Imaging Diagnosis of Nerve Entrapments in the Shoulder

14

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14.1 Introduction

Nerve injuries are an unusual source of shoulder pain. They can result from several causes including trauma, neoplasia, infection, neuropathy, autoimmune disease, and iatrogenic conditions [1]. Neuropathies of the shoulder are frequently considered entrapment syndromes and account for about 2% of cases of shoulder pain [2]. However, this frequency is likely to be underestimated because these conditions have been overlooked in the past [3]. As a result of increased familiarity regarding these conditions, they are diagnosed with growing frequency in patients with symptoms suggestive of nerve pathology.

The most commonly affected nerves in the shoulder region are the suprascapular nerve, axillary nerve, long thoracic nerve, cervical accessory nerve, and dorsal scapular nerve. These nerves may be involved in patients engaging in vigorous overhead activity, with massive rotator cuff tears, tears accompanied by fatty infiltration and/or atrophy of muscle, labral tear and paralabral cyst formation, and space-occupying lesions [3]. Muscle weakness with or without associated sensory deficit, sharp burning pain, and paresthesias over a localized skin area are a few signs and symptoms of entrapment neuropathies. Most of these cases are related to physical circumstances leading to a nerve being stretched or compressed into a fibrous or osteofibrous space. Particularly in neuropathy of the suprascapular nerve, the clinical diagnosis is often delayed because of nonspecific symptoms [4–6].

In this chapter, we review anatomic structures and landmarks of the most important nerves around the shoulder. We also discuss the pathologic conditions causing entrapment neuropathies such as compression, stretching, or iatrogenic lesions and outline different diagnostic imaging modalities. Finally, we review specific shoulder neuropathies and explain their MRI characteristics.

14.2 Pertinent Anatomy

14.2.1 Suprascapular Nerve

The suprascapular nerve originates from the upper trunk of the brachial plexus, with contributions from C5, C6, and sporadically C4 nerve root (Fig. 14.1). It is responsible for motor innervation of two rotator cuff muscles: the supraspinatus and infraspinatus. The nerve travels posterior to the clavicle and obliquely traverses towards the superior border of the scapula

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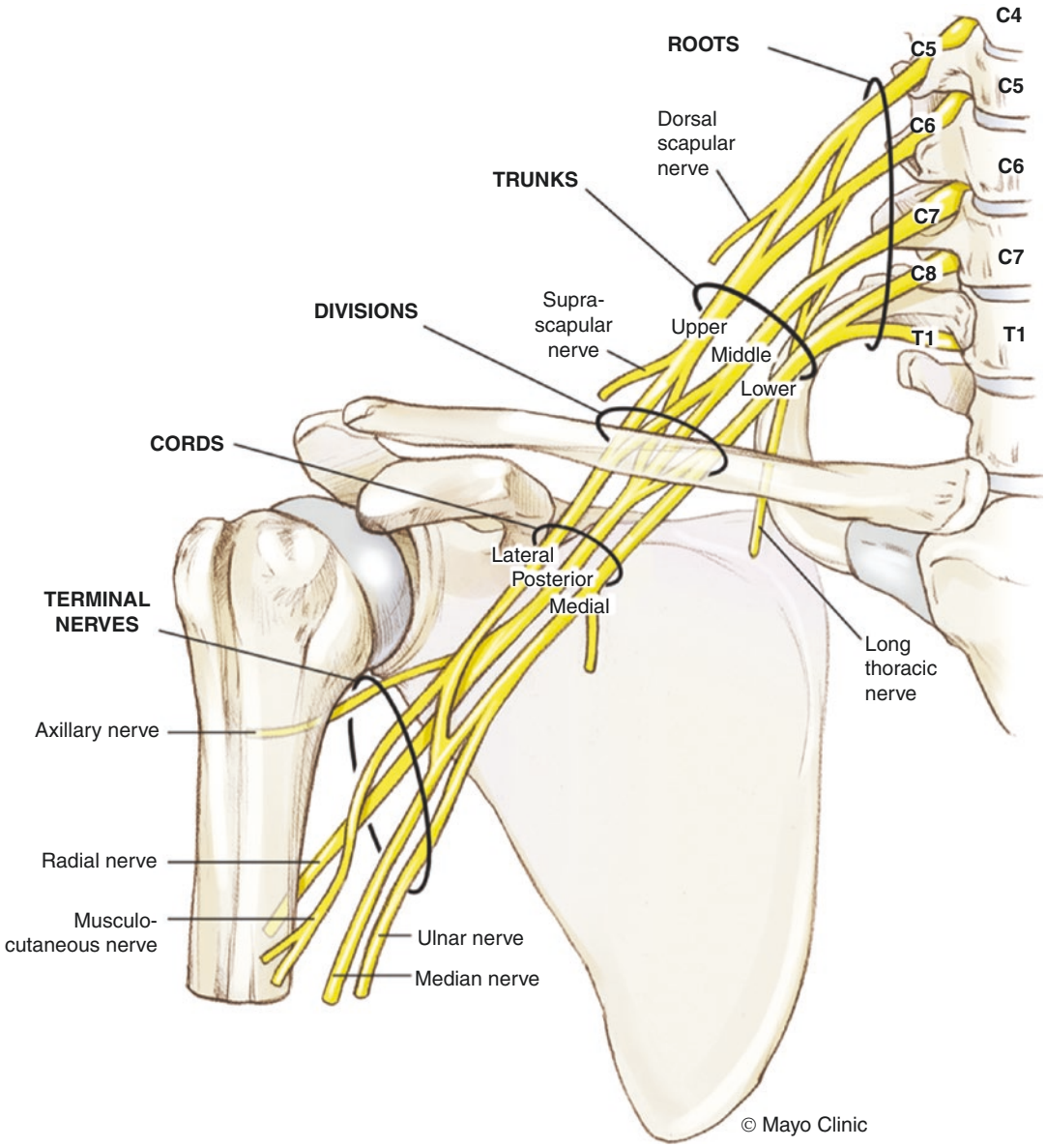

Fig. 14.1 Schematic representation of the anatomy of brachial plexus and its branches. Used with permission of Mayo Foundation for Medical Education and Research. All rights reserved

(Fig. 14.2). The nerve then passes through the suprascapular notch in an anterior-to-posterior direction. Generally, the nerve travels beneath the transverse scapular ligament in the suprascapular notch, while its associated artery passes over the ligament. Branches to the supraspinatus muscle emanate posterior to the suprascapular notch. The nerve continues towards the spinoglenoid notch of the scapula, where it travels beneath the spinoglenoid ligament (inferior transverse ligament) to provide branches to the infraspinatus muscle.

The presence of these two anatomical notches and the awareness of local muscle innervation patterns can assist the physician in diagnosing specific conditions. The role of these notches in entrapment neuropathies was first suggested by Aiello et al. [7] who discriminated between

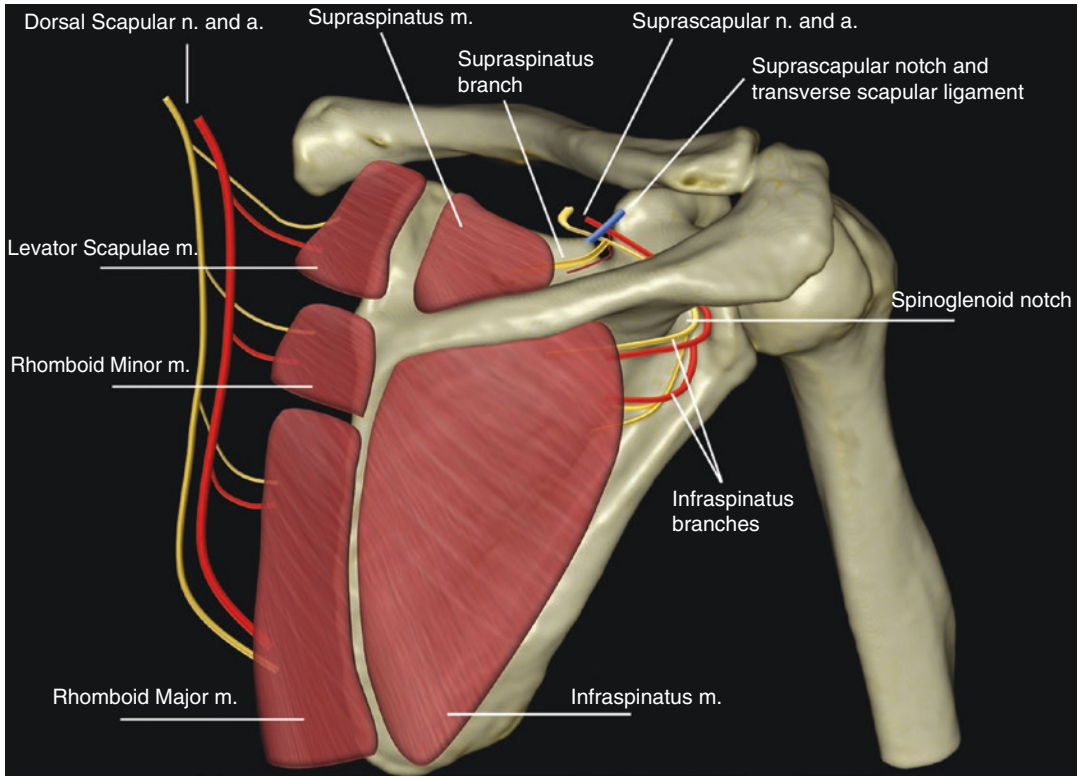

Fig. 14.2 Schematic representation of shoulder nerves and branches. Note the relationship between the suprascapular nerve with the suprascapular and spinoglenoid notches

entrapment of the nerve at the suprascapular notch and entrapment at the spinoglenoid notch. Although the suprascapular nerve has been considered a pure motor nerve, cadaveric studies have indicated sensory branches to the glenohumeral joint, acromioclavicular joint, coracoacromial ligament, and skin [8, 9]. Improved anatomic recognition of the sensory contributions of the suprascapular nerve helped to clarify the associated pain resulting from injury or traction of this nerve. Up to 70% of the sensation of the shoulder may be provided by the suprascapular nerve [10], and studies have shown improved postoperative pain after suprascapular nerve block in patients who had shoulder surgery [11, 12].

The suprascapular nerve and its concomitant vessels, well delineated by fat, are originally best identified on oblique coronal T1-weighted MRI showing the suprascapular notch, at the junction of the glenoid with the scapular neck, just medial to the superior glenoid rim. As the suprascapular neurovascular bundle arrives in the spinoglenoid notch, it can be seen on axial MRI images. A noticeable suprascapular vein, which occasionally is responsible for compressive neuropathy, is in some instances noted in the proximity of the nerve.

14.2.2 Axillary Nerve

The axillary nerve is a final branch of the posterior cord of the brachial plexus and originates from the ventral rami of C5 and C6 (Fig. 14.1). The first portion of the axillary nerve is located lateral to the radial nerve, posterior to the axillary artery, and anterior to the subscapularis muscle. It continues in an oblique direction across the inferolateral border of the subscapularis. It then travels through the quadrilateral space associated with the posterior humeral circumflex artery (Fig. 14.3). The quadri-

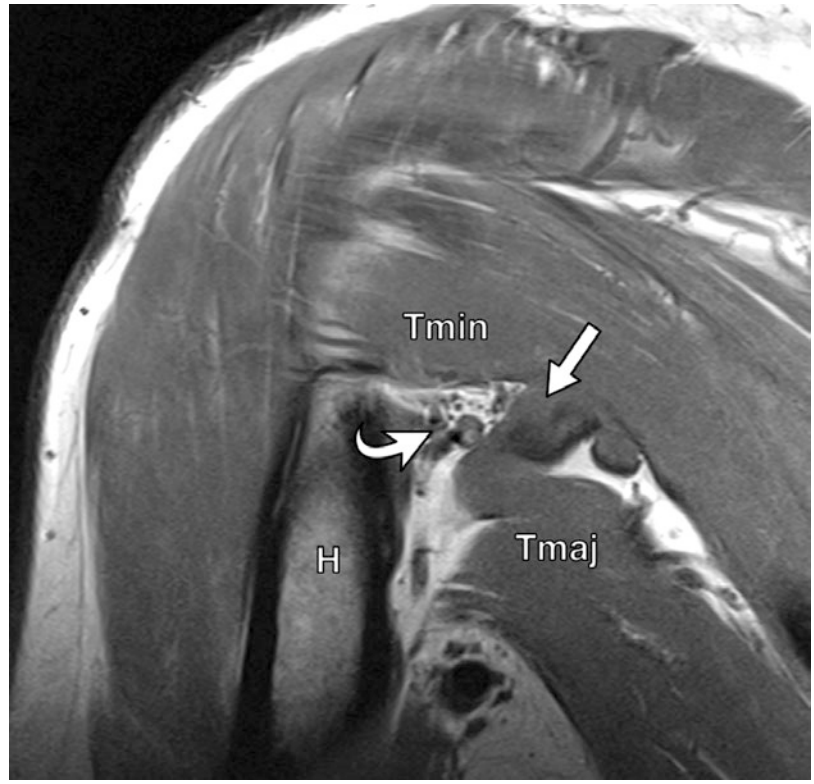

Fig. 14.3 Coronal oblique T1-weighted image of the right shoulder showing the teres major (Tmaj), teres minor (Tmin), triceps (arrow), and humerus (H) delimiting the quadrilateral space (curved arrow), where the posterior circumflex vessels and axillary nerve are identified

lateral space is a rectangular cuboid, located inferoposterior to the glenohumeral joint, with mean dimensions of about 2.5 × 1.5 cm [13, 14]. It is bordered superiorly by the teres minor muscle, inferiorly by the teres major muscle, medially by the long head of the triceps, and laterally by the surgical neck of the humerus [15]. The axillary nerve is the most superior structure in this space [13], where the nerve divides into its anterior (superior) and posterior (inferior) branches [14]. The anterior branch takes a circuitous route around the surgical neck of the humerus and provides branches for the anterior and middle parts of the deltoid muscle. The posterior branch supplies the subscapular muscle, the teres minor muscle, and often the posterior portion of the deltoid, as well as sensory innervation of the posterolateral shoulder.

The axillary neurovascular bundle is best identified on oblique sagittal T1-weighted MRI of the shoulder. It is well highlighted by surrounding fat and is seen below the inferior glenoid rim passing through the space between the teres minor and the teres major muscles. The quadrilateral space is best depicted next to the medial humeral cortex and lateral to the long head of the triceps muscle on oblique coronal images oriented along the humeral shaft.

14.2.3 Long Thoracic Nerve

The long thoracic nerve is a pure motor nerve particularly responsible for innervation of the serratus anterior muscle. Anatomically, it originates from C5 to C7 and occasionally C8 (8%) (Fig. 14.1) [16]. After advancing anteriorly to the posterior scalene muscle, it travels distally and laterally to pass below the clavicle and under the first and the second ribs. Distally, the nerve descends along the chest wall in the midaxillary line to the outer border of the serratus anterior, sending branches to each of the digitations of this muscle (Fig. 14.4) [16]. The serratus anterior muscle originates from the costomedial border of the scapula and inserts on the first through ninth ribs, forming the medial wall of the axilla and functioning as a scapular protractor.

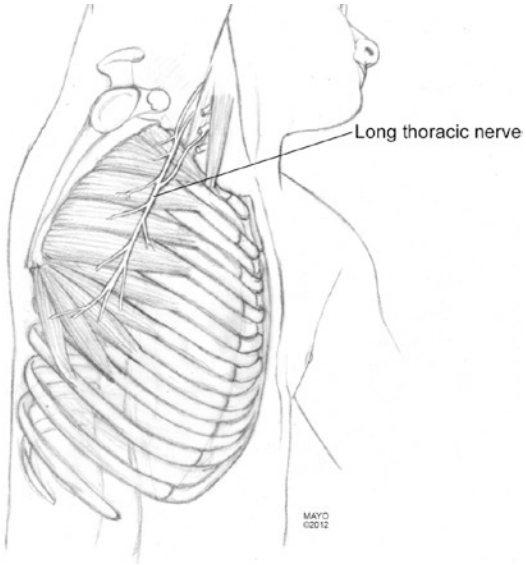

Fig. 14.4 Topographical anatomy of the long thoracic nerve, overlying the serratus anterior muscle. Used with permission of Mayo Foundation for Medical Education and Research. All rights reserved

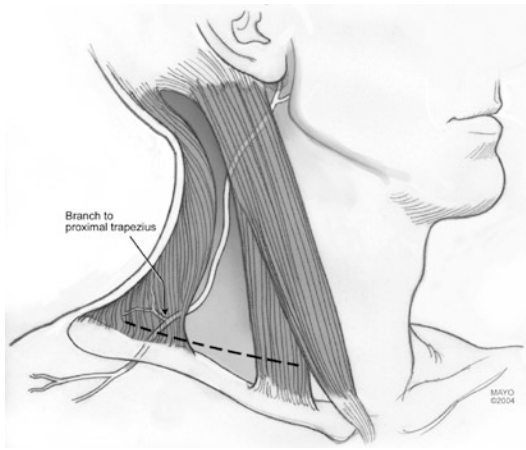

Fig. 14.5 Topographical anatomy of the accessory nerve as it travels along the posterolateral neck. Used with permission of Mayo Foundation for Medical Education and Research. All rights reserved

14.2.4 Spinal Accessory Nerve

The spinal accessory nerve is a cranial nerve (CN XI) originating from the upper segments of the spinal cord, ultimately supplying motor fibers to the sternocleidomastoid (SCM) and trapezius muscles (Fig. 14.5), being included in the gamut of shoulder nerves [17]. It is a pure motor nerve originating from C1 to C4 cervical segments and ascends through the foramen magnum, and returns through the jugular foramen. After providing motor innervation to SCM muscle, it enters the posterior triangle of the neck and eventually innervates the trapezius muscle. Additionally, the nerve proceeds caudally and dorsally in the subcutaneous tissue along the posterior triangle of the neck.

14.2.5 Dorsal Scapular Nerve

The dorsal scapular nerve is a motor nerve that arises from the C5 nerve root, with usual contribution from C6 (Fig. 14.1). After its origin, it runs in close proximity of the upper trunk of the brachial plexus, then traversing the middle scalene muscle and traveling posteriorly to innervate the levator scapula muscle. The nerve also reaches the deep surfaces of rhomboid major and minor, supplying both muscles that have a role in moving the scapula medially (Fig. 14.2) [13].

14.3 Pathologic Conditions

14.3.1 Definition

The term entrapment neuropathy refers to an isolated peripheral nerve injury at a specific location where a mechanical constriction occurs, most commonly from a fibrous or fibro-osseous tunnel, or by a fibrous band. However, there are cases in which the nerve is injured by chronic direct compression by space-occupying lesions (such as a cyst), or by chronic deformation, angulation, or stretching forces causing mechanical damage. Finally, iatrogenic causes can result in the nerve injury, including direct nerve injury or by means of deformation induced by postsurgical scarring.

14.3.1.1 Nerve Compression

Entrapment neuropathies of the shoulder secondary to nerve compression by mechanical or dynamic forces could be a cause of upper extremity weakness and pain in the athlete. Structurally narrow tunnels make individual

nerves susceptible to entrapment neuropathies. Dynamic changes within these narrow passages during repetitive athletic activity can create further compression of a nerve with only minimal anatomic variation [18]. Nerve compression may also be produced by space-occupying lesions such as cysts, tumors, and inflammatory processes or by posttraumatic conditions such as hematoma, myositis ossificans, and scar formation. Direct compression occurs mainly because a space-occupying lesion evolves in the proximity of the nerve, which can also develop if local anatomy has been altered by a fracture. Other causes for nerve compression are associated with hormonal alterations and systemic diseases, such as pregnancy, oral contraceptive ingestion, diabetes mellitus, and hypothyroidism.

14.3.1.2 Nerve Stretching

Along the trajectory of nerves, there are fixed points limiting its mobility potentially predisposing to focal deformation that exceeds its mechanical properties. Specific repetitive movements can create excess nerve traction, causing strain injuries at these fixed points, evolving with inflammation that impairs normal nerve conduction. Additional factors, such as fibrous bands or small arterial branches crossing a nerve, can create additional fixed points and also be the basis for entrapments.

14.3.1.3 Iatrogenic Lesions

Iatrogenic lesions from surgery can also be a source for shoulder neuropathies. For example, the spinal accessory nerve may be injured after biopsies of a cervical lymph node. Neurologic symptoms are present after approximately 0.2–3% of shoulder arthroscopic procedures, 4% of arthroplasties, and 8% of open surgeries for the treatment of instability [19–21]. The majority of these injuries are minor cutaneous nerve lesions and transient neurapraxias [22–25]. Permanent sequelae and injuries that require secondary surgical intervention are rare, and the long-term outcomes of patients with nerve injury from shoulder surgery have been rarely reported. These situations are less diagnostically challenging.

14.3.2 Radiographs and Computed Tomography

The decision on the timing and modality of shoulder imaging is based on multiple factors, including the acuity of the injury, the suspected tissue and nerve involved, the age of the patient, and demands that the patient applies on the shoulder. In general, acute traumatic injuries are imaged with plain films to exclude fracture or dislocation. For several bone and joint problems, radiographs are often the only required imaging study, being noninvasive and rather inexpensive compared to other imaging studies. They should be obtained to rule out likely osseous causes of nerve entrapment and to evaluate concomitant shoulder conditions, such as glenohumeral subluxation or osteoarthritis. Several views of the shoulder are necessary for comprehensive visualization of the osseous and articular anatomy. A Stryker notch view allows evaluation of the suprascapular notch and may display complete ossification or near obliteration of its foramen. A suprascapular notch view (X-ray beam directed 15–30° cephalad) allows visualization of osseous notch variants. Anteroposterior radiographs directly show superior migration of humeral head suggestive of chronic rotator cuff disease. Appropriate evaluation of conventional radiographs often encourages selection of higher level imaging studies. In situations in which osseous abnormalities are speculated to be the primary purpose of nerve injury, a computed tomography (CT) scan may be beneficial in recognizing specific regions of likely nerve compression [26]. Radiographs are not well suited for suspected nerve injuries of insidious onset given their preferential involvement of soft tissues.

CT scans allows for excellent detail regarding osseous pathology in addition to reliable identification of muscular fatty degeneration and atrophy. Although CT has limitations in directly assessing shoulder nerves, this method provides excellent visualization of suprascapular and spinoglenoid notches. In addition, CT arthrography can help diagnose paralabral cysts, in

particular those communicating with the glenohumeral joint, as well as identify rotator cuff tears as a potential source of shoulder pain. A low-dose CT scan can concurrently analyze the position of the scapula and the attached muscles and look for signs of denervation (fatty degeneration, etc.) and dynamic 3-dimensional (4D) reconstructions obtained with a large detector CT scanner can demonstrate scapular kinematics in suspected scapular winging [2, 27].

14.3.3 Ultrasound

Ultrasound is an excellent imaging modality to assess muscle, ligament, and tendon anatomy, with the unique advantage of allowing for dynamic imaging studies. In the shoulder, ultrasound has consistently demonstrated high levels of accuracy to detect rotator cuff pathology. However, this modality still has a limited role in comprehensively assessing the shoulder for entrapment neuropathies, given the difficulty in reliably demonstrating variable levels of muscle edema and directly showing the challenging anatomy of shoulder nerves throughout their entire course. Nevertheless, shoulder ultrasound can be used as a first intention modality for shoulder pain to rule out confounding symptoms. Although the initially suspected diagnosis is often a rotator cuff tendon tear, the radiologist must not ignore differential diagnoses that could relate to a neuropathy, particularly when no cuff tendon abnormality has been identified. For this reason, beyond the standard assessment for rotator cuff and biceps tendons, it is suggested to systematically include the following items in a shoulder ultrasound examination [28]:

A- Suprascapular and spinoglenoid notches to assess for a space-occupying lesion. A paralabral cyst can be readily differentiated from dilated suprascapular veins by combining color Doppler and compression of the structure; veins are filled with colored Doppler flow signal and collapse under pressure, whereas cysts do not.

B- Supraspinatus and infraspinatus muscles compared to contralateral side. Muscle atrophy can be readily determined with asymmetry of muscle volume, along with fatty degeneration that causes increased echogenicity. This can be done rapidly at the end of ultrasound examination while evaluating the posterior aspect of the glenohumeral joint.

Although this methodology only relates to suprascapular neuropathy, it can be valuable since this nerve can account for up to 97% of shoulder entrapment neuropathies. A direct search for specific nerves can be performed as needed but access can be limited by the complex nerve trajectory, depth, and overlapping osseous anatomy [29].

14.3.4 Magnetic Resonance Imaging

Besides providing direct visualization of a nerve and surrounding tissues, MRI is able to illustrate intrinsic signal abnormalities within the nerve and is considered superior in defining the associated indirect signs from muscle denervation [30, 31]. The signal intensity of a normal nerve on MRI is intermediate to low on T1-weighted sequences being slightly higher on T2-weighted and other fluid-sensitive sequences [31, 32]. Enlargement with obvious increase in T2 signal is regarded as an abnormal MRI appearance [32]. Additionally, a hyperintense signal of the denervated muscle is commonly seen when entrapment is acute, and fatty infiltration and muscle atrophy are signs of long-standing neuropathy in chronic cases [30–32]. Recognizing muscular denervation (muscular edema, atrophy, and fatty degeneration) in a neural distribution pattern is essential to the diagnosis of entrapment neuropathies. MRI is the only imaging modality that can reliably detect muscular edema, which is the earliest abnormality to appear in entrapment neuropathies. MRI also allows an assessment of the severity of the disease and the search for its etiology, being valuable in excluding differential diagnoses. The

MRI protocol should be adjusted to each situation: in case of suspected suprascapular or axillary neuropathy, the field of view (FOV) has to be centered on the glenohumeral joint, while in case of scapular winging the FOV needs to be enlarged to cover both scapulae and their attaching posterior and medial muscles.

14.3.4.1 Building an MRI Protocol

The most important pulse sequence is fast spin-echo T2-weighted imaging with fat suppression, carried out in the axial plane. This imaging allows the recognition of muscular edema, using echo times ≥45 ms for sufficient T2 weighting [2]. T1-weighted sequences without fat suppression are also important for the diagnosis of muscle fatty degeneration. Acquisition in the sagittal plane may be supplementary to grant better comparisons between the different muscles, particularly if the signal abnormalities are slight. It is reasonable to increase the gap between the slices to cover the total volume of the muscles, given that shoulder muscle may be partially involved with edema or chronic atrophy, depending on the site of entrapment. Typically, intravenous injection of gadolinium injection is not required for the assessment of shoulder neuropathies. However, denervated muscles frequently show contrast enhancement, making such images comparable to those obtained with T2-weighted fat-suppressed pulse sequences [1, 27].

The edema observed in entrapment neuropathies has many specific features [2]: it involves only the denervated muscles, being homogeneous in intensity and affecting the muscle diffusely. Muscle edema from neuropathies is an early phenomenon. Clinical studies report it to be detectable in the second week after trauma or at the onset of electromyography (EMG) abnormalities, but in experimental studies it manifests earlier, up to a few days after trauma [33]. Neuropathic muscle edema is also isolated because of the lack of other intramuscular abnormalities. The intensity of edema relies upon the severity and duration of the primary lesion. Experimental studies show that muscle edema peaks 2–4 weeks after the primary trauma but its intensity increases with the severity of lesions [33]. In sports-related shoulder neuropathy, this anomaly can be seen beyond 6 months [5].

Denervated muscle atrophy typically progresses slower than edema. Atrophy is best shown on sagittal T1-weighted images due to the optimal contrast between muscle and surrounding fat. The sagittal plane grants a comparison among scapular muscles, which are visualized along their short axes, allowing for an estimation of their bulk. The degree of atrophy can be assessed by examining the muscle surfaces, which are normally convex but turn flat and ultimately concave in the later stages [5]. Atrophic pseudohypertrophy has been described in which the afflicted muscle paradoxically enlarges in response to denervation; however it is diffusely infiltrated by adipose tissue [1]. However, unlike true muscle hypertrophy in which the signal is normal on all MRI sequences, the pseudohypertrophied muscle loses its normal signal intensity because of edema and fatty replacement. Fatty degeneration initiates during the subacute phase and is a hallmark of chronic muscle denervation [30]. Rotator cuff muscle fatty degeneration can be measured on sagittal T1-weighted images using the Bernageau and Goutallier classification method [34].

14.3.5 Specific Shoulder Neuropathies and Related MRI Features

14.3.5.1 Suprascapular Nerve Neuropathies

Suprascapular Nerve Syndrome

The suprascapular nerve can be injured by several mechanisms. These include repetitive overhead sports or activities and associated nerve traction [35–38], compression from a space-occupying lesion such as a cyst [39–41], trauma

[42–45], and rotator cuff tears [46]. Athletes who perform repetitive overhead activities such as tennis and volleyball have been described to experience neuropathy secondary to traction and microtrauma [35, 47, 48]. The mechanism is increased pressure on the nerve by the spinoglenoid ligament when the shoulder is in a position of overhead throwing [49]. Traction injury can also happen in the presence of a retracted superior or posterior rotator cuff tear. The secondary traction of the nerve is created at the suprascapular notch or around the base of the scapular spine. Repetitive adduction and internal rotation of the shoulder can stretch the nerve below the spinoglenoid ligament [50]. Ossification of the transverse scapular ligament or spinoglenoid ligament at the suprascapular or spinoglenoid notch, respectively, may increase the risk of suprascapular neuropathy [36, 51–54]. This may create stretching and compression of the suprascapular nerve and its branches below the suprascapular ligament. Nerve compression may also happen at either the suprascapular or the spinoglenoid notch by soft-tissue or bone tumor, or a cyst secondary to a labral or capsular injury [39–41].

The association between labral tears and paralabral cysts causing suprascapular neuropathy is well determined in both radiographic and clinical investigations [55–59].

Paralabral cysts are usually associated with tears of the superior and posterior glenoid labrum (from 8- to 11-o'clock positions), related to the passage of joint fluid into the cyst through a thin pedicle [60, 61]. During their growth, paralabral cysts may spread into the spinoglenoid notch, the suprascapular notch, or both, possibly causing nerve entrapment and muscle denervation. Ultrasound and MRI can identify the cyst and recognize secondary changes of nerve damage, including loss in bulk and echotextural or signal intensity changes in the innervated muscles due to edema and fatty replacement [62]. A direct correlation has been found between the size of paralabral cysts and the onset of denervation symptoms [60]. Due to their deep location, depiction of paralabral cysts requires a careful scanning technique with ultrasound [63]. Varicose veins in the spinoglenoid notch are the main differential diagnosis. Although enlarged spinoglenoid notch veins look like a cyst because they appear as fluid-filled images, they change their shape, collapsing in internal rotation of the arm and dilating maximally in external rotation [64].

Historically, suprascapular neuropathy was speculated as a diagnosis of exclusion; however several studies have shown multifactorial causes of suprascapular neuropathy. Other sources of suprascapular neuropathy include intimal damage to the suprascapular artery resulting in microemboli in the vasa nervorum [37], glenohumeral dislocation [43–45], fractures around the shoulder girdle [42, 65, 66], and penetrating injury to the shoulder or surgical procedures using a posterior approach to the scapula [67]. The clinical and imaging characteristics of entrapment neuropathies of the suprascapular nerve and its branches differ, depending on the location of compression, traction, or injuries. Entrapment of the suprascapular nerve at the scapular notch leads to supraspinatus and infraspinatus muscle denervation, while distal entrapment at the spinoglenoid notch typically causes isolated involvement of the infraspinatus muscle [68].

MRI is a valuable diagnostic modality in patients who have suprascapular nerve entrapment. The MRI characteristics of compressive neuropathy consist of direct signs involving the nerve and indirect signs pertaining to muscle denervation. Abnormalities in the signal intensity, size, and position of the affected nerve are direct signs of peripheral nerve entrapment [6]. The structural causes may be displayed including space-occupying lesions, such as ganglia and tumors, or osseous abnormalities, such as bony spurs, fracture fragments, and callus. Ganglia are isointense or hypointense in comparison with muscle on T1-weighted images, are homogeneously hyperintense on T2-weighted sequences, and show thin peripheral enhancement with

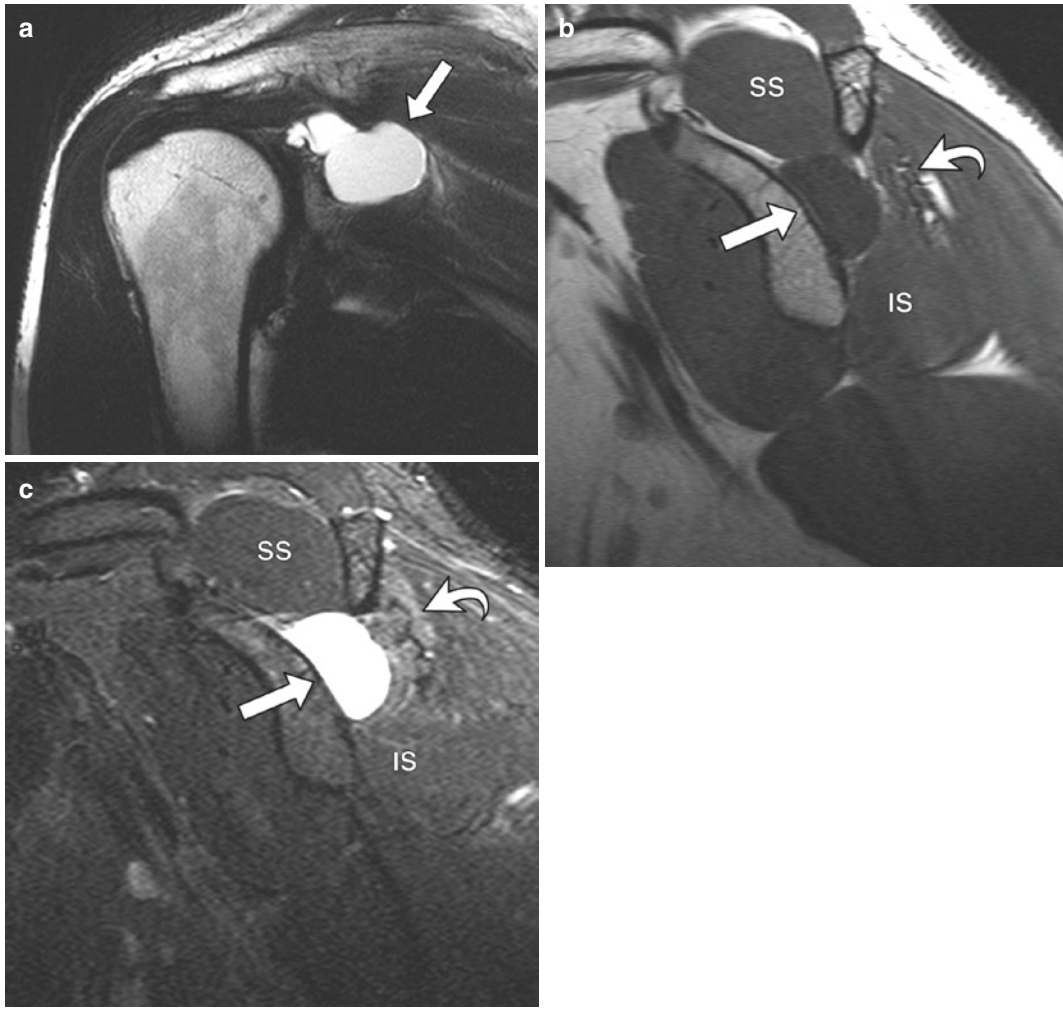

Fig. 14.6 30-Year-old male: (**a**) Coronal oblique T2 fast spin echo showing a hyperintense large cyst occupying the spinoglenoid notch (arrow). (**b**) T1-weighted sagittal oblique image shows the cyst as a low/isointense mass (arrow) and mild atrophy of the upper infraspinatus muscle. The supraspinatus muscle (SS) appears normal suggesting involvement of the infraspinatus branch of the suprascapular nerve. (**c**) STIR sagittal oblique image shows the cyst (arrow) in spinoglenoid notch with secondary edema of the upper portion of infraspinatus muscle (IS, curved arrow)

gadolinium (Figs. 14.6 and 14.7). The pattern of muscle denervation provides information about the duration of entrapment and can identify the location of neurologic compromise. Acute denervation is demonstrated as hyperintensity of the supraspinatus and infraspinatus or infraspinatus muscle alone on fluid-sensitive sequences. Chronic compression is displayed as a reduction in muscle bulk and fatty infiltration of the involved muscles. Involvement of both the supra- and infraspinatus muscles indicates compression at the suprascapular notch, while isolated involvement of infraspinatus reflects compression at the spinoglenoid notch [6].

14.3.5.2 Axillary Nerve Neuropathies

Axillary neuropathy can be secondary to stretching injures or extrinsic compression in the quadrilateral space induced by humeral fractures, improper use of crutches, casts, fibrous bands, space-occupying lesions, and inferior paraglenoid cysts [16, 69]. Iatrogenic nerve injury during

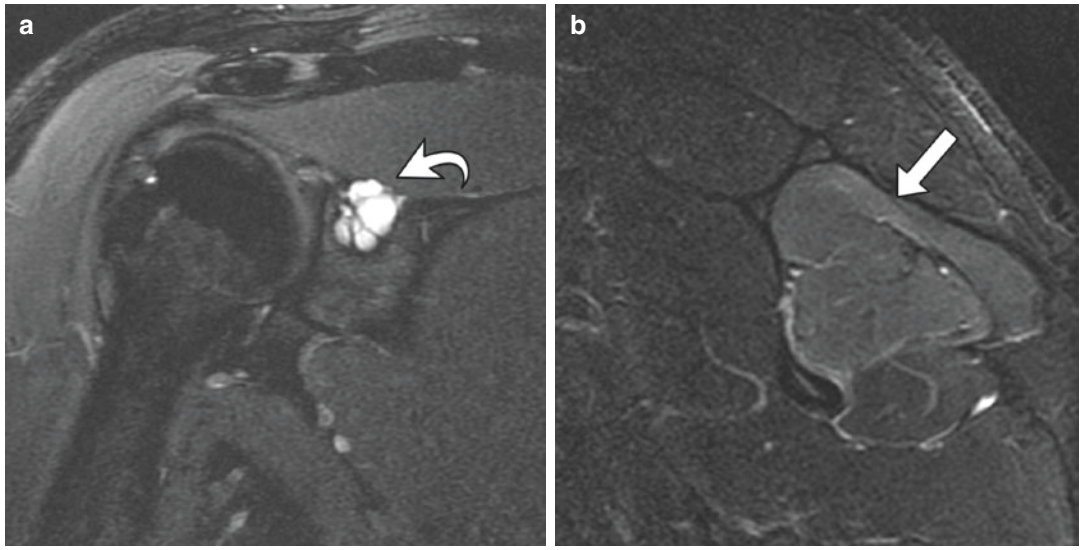

Fig. 14.7 39-Year-old male showing (**a**) coronal oblique T2-weighted FSE fat-suppressed image in which a multiloculated cyst (curved arrow) involves the suprascapular notch. (**b**) Sagittal oblique STIR image shows diffuse neurogenic edema involving the infraspinatus muscle (arrow)

arthroscopic procedures around the coracoid or by posterior surgical arthroscopic portals has also been described [16]. When entrapment of the axillary nerve happens in the quadrilateral space, there is isolated denervation of the teres minor muscle because the anterior branch of the nerve (supplying the deltoid) is spared (Fig. 14.8). Axillary neuropathy may be identified incidentally during routine MRI of the shoulder, since clinically this may not be apparent because the action of teres minor cannot be definitely separated from the contribution of infraspinatus. When symptomatic, axillary neuropathy appears with ambiguous, often nonspecific posterior shoulder pain, sensory disturbances over the external aspect of the shoulder, and weakness aggravated by overhead activity and heavy lifting. Even without any noticeable soft-tissue abnormality along the nerve course, the imaging diagnosis of axillary neuropathy is based on the signs of volume loss and signal alteration of the affected muscles in the absence of a tendon tear [6].

Quadrilateral Space Syndrome

Quadrilateral space syndrome is a rare condition referring to an isolated compressive neuropathy of the axillary nerve. The syndrome was first explained by Cahill and Palmer [70] in 1983 in which the neurovascular bundle, containing the axillary nerve and posterior humeral circumflex artery, is compressed by fibrous bands as it travels through the quadrilateral space. It commonly occurs in young athletes between the ages of 25 and 35 years without a history of serious trauma. The syndrome is determined clinically by poorly localized anterolateral shoulder pain and is aggravated by forward flexion, abduction, and external rotation of the humerus. This pain is typically associated with point tenderness over the posterior shoulder, near the teres minor insertion site. Skin paraesthesia in the sensory distribution of the axillary nerve (overlying the deltoid muscle) and atrophy or weakness of the teres minor and deltoid may occur as well [71]. The development of fibrous bands in the quadrilateral space sounds to be related to microtrauma due to repeated overhead activity such as throwing [72, 73]. Although extrinsic compression by fibrous bands is the most common cause of the syndrome, various other causes have now been discussed in the literature. Robinson et al. [74] were the first to report a case of quadrilateral space syndrome caused by a paralabral cyst.

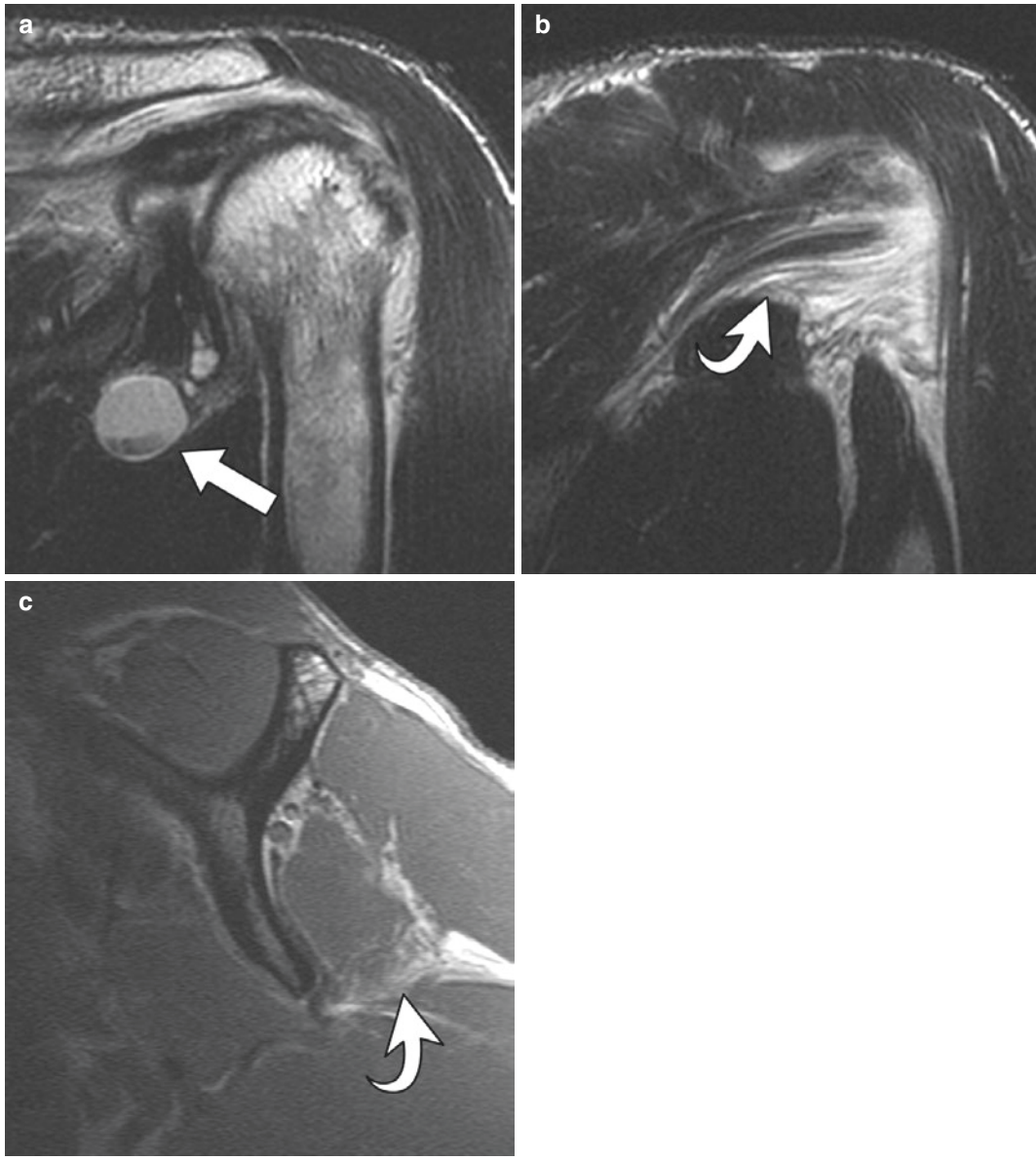

Fig. 14.8 43-Year-old male showing (**a**) coronal oblique T2-weighted FSE images with multiloculated cyst that dissected towards the quadrilateral space (arrow) and secondary edema of the teres minor muscle (**b**) (curved arrow). (**c**) Sagittal T1-weighted image shows loss of bulk of the teres minor muscle with fatty infiltration (curved arrow)

Juxta-articular cysts are a typical entity occurring next to large joints and are a well-established reason of compressive suprascapular neuropathy in the shoulder. Glenoid labral cysts are thought to arise from extrusion of joint fluid via labrocapsular tears. They develop most commonly in the superior and posterior aspects and are infrequent in the inferior region of the joint [75]. When large, inferior labral cysts can create mass effect on the neurovascular bundle in the tightly constrained quadrilateral space. Quadrilateral space masses such as soft-tissue tumors and hematomas have also been described to result in this syndrome. Other reported cases of axillary nerve injury include trauma (humeral neck or scapular fractures), acute translational

incidents such as glenohumeral subluxation or anterior dislocation, and surgical or arthroscopic intervention [76–78]. The relatively fixed position of the quadrilateral space makes it particularly susceptible, and such injuries usually affect both the teres minor and deltoid muscles due to the level at which the axillary nerve injury happens [79, 80].

The diagnosis of quadrilateral space syndrome may be difficult on the basis of clinical examinations alone and is typically one of exclusion. Generally poorly localized shoulder pain may be confused with a rotator cuff injury or impingement. When a clear structural lesion such as fibrous bands or a mass is discovered on imaging, diagnosis can be relatively easy. MRI is superior to ultrasound to display any space-occupying lesion in the quadrilateral space, such as paralabral cysts extending off the inferior aspect of the glenoid in association with a tear of the inferior labrum [63, 76, 81]. However, MRI generally shows no structural abnormality within the quadrilateral space but may display secondary features of denervation myopathy. These characteristics include atrophy of the teres minor and, less frequently, of the deltoid, which is seen as a reduction in muscle volume and fatty degeneration with chronic compression [71]. Fatty degeneration is best observed on T1-weighted sequences but can also be viewed as abnormal signal intensity within the muscle belly on T2-weighted images (Fig. 14.8). Quadrilateral space syndrome is a potentially reversible cause of shoulder pain. It should be considered when selective atrophy or a signal change of teres minor with or without involvement of the deltoid is observed in the appropriate clinical setting. Other diagnoses producing muscle atrophy or neurogenic edema in the absence of a clear cause, such as traumatic injury to the axillary nerve, brachial plexus, or nerve roots, must also be considered [81].

Posttraumatic Axillary Nerve Injury

Up to 45% of shoulder dislocation cases may have associated nerve injury [44]. The axillary nerve is most commonly involved, because it has a relatively tethered course within the quadrilateral space. The risk for axillary nerve and brachial plexus injury is higher if the shoulder is not reduced within 12 h. In spite of the high prevalence of axillary nerve injury following shoulder dislocation, only a few reports in the radiological literature address the association of teres minor atrophy with prior dislocation [76, 82]. Traction and compression of the axillary nerve and subscapularis muscle can be induced by the dislocated humeral head or during manipulative reduction in which traction with rotation or abduction is concurrently performed. Posttraumatic injury to the axillary nerve can also be secondary to proximal humeral fracture [66], and seldom due to a direct blow to the deltoid muscle. The clinical diagnosis of axillary nerve injury may be difficult because the signs and symptoms are often ambiguous. Because branches to the lateral cutaneous innervation and to teres minor muscle are closest to the glenoid rim, they are most vulnerable to posttraumatic injuries [83]. Injury to the infraspinatus muscle, however, may be clinically overlooked.

MRI may illustrate signs suggestive of teres minor denervation injury with increased signal on water-sensitive images or atrophy of the muscle. Unlike EMG studies, which can directly assess the function of nerves, MRI provides indirect indicators of nerve injury by finding changes in fat and water composition of muscle. Effects to T1 and T2 prolongation can be recognized within 15 days post-injury [69, 84]. Most typically, isolated fatty atrophy of the teres minor muscle is incidentally identified (Fig. 14.9). The diagnosis of teres minor atrophy in the absence of quadrilateral space lesions should prompt accurate assessment for signs suggestive of posttraumatic glenohumeral instability and prior dislocation [76, 82, 85].

14.3.5.3 Scapular Winging

Entrapments of the long thoracic, accessory, or dorsal scapular nerves directly prevent scapular movements due to the muscles involved. These entrapments account for the static or dynamic scapular prominence recognized as scapular winging. This clinical condition is distinct from suprascapular and axillary nerve entrapment

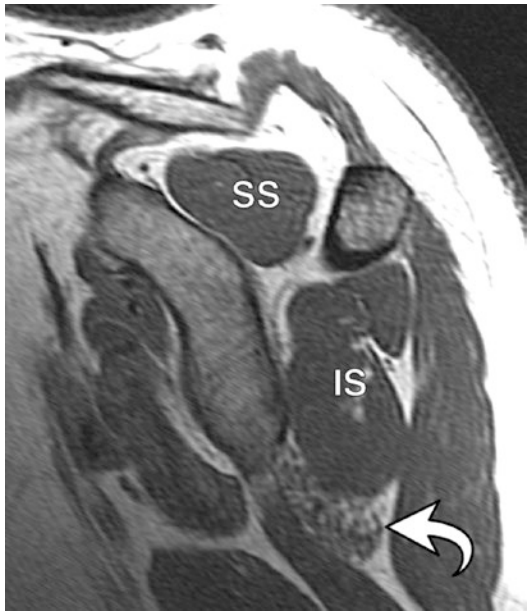

Fig. 14.9 36-Year-old male T1-weighted sagittal oblique image showing isolated atrophy of the teres minor muscle (curved arrow). SS, supraspinatus muscle, IS infraspinatus muscle

Table 14.1 Classification of scapular winging [2, 96]

Primary scapular winging	
Neurologic origin	Spinal accessory nerve (trapezius palsy)
	Long thoracic nerve (serratus anterior palsy)
	Dorsal scapular nerve (rhomboids palsy)
Osseous origin	Osteochondromas
	Fracture, malunions
Soft-tissue origin	Contractural winging
	Muscle avulsion or agenesis
	Scapulothoracic bursitis
Secondary scapula winging	Accompanies glenohumeral disorders and should resolve once that disorder has been addressed
Voluntary scapula winging	

because there is a clinical abnormality suggestive of it, which can be identified on physical examination (Fig. 14.10). The patient is likely to be referred for the exploration of this abnormality [27] (Table 14.1).

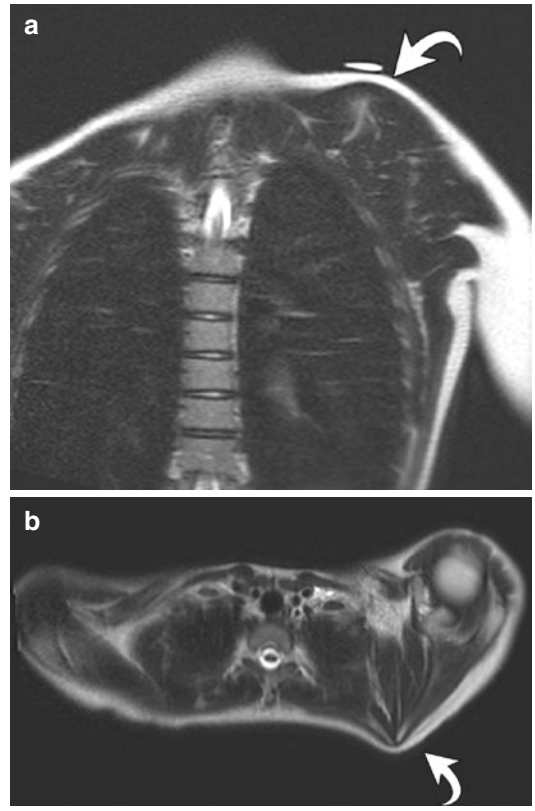

Fig. 14.10 13-Year-old female with clinical evidence of left scapular winging after a fall. Coronal (**a**) and axial (**b**) T2-weighted images show left scapular winging (curved arrow) characterized by deformity and scapular malalignment without muscle edema

The MRI protocol should be adjusted in case of scapular winging. In this case the FOV has to be expanded to include both scapulae, covering the posterior and medial muscles attaching to them. The serratus anterior, the rhomboid muscles, and the trapezius should be inspected (Fig. 14.11). Because this coverage is beyond the reach of a dedicated shoulder coil, a multichannel phased array body coil should be used and the whole width of the scapular girdle must be viewed. Craniocaudally, the investigation must range from the upper edge of the shoulder to the tip of the scapula. This allows a comparison between both sides, which can be helpful when dealing with subtle abnormalities. T2-weighted images with fat suppression and T1-weighted images are performed in the axial and sagittal planes.

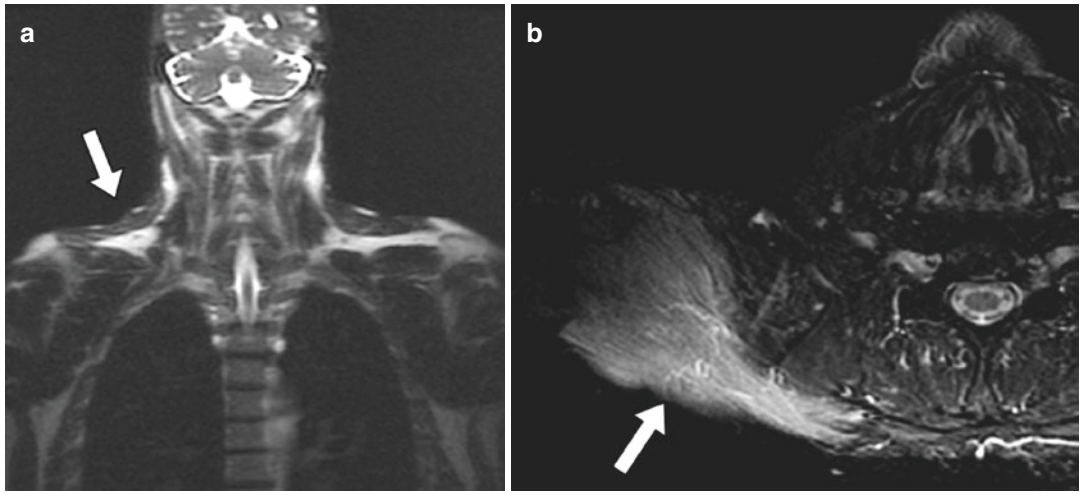

Fig. 14.11 45-Year-old male with right-shoulder pain and weakness with scapular winging for a month. (**a**) Axial STIR and (**b**) coronal T1-weighted MRI show diffuse neurogenic edema involving right trapezius muscle (arrow)

Long Thoracic Nerve Neuropathy

Long thoracic neuropathy most often results from repeated microtrauma due to stretching injury. This usually occurs in athletes (e.g., throwing sports such as javelin, baseball, or when serving or spiking a tennis or volleyball ball) when the head is tilted and rotated laterally away from the affected extremity and the arm is elevated overhead [16]. Direct trauma over the upper anterior chest and whiplash injury may also cause nerve compression [86]. Nontraumatic causes consist of compression by enlarged bursae, such as the subcoracoid bursa or the subscapularis recess. Irrespective of mechanism, it is still unknown where the injury occurs along the course of the nerve. Injury to the long thoracic nerve paralyzes the anterior serratus muscle, which causes medial winging of the scapula and a deficit in active forward flexion, which is more common than lateral winging. The scapular asymmetry (diagnosed by comparing the distance between the spinal processes and the medial edge of the scapula on both sides) may be clinically evident on physical examination. It can be aggravated more on forward flexion of both arms or by the wall push-up test. In the most severe cases, thorough elevation of the arm may be impossible [87]. Physical examination shows an obvious clinical picture with scapular winging, especially when the patient extends his or her arms and pushes against a wall [88]. Direct assessment of the long thoracic nerve is possible only in part and for limited segments with ultrasound. The diagnosis basically will be confirmed by EMG or MRI, which demonstrates signs of denervation (edema, atrophy) of the serratus anterior muscle. The serratus anterior muscle is sometimes outside the usual scope of exploration of the shoulder and standard joint examination may miss the anomaly. A shoulder coil with a large field is sufficient in thin patients. A spine coil and a multielement body coil will give satisfactory results in larger subjects. MRI also allows for elimination of alternative causes of scapular winging.

Accessory Nerve Neuropathy

Spinal accessory nerve neuropathy most frequently follows a stretch injury associated with lifting, heavy load bearing on the shoulders, whiplash, or trauma [89]. It has also been referred to skull deformities, infiltrative lesions, and radiation fibrosis [90]. Moreover, it may follow operations involving the head and neck in which the dissection of a lymph node occurs. Patients present with mild shoulder droop, weakness of shoulder elevation, and scapular winging during shoulder abduction. Atrophy of the trapezius

muscle causes scapular instability and painful shoulder abduction [17]. Lateral scapular winging is always present [91]; however only more severe types like the "droopy shoulder" may be observed, associated with trapezius atrophy, shoulder drop, and lateral winging. Abduction and external rotation against resistance aggravate the scapular displacement, while forward flexion reduces the deformation because of serratus anterior contraction [29]. MRI is indicated in difficult cases to confirm the diagnosis. Focused on the trapezius and anterior serratus muscles, it shows classic signs of denervation affecting the trapezius and allows for exclusion of long thoracic neuropathy.

Dorsal Scapular Nerve Neuropathy

The dorsal scapular nerve supplies the rhomboids and is rarely injured in isolation, being generally involved with a C5 radiculopathy. Reports have been made of injury of the dorsal scapular nerve due to muscle hypertrophy in bodybuilders [92]. Neuropathies of the dorsal scapular nerve are uncommon but their frequency is perhaps underestimated because their impact is generally minor [93]. They result in denervation of the rhomboids, which clinically leads to discomfort or pain in these muscles, minimal scapular winging, and difficulty on arm elevation. The location of nerve compression is unclear, but could be a specific form of thoracic outlet syndrome in the scalene space [94]. One article reports diagnostic confirmation of damage to this nerve by MRI [95], the positive finding being thinner rhomboid muscles on MRI of the thorax. In addition, T2-weighted images may show an increase of pathologic signals suggestive of muscular denervation [95].

14.3.5.4 Polyneuropathies (Parsonage-Turner Syndrome)

Parsonage-Turner syndrome is an uncommon, self-limiting disorder characterized by immediate onset of nontraumatic shoulder pain accompanied with progressive weakness of the shoulder girdle musculature. It was first recognized in 48 patients by Spillane in 1943 [97] and then by Parsonage and Turner [98] in 1948 who described the condition in 136 servicemen, which they called "neuralgic amyotrophy" or "shoulder-girdle syndrome." Afterwards, the pathology has commonly been referred to as Parsonage-Turner syndrome or acute brachial neuritis, although the terms "brachial plexus neuropathy" [99], "acute brachial radiculitis" [100, 101], and "paralytic brachial neuritis" [102, 103] have all been used to describe the entity. Clinical diagnosis may be difficult because symptoms can simulate those of more common disorders such as cervical spondylosis, rotator cuff tears, impingement syndrome, adhesive capsulitis, and calcific tendinitis [97–99, 104]. The exact cause of Parsonage-Turner syndrome is unclear, although viral neuritis [99, 105], immunization [102], autoimmune mechanisms [106], trauma, strenuous exercise, and surgery [107] have all been noted. Prior infection has been reported in up to 25% of cases [99]. The overall incidence has been estimated at 1.64 per 100,000 individuals in one population [108]. The age range of affected patients is very wide, with most patients presenting in the third to seventh decades of life [104, 109]. Males are mostly affected; bilateral involvement is observed in up to one-third of patients [99, 108]. Originally the long thoracic nerve was thought to be the most commonly involved in Parsonage-Turner syndrome [98]. However, future studies showed that the most frequently involved muscles are those innervated by the suprascapular nerve (supraspinatus and infraspinatus) [109], although the entire brachial plexus can be affected. In a study of 27 patients with Parsonage-Turner syndrome, Gaskin and Helms [110] found that the suprascapular nerve was involved in 97% of the subjects and the axillary nerve in 50% [80].

Imaging Findings

There is no specific test for the diagnosis of Parsonage-Turner syndrome. EMG, nerve conduction studies, and MRI must be interpreted in light of the patient's clinical history. MRI is the modality of choice in patients with shoulder pain and weakness, being sensitive for changes indicating denervation injury. The studies by Gaskin and Helms [110] and Scalf et al. [104] are the

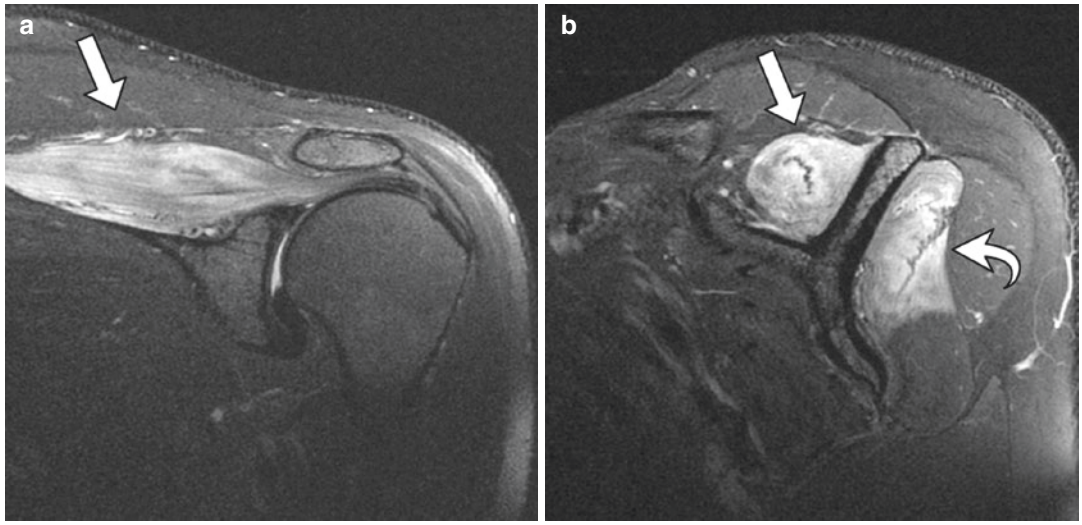

Fig. 14.12 37-Year old male sagittal T2-weighted fat-suppressed coronal (**a**) and sagittal (**b**) oblique showing diffuse neurogenic edema of supraspinatus (arrow) and infraspinatus (curved arrow) muscles. The patient had no history of trauma or other pathology and no evidence of compressive lesions in the suprascapular notch or rotator cuff tear (not shown)

largest reported series describing the MRI features of Parsonage-Turner syndrome to date. The diagnosis is proposed when there is an abnormality of muscles innervated by the brachial plexus in the absence of history of excessive overhead activity, trauma, or morphologic cause at MRI. The earliest recognizable change in denervated muscles is diffusely high signal on fluid-sensitive sequences such as STIR or T2-weighted images (because of increase in extracellular water content) and normal signal on T1-weighted sequences [1]. After a few weeks in the subacute to chronic phase, the denervated muscle may decrease in volume with increased T1 signal because of fatty infiltration (Fig. 14.12) [104]. Intramuscular signal may return to normal several months after the chronic phase; however, in complete muscle denervation (>1 year after injury), changes are irreversible [111].

MRI is also helpful in excluding intrinsic shoulder abnormalities that can create symptoms equivalent to Parsonage-Turner syndrome such as rotator cuff tears, impingement syndrome, and labral tears. MRI can display structural lesions that may produce similar denervation changes in the rotator cuff musculature such as cuff tears or masses compressing the brachial plexus or peripheral nerves and other causes of intramuscular high signal including myositis, rhabdomyolysis, inflammatory myopathies, compartment syndrome, and tumor involvement of muscle [112, 113].

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