MRI of the brachial plexus: A practical review

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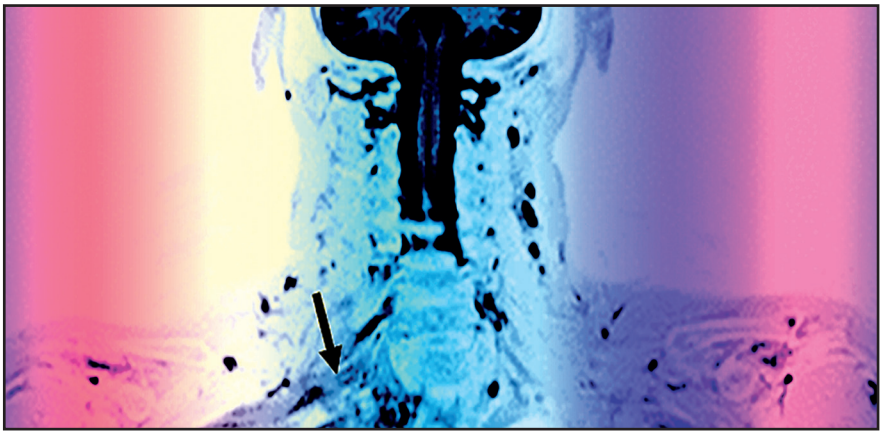
Diagnosing brachial plexus pathology can be clinically challenging, often necessitating further evaluation with MRI. Owing to its vague symptomatology, uncommon nature, and complex anatomy, the brachial plexus presents a diagnostic dilemma to clinicians and radiologists alike and has been the subject of many prior reviews offering various perspectives on its imaging and pathology.[1-5] The objective of this review is to provide the general radiologist with an up-to-date, practical approach to understanding the anatomy, pathology, and imaging of the brachial plexus.

Imaging anatomy of the brachial plexus

The brachial plexus (BP) provides sensory and motor innervation to the ipsilateral shoulder, chest, arm, and hand. Arising from the C5-T1 ventral rami of the spinal cord, the brachial plexus is divided anatomically into roots, trunks, divisions and cords (Figure 1). Upon exiting their respective neural foramina, the roots travel in the interscalene space, bounded anteriorly by the anterior scalene muscle, posteriorly by the middle/posterior scalene muscles, and inferiorly by the subclavian artery/first rib.[2, 6] At the lateral aspect of the middle scalene muscle, the upper two roots (C5 & C6) join to form the upper trunk, the middle root (C7) continues on as the middle trunk, and the lower two roots (C8 & T1) join to form the lower trunk.[1, 2, 6] The trunks course superoposterior to the subclavian artery and each divides into anterior and posterior divisions in the costoclavicular triangle. The costoclavicular triangle has the following boundaries: the clavicle superiorly, subclavius muscle anteriorly, and the first rib and middle scalene muscles posteriorly.[1, 2, 6] At the lateral border of the first rib, the divisions unite to form the medial, lateral, and posterior cords according to their relation to the ipsilateral subclavian/axillary artery.[1, 2, 5]



Terminal branches and innervation

The cords in turn divide into the terminal nerves: ulnar, median, musculocutaneous, radial, and axillary at the border of the pectoralis minor muscle.[1, 2, 6] Smaller branches arise from various segments of the brachial plexus, and are depicted in the graphical representation of the brachial plexus (Figure 1). Various segments of the brachial plexus innervate several important structures. Specifically, the posterior cord innervates the latissimus dorsi, teres major, and subscapularis muscles. The supraspinatus and infraspinatus muscles receive innervation from the upper trunk via the suprascapular nerve. The lateral cord innervates the biceps and coracobrachialis muscles via the musculocutaneous nerve. The medial cord

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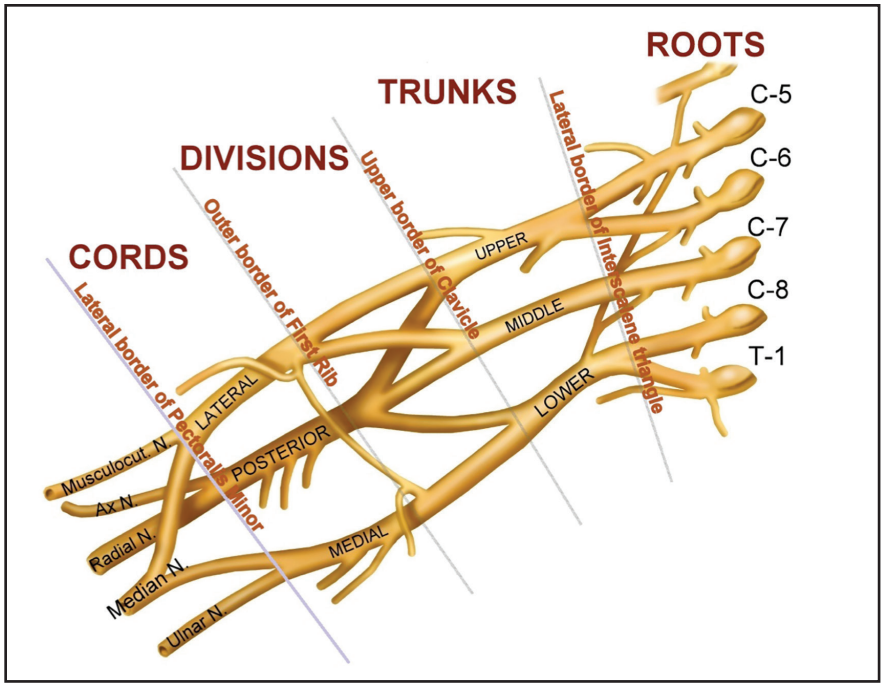

FIGURE 1. *Brachial plexus anatomy. Medical illustration showing the normal anatomy of the brachial plexus. (Used with permission from Rehman I, Chokshi FH, Khosa F. MR Imaging of the Brachial Plexus.* Clinical Neuroradiology. *September 2014, Volume 24 (3), pp 207-216. Springer.)*

innervates cutaneous structures and the ulnar nerve.[1,2,6]

Tracing the peripheral nerves back to the spinal level, the C5 root is responsible for supraspinatus, infraspinatus, and the deltoid; C6 is responsible for biceps; C7 for forearm extensors and triceps; with C8 and T1 providing innervation for finger flexors and the intrinsic muscles of the hand. The normal MR anatomy of the brachial plexus is included in Figure 2.

A practical MR imaging protocol

The goal of imaging the BP is to visualize the entire course of the neural network from its preganglionic segments (eg, nerve rootlets and intraforaminal nerve segments) and the postganglionic segments from the dorsal root ganglion (DRG) to the terminal branches. Focused multiplanar, multisequence MRI is required to completely image the brachial plexus and offers superior contrast resolution compared to computed

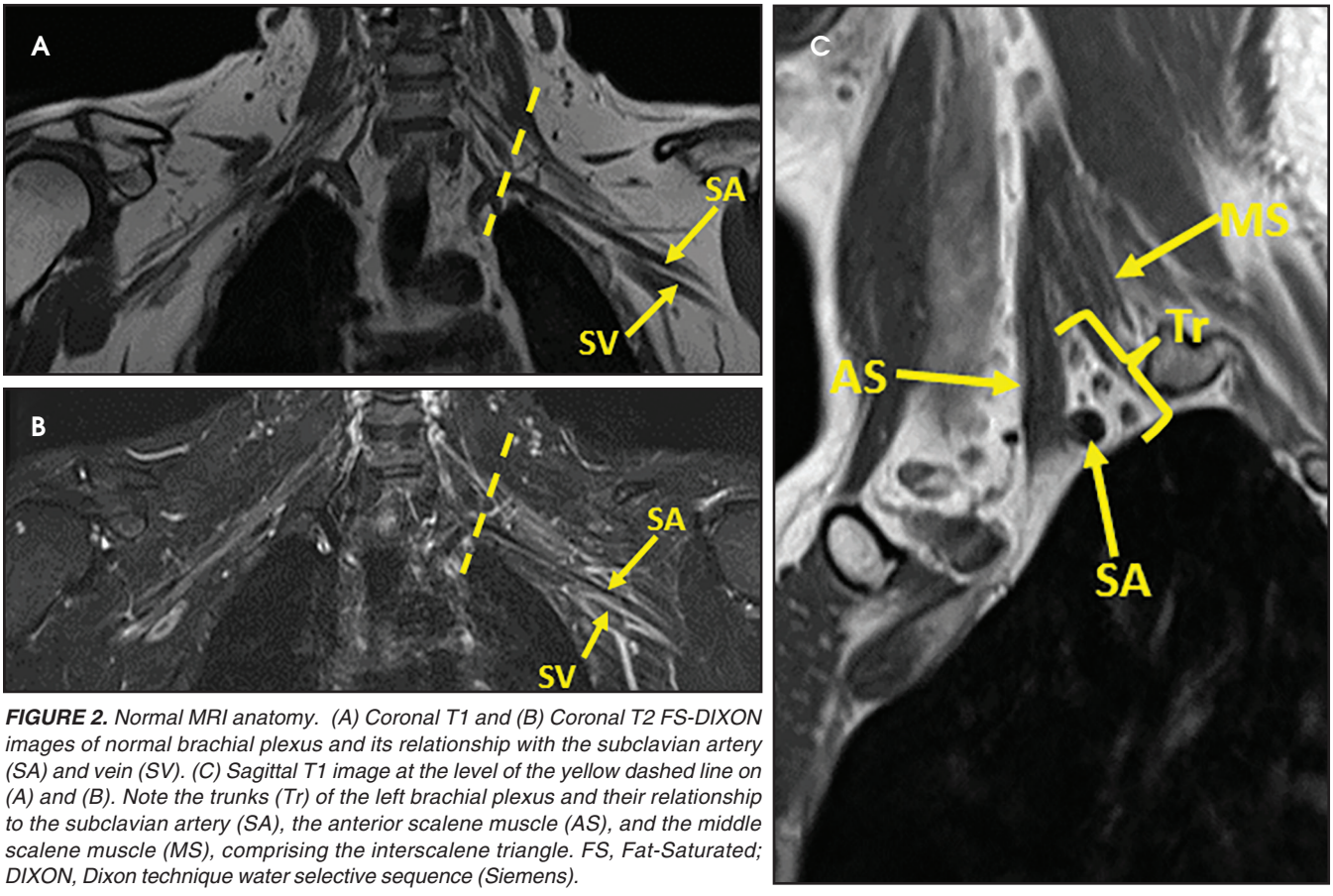

FIGURE 2. *Normal MRI anatomy. (A) Coronal T1 and (B) Coronal T2 FS-DIXON images of normal brachial plexus and its relationship with the subclavian artery (SA) and vein (SV). (C) Sagittal T1 image at the level of the yellow dashed line on (A) and (B). Note the trunks (Tr) of the left brachial plexus and their relationship to the subclavian artery (SA), the anterior scalene muscle (AS), and the middle scalene muscle (MS), comprising the interscalene triangle. FS, Fat-Saturated; DIXON, Dixon technique water selective sequence (Siemens).*

Table 1. 3T Imaging protocol of the brachial plexus

Practical Brachial Plexus MRI protocol

Sequence	TR (ms)	TE (ms)	FOV (cm)	ST (mm)	No. slices	NEX	Time (min:sec)
Cor T1 TSE	632	8.5	30	4	30	2	3:21
Cor T2 FSE	4500	95	32.5	4	30	2	2:35
Axial T1	707	8.9	30	4	25	2	5:09
Axial T2	4500	101	30	4	25	2	3:47
Sag T1 SE (each side)	647	8.5	30	4	40	2	4:38
Sag T2 FSE (affected side)	4500	95	30	4	40	2	3:24
Axial FS T1 post contrast	707	8.9	30	4	25	2	4:27
Sag T1 FS (affected side) post contrast	647	8.5	30	4	40	2	4:38

FSE = fast spin echo, FOV = field of view, TSE = turbo spin echo, SE = spin echo, FS = fat sat, TR = time to repeat, NEX = number of excitations, ST = slice thickness

Table 2. 1.5T brachial plexus imaging protocol

Practical Brachial Plexus 1.5 T MRI protocol (Siemens Aera)

Sequence	TR (ms)	TE (ms)	FOV (cm)	ST (mm)	No. slices	NEX	Time (min:sec)
Cor T1 TSE	590	7.3	30	3	30	3	3:30
Cor T2 – DIXON-W FSE	2410	85	34.5	3	51	2	1:34
Axial T1	590	7.3	30	3	50	3	3:30
Axial T2 – DIXON-W FSE	2410	85	34.5	3	51	2	1:34
Sag T1 SE (each side)	630	7.9	30	3	65	2	3:30
Sag T2 FSE DIXON-W FSE (affected side)	2400	85	30	3	65	1	3:00
Cor FS T1 post contrast DIXON-W	602	11	30	3	51	2	1:03
Sag FS T1 post contrast DIXON-W (affected side)	721	11	30	3	55	1	2:04
Sag T2 SPACE C-spine	1500	120	30	1	97	2	4:10

FSE = fast spin echo, FOV = field of view, TSE = turbo spin echo, SE = spin echo, FS = fat sat, TR = time to repeat, NEX = number of excitations, ST = slice thickness

tomography (CT).[4, 6, 7, 8] Additionally, scanning coverage should include the C4-T2 levels for pre-fixed (C4-C8) and post-fixed (C6-T2) BP anatomic variation, and extend out to the axilla to include the proximal terminal branches.

Preganglionic and postganglionic segments

The preganglionic segments include the cervical spinal cord root entry zone of the ventral and dorsal nerve rootlets, the intrathecal course of these rootlets, and their intraforaminal course up until the DRG. Three-dimensional T2-weighted techniques using steady state free procession (SSFP) can help visualize the intrathecal and intraforaminal segments with high contrast and spatial resolution.[9]

The normal orientation of the BP (medial-superior to lateral-inferior) makes traditional sagittal, axial and coronal images difficult to interpret, as the plane of imaging is not aligned with the course of the brachial plexus structures.[4] Consequently, oblique sagittal and coronal sequences are preferred techniques to capture the anatomy of the brachial plexus and its relationship to critical surrounding structures.[1, 4, 10] In the setting of unilateral injury, bilateral coronal T2 sequences provide effective comparison between the affected side and the normal contralateral side[2]. Smaller field of view, unilateral sagittal sequences are preferable to evaluate for edema, enlargement of the segments of the brachial plexus, or enhancement if contrast is administered.[1]

Gadolinium-based IV contrast is not required for routine BP evaluation. However, if infection, trauma or neoplasm is a primary consideration, contrast should be administered to delineate the extent of involvement and to assess for potentially drainable collections.[1,2]

Tables 1 and 2 include practical brachial plexus MR protocols on 3T and 1.5T scanners, with the final two sequences (post-contrast) considered optional based on indication. Additional sequences, such as heavily T2-weighted 3D imaging with steady state free procession (SSFP) can also be performed if visualization of intradural nerve roots / myelographic images are desired.[9] Of note, 1.5 Tesla (T) is the minimum required field strength for diagnostic quality images of the brachial plexus, with 3T being preferred secondary to improved signal to noise and contrast to noise ratios. Imaging accuracy, however, has been shown to be comparable between 1.5T and 3.0T systems[7]. Imaging can be performed with a surface coil such as a neurovascular array with multichannel phased array architecture or a wrap-around body surface coil in order to improve the signal to noise ratio.[4, 6]

Common presenting symptoms

Presenting symptoms referable to the BP can range from vague and nonspecific (eg, regional shoulder pain, upper extremity weakness, or altered upper extremity/shoulder sensorium) to symptoms with specific nerve distribution (eg, pain or motor/sensory deficit). More specific symptoms such as scapular winging (due to long thoracic nerve injury), diaphragmatic dysfunction (involvement of the phrenic nerve C3-C5), or Horner's syndrome (postganglionic C8-T1 involvement) can also be presenting complaints.[2]

Pathology categorization

Disease processes affecting the brachial plexus can be subdivided into the following broad categories: traumatic injury and several nontraumatic subtypes, including: infection, inflammatory brachial neuritis/neuropathy, benign or malignant neoplasms, radiation-induced plexopathy, vascular abnormalities, and compression of the plexus.

Traumatic injury

Perhaps the most significant utility of brachial plexus MR in the setting of trauma is to differentiate pre and post ganglionic injury, a distinction that has significant management implications.[8, 11, 12] Identification of a preganglionic injury can be difficult and often requires recognition of a combination of direct (eg, high resolution 3D MRI or CT myelography) and indirect imaging characteristics.

Direct signs

High-resolution 3D T2- and CT myelography images can show anatomical continuity or lack thereof of intradural nerve rootlets.[5, 6, 9, 12] The course of the rootlets should be followed from the root entry zone to the DRG in the neural foramen. In the setting of a structurally intact yet injured nerve root, post contrast sequences may show abnormal enhancement of the injured nerve root relative to the control side.[5, 9, 14]

Indirect signs

A traumatic pseudomeningocele may be present on T2-weighted images. The pseudomeningocele will usually show invagination into the affected neural foramen, with or without the detached nerve root within it (Figure 3). Contralateral deviation of the spinal cord is another indirect sign. Normally, the nerve rootlets anchor the spinal cord in the middle of the thecal sac, much like the taut ropes attached to large tents. Avulsion of nerve rootlets results in unopposed traction by the contralateral, intact nerve rootlets. Postganglionic traumatic injuries can demonstrate focal edema (hyperintense T2 signal) involving any part of the plexus distal to the DRG, anatomic discontinuity with or without clumping/retraction, or a periplexus hematoma.[1, 6, 9, 13]

Infection

Infection of the brachial plexus can arise from a variety of sources, but it usually spreads directly from an adjacent structure such as an extension of spinal osteomyelitis, empyema/pulmonary parenchymal infections, glenohumeral septic arthritis, overlying soft tissue infection, or iatrogenic introduction of pathogens.[2, 15, 16] Imaging characteristics of brachial plexus infections are similar to infectious processes elsewhere in that they result in T2 hyperintense edema, variable enhancement pattern (non-mass like), surrounding soft tissue inflammation and presence or absence of a demonstrable source collection.[4, 6, 13, 17, 18]

Inflammation

Spontaneous brachial plexitis, also known as Parsonage Turner Syndrome, typically presents with the constellation of spontaneous acute severe burning shoulder pain, subsequent sensory disturbance, and delayed weakness and atrophy.[19, 20] The inflammatory processes involving structures adjacent to the brachial plexus can also secondarily involve the brachial plexus. The imaging characteristics of brachial plexus inflammation are nonspecific but consistent with inflammatory conditions elsewhere in the body. Commonly encountered features include T2 hyperintensity, thickening and variable enhancement of brachial plexus components, as well as enlargement of the

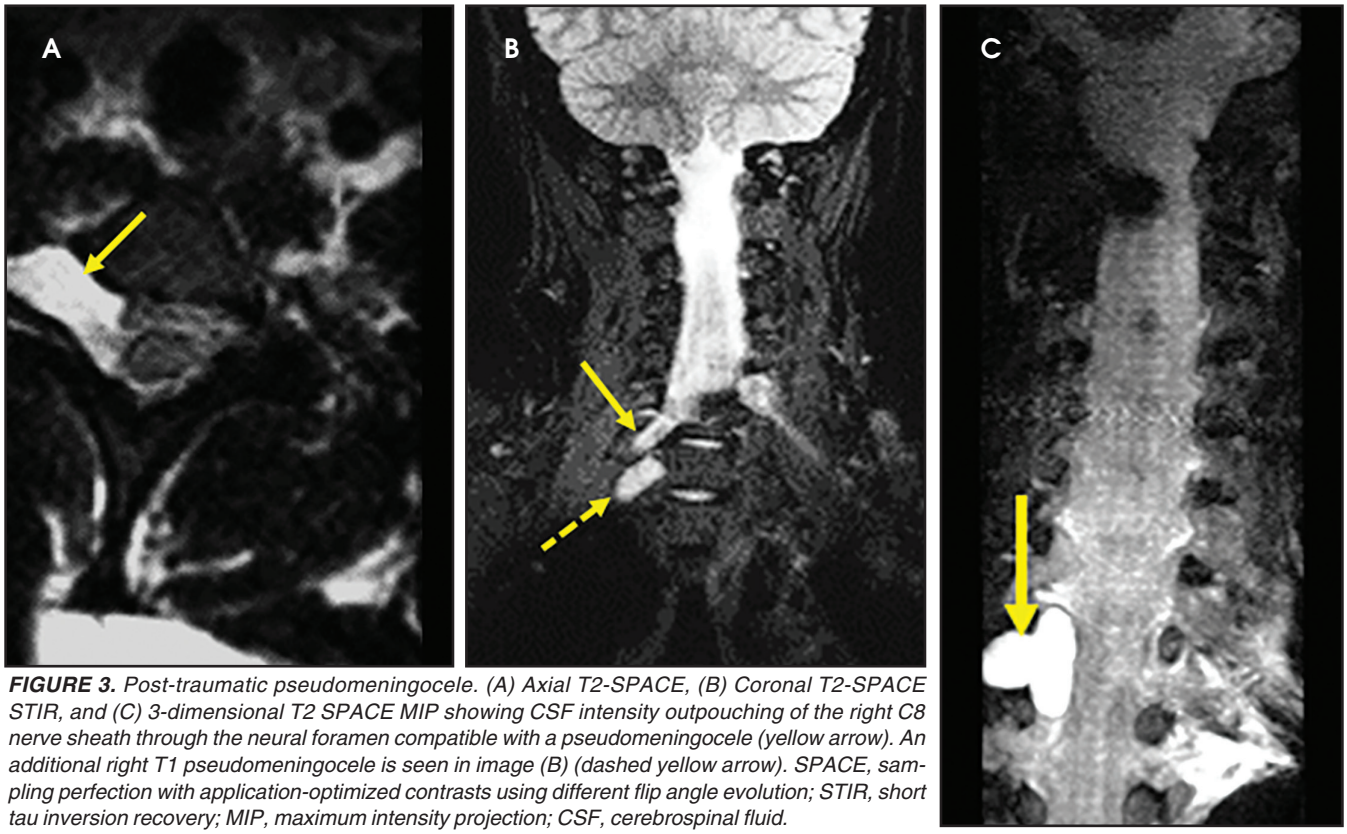

FIGURE 3. *Post-traumatic pseudomeningocele. (A) Axial T2-SPACE, (B) Coronal T2-SPACE STIR, and (C) 3-dimensional T2 SPACE MIP showing CSF intensity outpouching of the right C8 nerve sheath through the neural foramen compatible with a pseudomeningocele (yellow arrow). An additional right T1 pseudomeningocele is seen in image (B) (dashed yellow arrow). SPACE, sampling perfection with application-optimized contrasts using different flip angle evolution; STIR, short tau inversion recovery; MIP, maximum intensity projection; CSF, cerebrospinal fluid.*

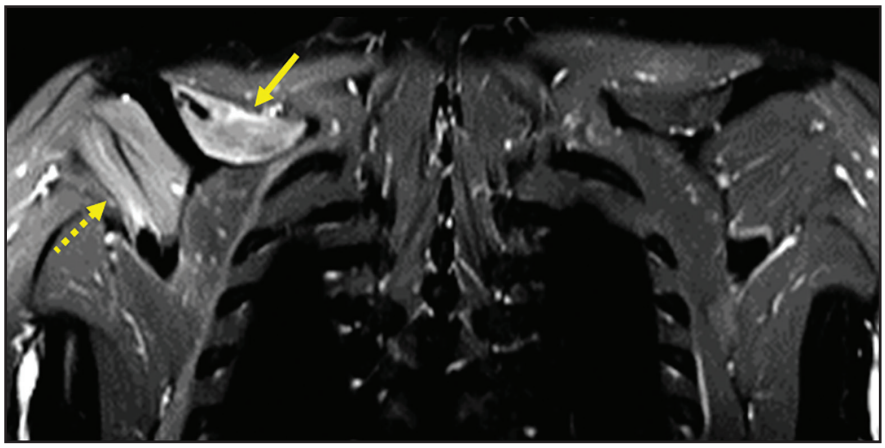

FIGURE 4. *Acute denervation muscle atrophy. Coronal T2 FS-DIXON image showing hyperintense T2 signal within the supraspinatus (solid yellow arrow) and the infraspinatus (dashed yellow arrow) in this patient with right C5 nerve impingement. FS, Fat-Saturated; DIXON, Dixon technique water selective sequence (Siemens).*

affected shoulder girdle muscle with enhancement and hyperintense T2 signal (signs of acute/subacute denervation) (Figure 4).[1,2,6,8,13]

Benign neoplasms

A variety of benign lesions can involve the brachial plexus. Specifically, these entities include fibromatosis, proliferative fasciitis, lipoma, hemangioma, brachial cleft cyst, lymphangioma, and benign neural and nerve sheath neoplasms (Figure 5).[2] Fibromatosis (extra-abdominal desmoid) (Figure 6) and proliferative fasciitis can both present as rather large masses involving the brachial plexus, with the former usually presenting as painless, and the latter as exquisitely tender.[21-24] Both of these lesions typically demonstrate T1 isointensity to surrounding muscle/soft tissue, heterogenous T2 hyperintensity, with mild enhancement in the case of proliferative fasciitis and avid enhancement in fibromatosis.[23,24]

Lesions such as lymphangiomas, lipomas, brachial cleft cysts, and hemangiomas occur rarely in the BP and demonstrate similar imaging characteristics as they would in other areas of the body.[25] Lipomas are usually easily discernible from nonfatty lesions, as they characteristically demonstrate T1 and T2 hyperintensity, with signal dropout on fat-saturated sequences. One potential pitfall is differentiating benign lipoma from low-grade liposarcoma. In the setting of large lesions (>10 cm), significant non-fatty components, numerous septae, or heterogenous enhancement, PET-CT can be performed for better characterization.[26,27]

Benign nerve sheath tumors have been described in a plethora of prior publications. Briefly, they are commonly observed as T2 hyperintense lesions in the neural foramen, sometimes resulting in expansion and osseous remodeling.[1,4,5,25]

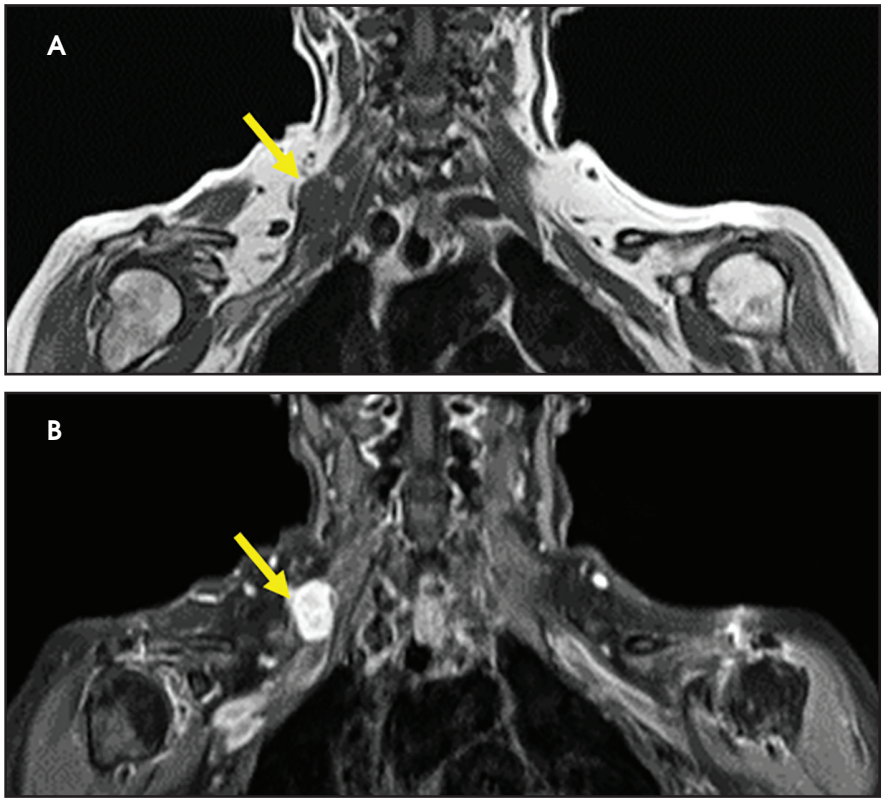

FIGURE 5. *Nerve sheath tumor. (A) Coronal T1 and (B) coronal T1 post-contrast FS-DIXON images show a well-circumscribed mass (yellow arrows) arising from the divisions of the right brachial plexus, a neurofibroma. FS, Fat-saturated; DIXON, Dixon technique water selective sequence (Siemens).*

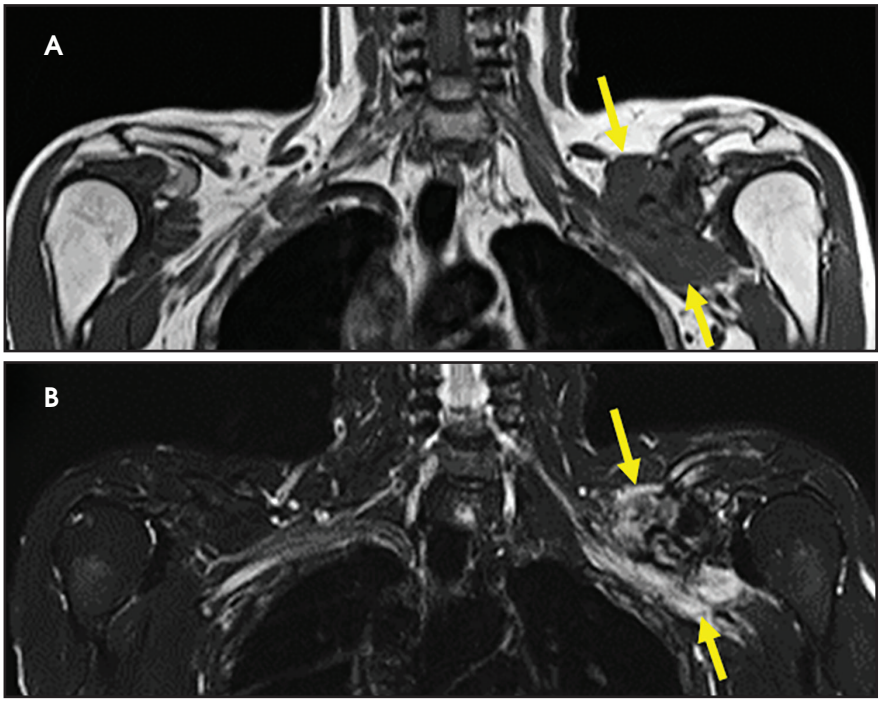

FIGURE 6. *Fibromatosis (extra-abdominal desmoid). (A) Coronal T1 and (B) coronal T2 FS-DIXON images show an infiltrating mass invading the left brachial plexus divisions and cords (yellow arrows). Biopsy revealed fibromatosis. FS, Fat-Saturated; DIXON, Dixon technique water selective sequence (Siemens).*

Malignant neoplasms

Malignant lesions can either primarily arise within the brachial plexus or spread to the brachial plexus secondarily. Primary malignant lesions involving the brachial plexus are predominately sarcomatous (low grade sarcoma, radiation induced sarcoma, osteosarcoma, Ewing Sarcoma, leiomyosarcoma, liposarcoma).[2, 21] These soft tissue masses often demonstrate overlapping imaging characteristics and can be difficult to differentiate based on imaging alone. Primary mesothelioma, malignant nerve sheath tumors and involvement from primary vertebral tumors such as chondrosarcoma or chordoma are also rarely seen involving the brachial plexus.[2, 21]

In regards to metastatic disease, the most common primary malignancies are breast, lung, lymphoma, and head/neck cancer.[2] Lung adenocarcinoma typically secondarily involves the brachial plexus via direct extension in the setting of a Pancoast tumor involving the superior sulcus (Figure 7). Breast carcinoma (Figure 8), lymphoma, and head/neck malignancies usually involve the brachial plexus via metastatic regional lymphatic spread [2, 25]. In the setting of primary or metastatic brachial plexus involvement it is important to determine presence or absence of leptomeningeal enhancement/spread, relation to the ipsilateral vertebral artery, and extent of nerve root involvement.

Radiation plexopathy

Radiation plexopathy typically manifests as T2 hypointense thickening of the brachial plexus components without focal mass (Figure 9).[1] In the setting of prior malignancy and local radiation to the brachial plexus, it is crucial for the clinician to attempt to differentiate tumor progression/recurrence from benign radiation-induced plexopathy changes. Time course is key to discerning these entities, as radiation induced plexopathy occurs between 5 and 30 months post radiation therapy (peak incidence 10-20 months post radiation).[4, 6, 8, 13, 25] Furthermore, details of clinical presentation can help aid in diagnosis.

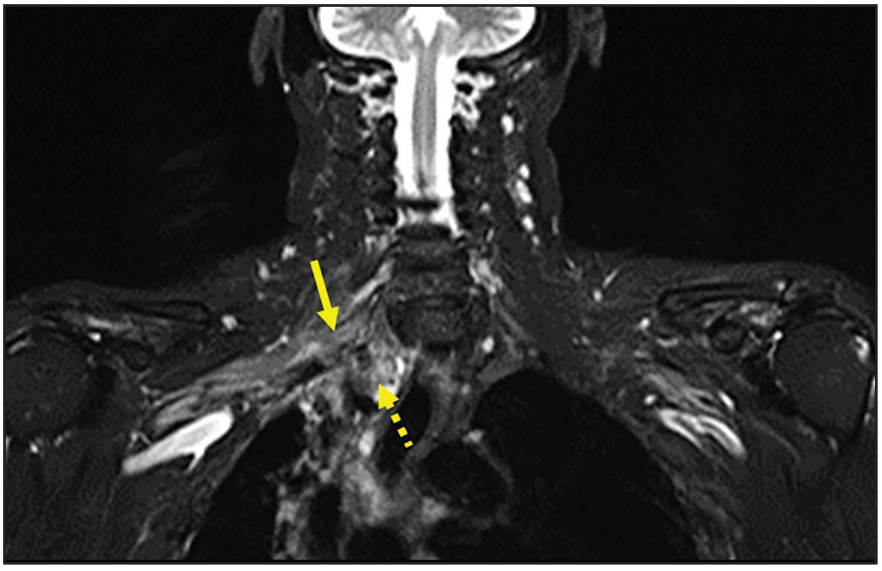

FIGURE 7. *Pancoast (superior sulcus) tumor. Coronal T2 FS-DIXON image shows direct extension of a right lung apex mass (Pancoast tumor, dashed yellow arrow) into the lower trunks and divisions of the right brachial plexus (solid yellow arrow). FS, Fat-Saturated; DIXON, Dixon technique water selective sequence (Siemens).*

For example, increasing/new pain or new Horner syndrome are more likely to reflect tumor recurrence/progression; while unilateral edema or parasthesia is more likely to reflect radiation-induced plexopathy.[1]

Compression of the brachial plexus

The neurovascular bundle can be compressed at several areas along the brachial plexus (Figure 10), resulting in a clinical constellation of symptoms commonly referred to as thoracic outlet syndrome. Particularly, the brachial plexus components can be affected at the interscalene triangle, costoclavicular space, or less commonly, the pectoralis minor space. Clinically, this syndrome can result in ulnar distribution hand weakness, hand/arm/neck pain/parasthesias,

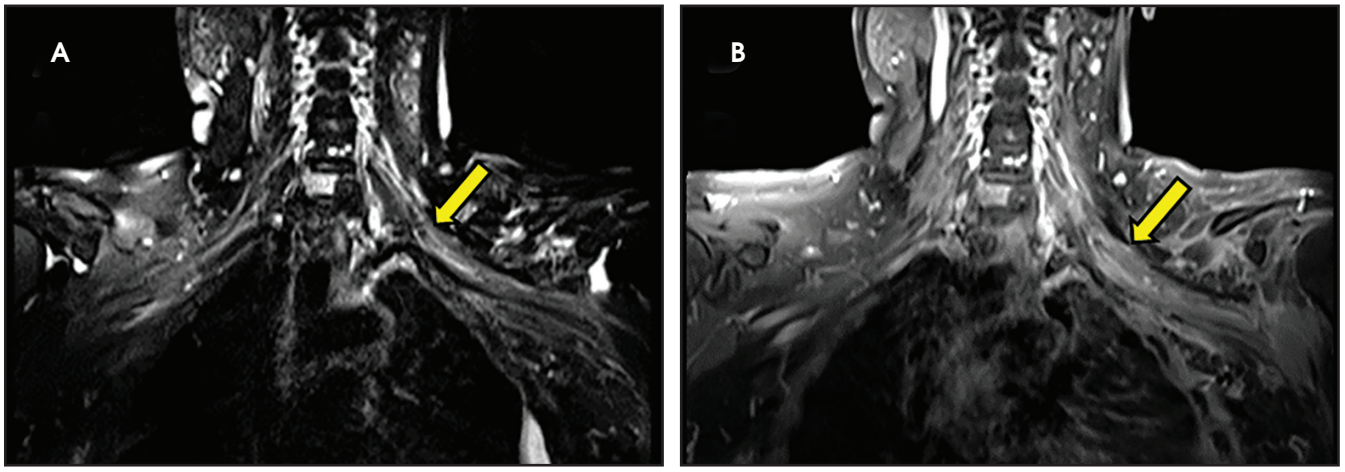

FIGURE 8. *Breast cancer metastasis. (A) Coronal T2 FS-DIXON and (B) coronal T2 FS-DIXON images show abnormal T2 signal and enhancement of the divisions and cords of the left brachial plexus (yellow arrows) in this patient with metastatic breast cancer. FS, Fat-Saturated; DIXON, Dixon technique water selective sequence (Siemens).*

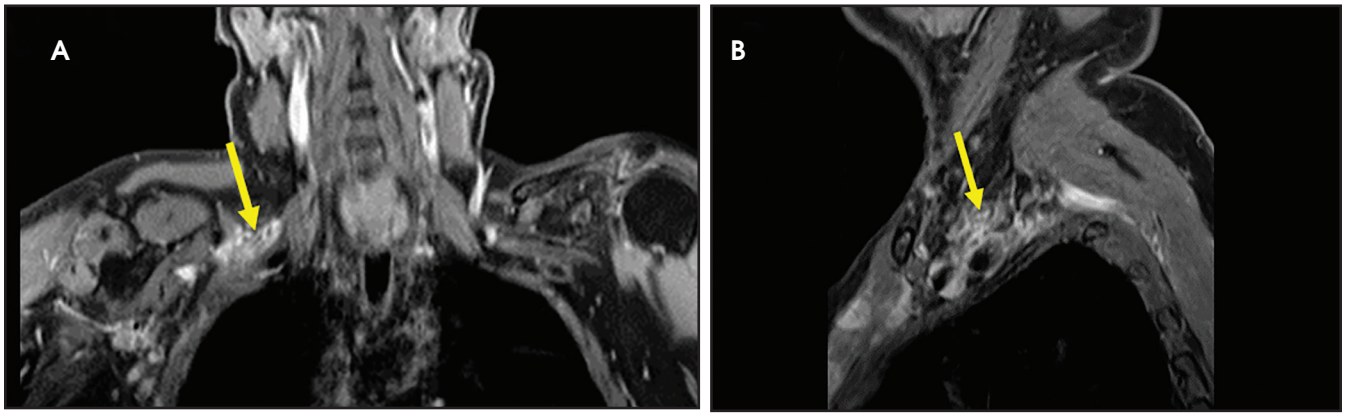

FIGURE 9. *Radiation plexitis. (A) Coronal and (B) sagittal T1 post-contrast FS-DIXON images show ill-defined enhancement of the divisions of the right brachial plexus (yellow arrows) in this breast cancer patient who underwent extensive locoregional radiation therapy. Biopsy confirmed lack of tumor or infection. FS, Fat-Saturated; DIXON, Dixon technique water selective sequence (Siemens).*

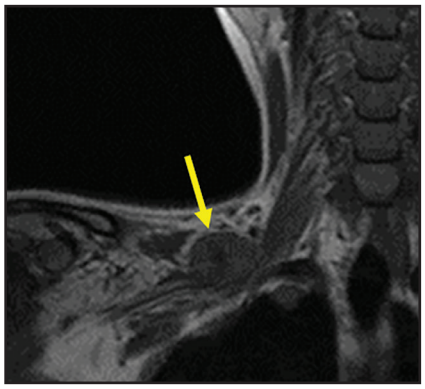

FIGURE 10. *Compression brachial plexopathy. Coronal T1 image of the right brachial plexus shows a soft tissue mass (metastatic lymph node, yellow arrow) compressing the divisions in this patient with renal cell carcinoma presenting with arm numbness and weakness upon raising his right arm (thoracic outlet syndrome symptoms. Removal of the metastatic lymph nodes caused cessation of the symptoms.*

and upper extremity muscle atrophy.[22] Symptomatology is often exacerbated/reproducible by arm raise. The syndrome is typically caused by anatomic variants such as a cervical rib, prominent lower cervical transverse processes, posttraumatic fibrous bands, or pectoralis muscle hypertrophy.[28] MRI can be used to identify any of the aforementioned causative factors, and should include provocative testing in order to reproduce symptomatology during the time of the scan.[2, 22]

Vascular abnormalities

A variety of space-occupying vascular abnormalities can result in brachial plexus compression, including but not limited to pseudoaneurysm, arteriovenous fistula (AVF), or arteriovenous malformation (AVM).[2] The involved vessels include the subclavian, axillary, common carotid, and vertebral arteries. A variety of predisposing conditions can result in these lesions, which are best characterized on dedicated vascular studies such as CT angiography, MR angiography, and/or conventional angiography.

Conclusion

The brachial plexus can be efficiently imaged and effectively interpreted by the general radiologist when approached from a practical standpoint. Optimization of a practical BP imaging protocol is paramount to identify normal anatomy and associated pathology. Practical and useful information that can help the referring physician include, pre- vs. postganglionic location of lesion, mass vs. non-mass like enhancement, laterality or bilateral nature of disease, location of injury/mass/abnormality in BP segments (eg root, trunk, division, etc.), and anatomical region and surrounding structures involved (eg, interscalene space, costoclavicular triangle, relationship to subclavian/axillary vessels).

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