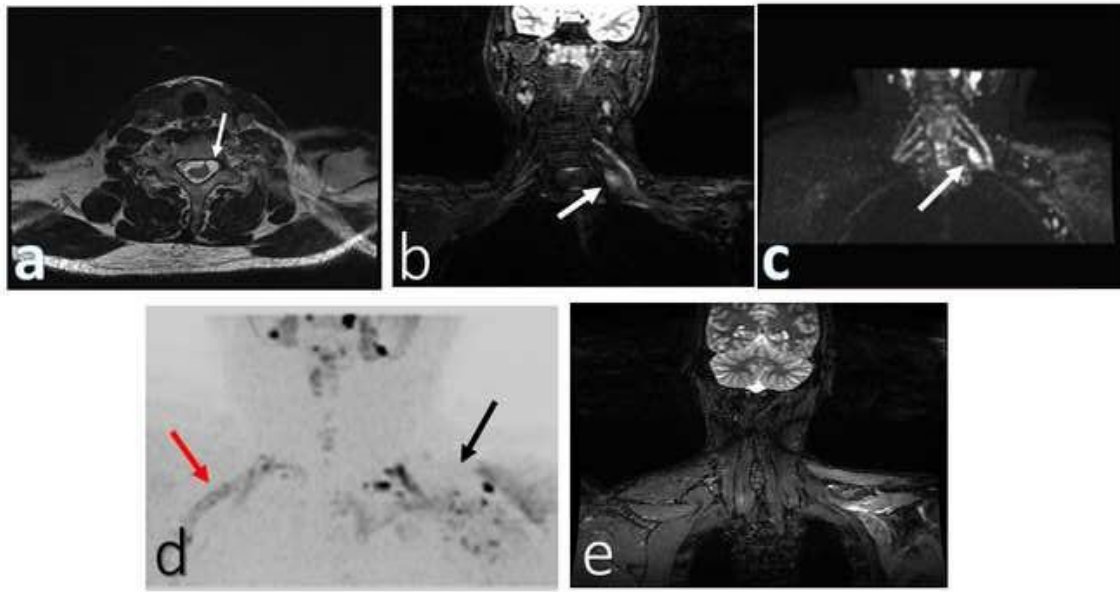


Fig.7

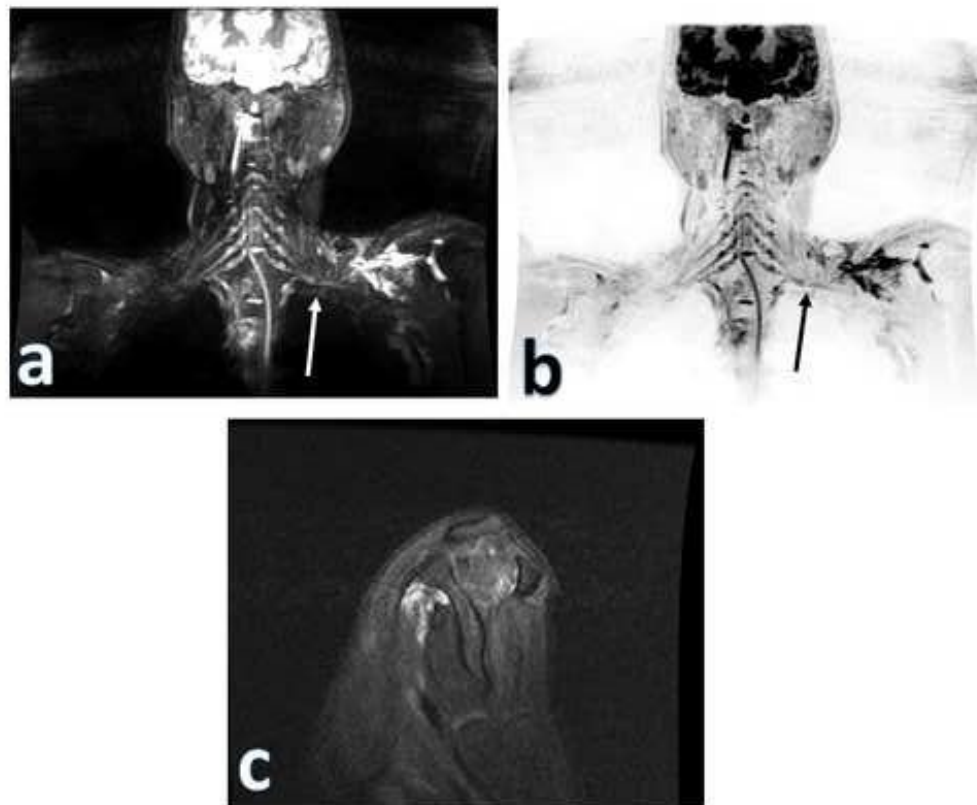


Fig.8

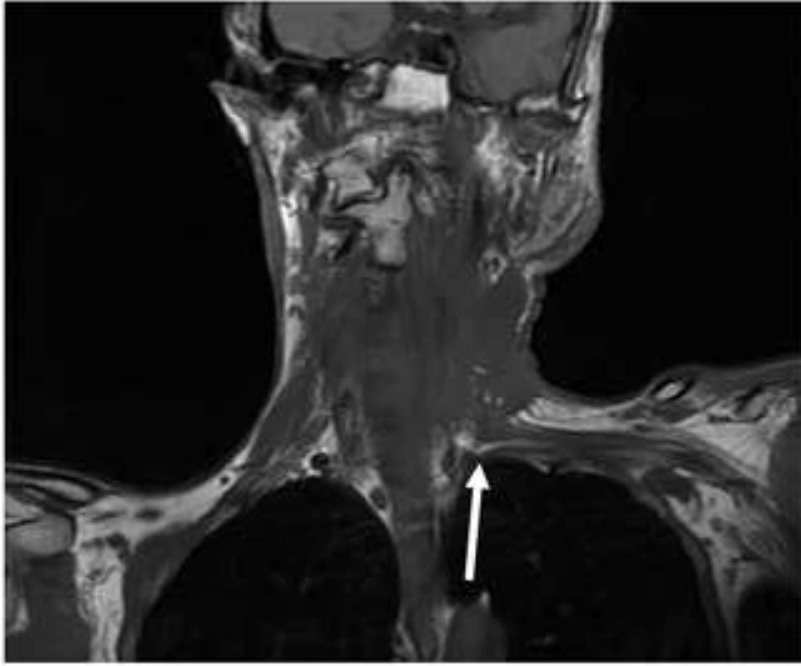


Fig.9

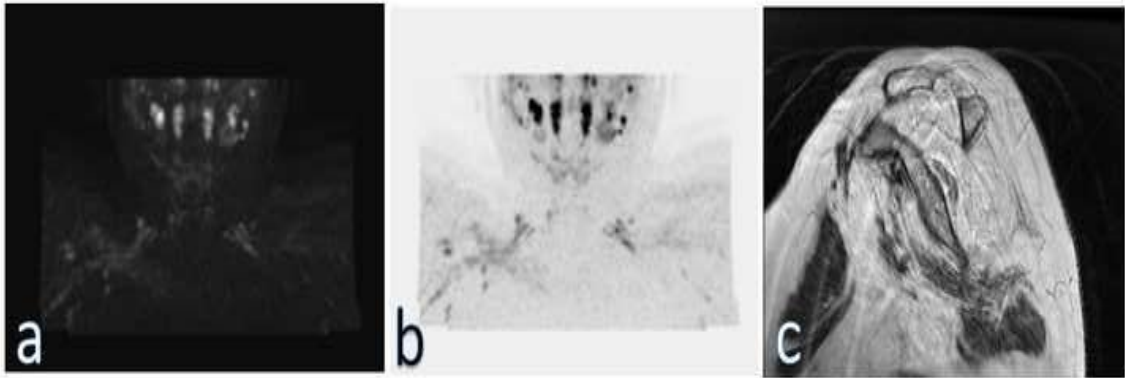


Fig.10