Musculoskeletal

MR imaging evaluation of suprascapular nerve entrapment

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Abstract. The aim of this study was to assess the significance of muscular edema, atrophy, and fatty changes in the diagnosis of suprascapular nerve entrapment (SSNE), and to confirm muscular edema as the most significant sign of neuropathy. A retrospective study of 18 patients with suprascapular nerve entrapment was performed. All patients underwent electromyographic studies and MR imaging with a 1.5-T Echo Speed system (General Electric, Milwaukee, Wis.). The diagnosis of muscle edema was reached when muscles presented a high signal on T2-weighted fast spin-echo (SE) fat-suppressed images. Muscular trophicity and fatty changes were analyzed on a sagittal oblique cut using SE T1-weighted images. Intra- and inter-observer reproducibility using kappa test, sensitivity, and specificity were analyzed, together with negative and positive predictive value of each criterion. The topographic diagnosis was correct as edema affected the infraspinatus muscle alone when the suprascapular nerve was entrapped at the spinoglenoid notch. Both the supraspinatus and infraspinatus muscles were affected when nerve was compressed at the suprascapular notch. Sensitivity and specificity of muscular edema were, respectively, 94.5 and 100%. Muscular atrophy sensitivity and specificity were 81 and 80%, respectively. Fatty changes sensitivity and specificity were 25 and 96%, respectively. Muscular edema seems to be a more sensitive sign of SSNE than muscle atrophy and fatty changes when compared with EMG results. Magnetic resonance imaging can reach a positive,
Introduction

Suprascapular neuropathy is a rare occurrence accounting for approximately 0.4-2% of shoulder pain, which affects mostly athletes and young people [1,2]. The suprascapular nerve is a mixed nerve, functioning essentially as a motor nerve originating from roots C5 and C6. It extends from the suprascapular notch to the supraspinatus fossa and innervates the supraspinatus muscle. It goes round the scapular spine base before entering the infraspinatus fossa innervating the infraspinatus muscle. This anatomical layout explains the topography of abnormalities. The suprascapular nerve follows a Z-shaped pattern with two fixed points: the first situated at the suprascapular notch and the second at the spinoglenoid notch. When nerve entrapment occurs at the suprascapular notch, both infraspinatus and supraspinatus muscles are denervated. When compressed at the base of scapular spine, infraspinatus denervation occurs while the supraspinatus remains intact.

The diagnosis of SSNE remains a challenge for clinicians; electromyography (EMG) is the gold standard and is essential to confirm muscle denervation; however, it is seldom carried out initially because clinical data rarely point to the diagnosis of neuropathy. Diagnosis of neuropathy is often missed with plain radiographs or CT arthrography as muscle denervation can only be suspected when muscle atrophy or fatty replacement are visible.

Magnetic resonance imaging reveals morphological modifications and signal changes of denervated muscles such as muscle edema, muscle atrophy, and fatty changes [3,4]. It can detail the anatomical site of nervous damage [5,6] but is limited with regards to etiology of nerve entrapment when no mass or cyst is affecting or compressing the nerve. Magnetic resonance imaging can also depict a paralabral cyst which may be overlooked with CT arthrography because of beam-hardening artifact or the absence of cyst opacification [6,7].

The purpose of the present study was to evaluate muscular edema, muscle atrophy, and fatty changes following suprascapular neuropathy. The imaging findings were compared with EMG in order to determine their sensitivity relative to this standard procedure.

Materials and methods

Population

In this retrospective study, two groups of patients were selected: the first group with suprascapular neuropathy and the second as a control group.

The first group included 18 patients (12 men and 6 women) ranging in age from 16 to 54 years (mean age 34 years). Nerve injury was confirmed by electromyographic data for all patients, excluding Parsonage-Turner syndrome or cervical spine impingement. All patients also underwent MRI. The absence of any major pathology of the rotator cuff, such as full-thickness tendon tear, tendinitis, or calcific tendinitis, was a criterion for selection. The absence of labral tear, such as instabilities or SLAP lesions, was a criterion for selection, too. Standard radiography had previously ruled out tendinous calcification, and shoulder CT arthrography (7 patients) and MRI ruled out any significant rotator cuff tear.
The control group included 10 patients (7 men and 3 women). Subjects ranging in age from 15 to 65 years, mean age 35 years, complained of shoulder pain. All these nonconsecutive patients underwent an EMG and MRI of the shoulder which confirmed the absence of nerve injury.

Muscle atrophy of supra- and infraspinatus was clinically suspected in 5 patients in the group with suprascapular nerve entrapment. An isolated infraspinatus muscular atrophy was discovered in 3 patients in the same group. Indeed, suprascapular neuropathy was only diagnosed in 8 patients of 18, i.e., less than 50% of the cases. Following clinical examination, EMG and MRI were performed. The interval between EMG and MRI varied from 1 day to 6 months: in 9 cases, EMG was carried out before MRI (5 days to 8 weeks), in 3 cases exams were performed simultaneously, and in 6 cases MRI led to an EMG (1 day to 6 months). The delay between the first exam (EMG or MRI) indicating the beginning of symptomatology, i.e., pain and muscle weakening, was variable (from 15 days to 2 years). A review of 18 EMG revealed 8 neuropathies at the scapular notch and 8 at the spinoglenoid notch. Two patients had negative initial EMG findings. A second more meticulous EMG was performed after MRI in these 2 patients confirming injury at the suprascapular notch and at the spinoglenoid notch, respectively.

Various causes were determined for suprascapular nerve compression. In 8 patients it was due to a dynamic entrapment at the suprascapular notch located beneath the superior transverse scapular ligament and in 1 patient to a cyst impingement at the scapular notch. In 3 patients it was also related to a dynamic entrapment at the spinoglenoid notch located beneath the lower transverse scapular ligament. In 1 patient a large scapular ganglion cyst with soft tissue extension was responsible for the nerve compression. A spinoglenoid cyst was causing nerve impingement in 4 patients, four of which with posterior labral tear. Surgical removal of a scapular brown tumor resulted in nerve injury at the spinoglenoid notch in 1 patient.

Ten of 18 patients underwent conservative treatment, 6 had surgery (surgical repair of entrapment injury), and 2 patients were treated with fluoroscopically guided aspiration for cyst reduction. Fifteen patients had a clinical follow-up (1.5-23 months); 7 patients had a follow-up EMG, 1-6 months after initial exam and 10 patients had a follow-up MRI (4-23 months after initial exam).

Clinical examination revealed residual pain in 2 women. Eleven patients did not report any pain and 1 patient had external rotation deficit. The evolution of electromyographic signs and MRI characteristics are displayed in Table 1.

Table 1. Magnetic resonance imaging and electromyographic follow-up. SNC suprascapular notch cyst; DSSNE dynamic suprascapular notch entrapment; SGC spinoglenoid cyst; GC ganglion cyst; DSGNE dynamic spinoglenoid notch entrapment; SBT surgery of a brown tumor; RG radio-guided aspiration; D days; M months
<table>
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<tr>
<th>Patient no.</th>
<th>Age (years)</th>
<th>Gender</th>
<th>Diagnosis</th>
<th>Treatment</th>
<th>EMG follow-up</th>
<th>Muscular edema</th>
<th>Muscular atrophy</th>
<th>Fatty changes</th>
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<td>SNC</td>
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<td>-</td>
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<td>No (7 M)</td>
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<td>RG aspiration</td>
<td>Incomplete</td>
<td>No (6 M)</td>
<td>Grade 2 (6 M)</td>
<td>Grade 0 (6 M)</td>
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<tr>
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<td>Grade 2 (3.5 M)</td>
<td>Grade 2 (3.5 M)</td>
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</table>

**MR imaging technique**

All MR imaging in the first group, except in 3 cases, and all MR imaging in the control group were performed with a 1.5-T superconducting magnet (Signa, GE Medical Systems, Milwaukee, Wis.) using only a dedicated shoulder coil associated to a 12.5-cm circular coil. The following pulse sequences were used: spin-echo (SE) T1-weighted axial and sagittal oblique images (TR/TE:...
400 ms/16 ms; 5-mm section thickness; 2-mm intersection gap; 16-cm field of view (FOV); and 256 × 192 matrix. Fast SE T2-weighted axial, sagittal, and coronal oblique images with frequency-selective fat-saturated (TR/TE: 3000 ms/45 ms; 4-mm section thickness; 1-mm intersection gap; 16-cm FOV; 256 × 192 matrix) or conventional SE T2 for 3 patients. Two patients did not undergo SE T1-weighted sagittal images. In addition, 13 patients with suprascapular nerve entrapment underwent an SE T1-weighted axial sequence with IV gadolinium injection with fat-saturated (TR/TE: 400 ms/16 ms; 16-cm FOV; 256 × 192 matrix). In 2 cases contrast material was directly injected into the shoulder joint prior to MRI to obtain an MRI arthrography (Visipaque 300, Nycomed, Oslo, Norway). Three patients from the first group underwent an MRI for shoulder investigation using a 1-T superconducting magnet (Signa, GE Medical Systems, Milwaukee, Wis.; or Gyroscan, Philips Medical Systems, Eindhoven, The Netherlands) with similar protocol.

**Image analysis**

All images were independently read by two radiologists without prior knowledge of clinical and EMG data. Selected diagnostic criteria included muscular edema, muscle volume, and fatty changes of the infraspinatus and supraspinatus.

We considered that muscular edema existed when muscles presented a high signal on fast SE (FSE) T2-weighted fat-suppressed images. Muscle signal was compared with that of adjacent muscles (deltoid, subscapularis, and teres minor) and soft parts in order to be free of coil artifact generating a signal gradient decreasing in intensity as it penetrates tissues depth.

Muscle volume was graded on a sagittal oblique cut with SE T1-weighted images intersecting 2 cm from the inward base of the scapular spine. A convex muscle surface was considered normal (grade 0), whereas a straight (grade 1) or concave muscle surface (grade 2) was considered pathological (Fig. [1]).
Fig. 1. The different grades of muscular trophicity. Muscle volume is graded on a sagittal oblique cut with spin-echo (SE) T1-weighted images intersecting 2 cm from the inward base of the scapular spine. A convex muscle is considered normal (grade 0), whereas a straight (grade 1) or concave muscle surface (grade 2) is considered pathological.

Fatty changes were graded 0-4, according to Goutallier et al. ’s classification adapted to MRI specifications, using axial and sagittal oblique SE T1-weighted images without fat saturation: stage 0, no fatty infiltration; stage 1, some fatty streaks; stage 2, less fat than muscle; stage 3, as much fat as muscle; stage 4, more fat than muscle. In this classification, stages 0 and 1 are normal and stages 2-4 are pathological. In our study, according to Fuchs et al., stage 2 was considered as minor fatty changes, and stages 3 and 4 as severe. We did not evaluate muscle volume and fatty changes for 2 patients in the first group because they did not undergo sagittal oblique SE T1-weighted images.

Spin-echo T1-weighted fat-saturated images with IV gadolinium injection were screened for infra- or supraspinatus muscle enhancement in 13 patients in the first group.

**Statistical analysis**

We analyzed the intra- and inter-observer reproducibility using kappa test, sensitivity, and specificity, together with negative and positive predictive values of each criterion.

Spin-echo T1-weighted fat-saturated sequence with IV gadolinium injection was not included in this independent reading.

**Results**

Intra- and interobserver reproducibility for depicting muscular edema were, respectively, 1 and 1. Intra- and interobserver reproducibility for depicting muscle atrophy were, respectively, 0.92 and 0.88. Intra- and interobserver reproducibility for depicting fatty changes were, respectively, 0.90 and 0.81. The excellent interobserver reproducibility of each criterion enabled us to use only one series of results.

Following muscular edema analysis (36 explored muscles), 25 muscles (8 supraspinatus, 17 infraspinatus) presented a high homogeneous signal on FSE T2-weighted fat-suppressed images. Nine supraspinatus muscles did not present any signal anomaly; however, in one female patient, supraspinatus and infraspinatus muscles did not present any signal anomaly on FSE T2-weighted fat-suppressed images, whereas the patient’s EMG was positive for the diagnosis of SSNE. So, 8 patients were diagnosed with infraspinatus and supraspinatus muscle edema (total of 16 muscles).
Nine patients were diagnosed with infraspinatus muscle edema (total of 9 muscles) and in one female patient, there was an apparent discrepancy between MRI and EMG results. In the control group, no muscle presented a high homogeneous signal on FSE T2-weighted fat-suppressed images. As a result, muscular edema sensitivity was 94.5% and specificity was 100% in the presence of suprascapular nerve entrapment. The positive predictive value of this sign was 100%, and negative predictive value was 91% (Fig. 2).
Fig. 2a-d. Magnetic resonance arthrography (Visipaque 300, Nycomed, Oslo, Norway) in a 19-year-old woman with suprascapular nerve entrapment at the scapular notch showing muscular edema. a Coronal fast SE (FSE) T2-weighted fat-suppressed image demonstrates homogeneous hypersignal of supraspinatus muscle (arrow). b Axial and c sagittal FSE T2-weighted images show muscular edema of supra (black arrow) and infraspinatus (white arrow) muscles. b Demonstrates the difference in signal between infraspinatus (arrow) and teres minor muscle (arrowhead). d Fat-suppressed axial SE T1-image shows a homogeneous enhancement of the infraspinatus muscle (arrow) following IV gadolinium injection

Following muscular mass analysis (32 explored muscles; 2 patients did not undergo SE T1-weighted images), 12 muscles were classified as grade 0, 5 muscles as grade 1, and 15 muscles as grade 2 in the first group. In the control group, 16 were graded 0, 4 graded 1, and 0 graded 2. Consequently, muscle atrophy sensitivity and specificity were, respectively, 81% and 80% with a positive predictive value of 72% and a negative predictive value of 87% (Fig. 3).
Fig. 3. a Normal 33-year-old male with a convex supraspinatus (black arrow) and infraspinatus (white arrow) muscle surface. b A 16-year-old boy with suprascapular nerve entrapment at the scapular notch due to a cyst with a convex supraspinatus (grade 0, black arrow) and straight infraspinatus (grade 1, white arrow) muscle surface on a sagittal oblique SE T1-weighted image. c A 19-year-old man with a dynamic suprascapular nerve entrapment at the scapular notch with a muscle atrophy (grade 2) of supraspinatus (black arrow) and infraspinatus (white arrow) muscles on a sagittal oblique SE T1-weighted image.

Following muscular fatty changes in the first group (32 explored muscles; 2 patients did not undergo SE T1-weighted images), 15 were graded 0, nine were graded 1, and 6 were graded 2. Two patients were graded 3 and none were graded 4. Thus, 75% of patients were considered normal, 19% with minor fatty changes and 6% with severe fatty replacement. In the control group, 16 muscles were graded 0 and 4 were graded 1. Consequently, sensitivity and specificity of fatty muscular changes were, respectively, 25% and 96%. Positive and negative predictive values were, respectively, 80% and 67% (Fig. 4).
Fig. 4a-c. Magnetic resonance SE T1-weighted images demonstrating the different grades of pathological muscular fatty changes according to Goutallier et al.’s classification adapted to MRI.

a A 42-year-old man with a dynamic suprascapular nerve entrapment at the scapular notch. Both arrows point to a grade-2 muscular fatty changes of the supraspinatus (black arrow) and infraspinatus muscle (white arrow).

b A 19-year-old man with a dynamic suprascapular nerve entrapment at the scapular notch. Black arrow points to grade-3 muscular fatty changes of the supraspinatus and white arrow points to grade-3 muscular fatty changes of the infraspinatus muscle.

c A 52-year-old man with a significant rotator cuff tear affecting the infraspinatus tendon, illustrating severe fatty changes of the muscle (grade 4, white arrow). There is no evidence of muscular fatty changes of the supraspinatus muscle (black arrow).
Finally, in the 13 patients who underwent SE T1-weighted axial sequence with IV gadolinium injection and fat saturation, all denervated muscles displayed homogeneous enhancement (Fig. 2d).

**Discussion**

Nerve injury or compression can vary in origin; some are related to anatomically predisposing factors, others to violent sports, and others result from less frequent etiologies. Anatomically predisposing nerve compression factors, such as narrow suprascapular notch, sharp edges, thick superior transverse scapular ligament, or the presence of a lower transverse scapular ligament, more frequent in male subjects [10], are well documented in the literature [11, 12]. Nerve injury may occur during violent and sudden or repeated scapular motion, especially in such sports as volleyball, tennis, or weightlifting [13]. In athletes, this dynamic nerve entrapment appears at the spinoglenoid notch, whereas in the general population it occurs mainly at the suprascapular notch [14]; however, a recent study involving professional volleyball players shows high frequency of asymptomatic suprascapular nerve injury in athletes presenting with muscle atrophy and minimal functional impact [13]. On a more general basis, posterior paralabral cysts could be responsible for nerve compression [11, 12]. In this case, cysts are associated with labral tear and posterior instability if the posterior portion of the labrum is ruptured, or with a SLAP lesion if the superior part of the labrum is ruptured [15, 16, 17]. Other less common etiologies, such as a scapular tumor extending into soft tissue or a soft-parts tumor, may cause nerve entrapment [18]. A scapular ganglion cyst may be an example of such an occurrence. Exceptionally, nerve injury may be linked to iatrogenic surgical manipulation [18, 19, 20, 21]. Finally, the mechanism of injury may be precipitated by anterior shoulder dislocation [17].

**Difficulty in defining clinical diagnosis**

Clinical examination is often not very contributory and specific, particularly in athletes. The clinical differential diagnosis can be quite diverse and includes rotator cuff abnormalities such as tear or impingement syndrome, calcific tendinitis, adhesive capsulitis, cervical radiculopathy, and traumatic lesion [22]. This can perhaps explain the long and variable delay (15 days to 2 years) between the beginning of symptoms and EMG or MRI.

As a follow-up to clinical evaluation, electromyographic evaluation is the gold standard to establish a positive diagnosis, showing the increase in distal motor latencies and pointing out the site of injury [2, 23, 24]. However, it is not always proposed as a first investigative tool of recommended exams in shoulder pain. In our study, MRI was followed by electromyographic evaluation in a significant number of patients (approximately 33%). Nevertheless, this invasive method is not always helpful in locating a lesion, as was the case for two of our patients. A diagnosis of neuropathy was clinically reached and confirmed by MRI. Prior EMG examination was too superficial, due to severe supraspinatus and infraspinatus atrophy, and delayed the diagnosis by measuring a normal deltoid muscular activity. Indeed, negative EMG results do not exclude a positive diagnosis [25].

In addition, a spinoglenoid cyst can damage only one of the motor branches innervating the infraspinatus muscle producing false negatives [25], thus requiring to enlarge the exploration sites.

Lastly, a cyst can affect only sensory branches of the suprascapular nerve, inducing painful symptoms (without muscular atrophy) which are not readily measurable on motor latencies [25].
Muscular edema

Some authors have based their studies, using MRI, on muscle atrophy and fatty changes to point out a neuropathy and to depict its etiology \[15,16,26\]. In our observation, muscular edema appears to be a more significant sign of neuropathy. Indeed, our results showed that muscular edema was more sensitive (94.5%) and more specific (100%) than muscle atrophy (sensitivity 81%, specificity 80%) and fatty changes (sensitivity 25%, specificity 96%); however, the specificity of muscular edema should not be taken into consideration as patients in the control group presented no pathology conducive to muscular edema, which may have affected the results. Overall, muscular edema is not very specific and is observed in conditions other than neuropathy as in intense muscular exertion or following intramuscular hemorrhage \[27\]. Muscular edema can also be associated with tumors, polymyositis, acute inflammation or infection, effects of ionizing radiation, or rhabdomyolysis \[27\]. In acute brachial neuritis, supra- and infraspinatus muscles are both vulnerable along with other scapular muscles \[28\]. In the majority of these shoulder lesions, muscular edema is heterogeneous and may involve subcutaneous soft tissue parts and/or several muscular groups innervated by various nerves \[28\].

It can be assumed that muscle edema becomes specific to suprascapular nerve injury when only affecting supraspinatus and infraspinatus muscles, as noted in axillary nerve lesions when only deltoid and teres minor are involved \[26\]: As nerve compression occurs at the suprascapular notch, both infraspinatus and supraspinatus present muscular edema. When compressed at the base of the scapular spine, the infraspinatus shows muscular edema while the supraspinatus remains intact. Consequently, muscular edema can determine the site of nerve injury. Muscular edema becomes specific when it is homogeneous throughout the whole muscle and when clinical and MR data exclude any other organic etiology \[29,30,31,32\].

Muscular edema may be explained by hydration modifications of the muscular mass, in the case of denervated muscles \[27,33,34,35\]. Signal modifications of muscles on T2-weighted images appear as early as the fifteenth day of the onset of neuropathy \[34\]. In one of our cases, a patient underwent a shoulder MRI 15 days after the onset of symptoms. A high T2 signal intensity was diagnosed in the supra- and infraspinatus muscles. This finding was corroborated by Küllmer et al.’s experimental study pertaining to complete suprascapular nerve section in the rabbit \[35\]. Küllmer et al. identified an increasingly high-intensity T2-weighted signal with a peak between days 21 and 35 after nerve section. Signal modifications were not visible prior to day 15; however, our study diverged from Küllmer et al.’s fundamental data in the latter phase of neuropathy. In fact, in their study \[35\], the muscular signal becomes normal on T2-weighted images from day 64 following nerve section. In contrast, our study revealed muscular edema in 15 patients after day 64 following nerve injury with a delay between 3 and 24 months. Moreover, Grainger et al. \[36\] identified a muscular edema of anterior interosseous nerve syndrome in 3 cases with examination delays comparable to ours (3-8 months). Lastly, Yu and Fischer \[37\], while excluding an isolated case of suprascapular nerve injury due to a clavicular fracture, described supra- and infraspinatus edema 1 year after initial trauma. Küllmer et al.’s results may diverge from Grainger et al.’s \[36\], Yu and Fischer’s, and ours because their study involved total nerve section, whereas Grainger et al.’s \[36\], Yu and Fischer’s \[37\], our clinical study investigated partial and reversible nerve injury.

In addition, homogeneous enhancement should be underlined on SE fat-saturated T1-weighted images after IV gadolinium injection of the supra- and/or the infraspinatus muscle. This contrast enhancement was visible in all patients with muscle denervation; however, no specific experimental or clinical studies have been described showing a connection between neuropathy and muscular enhancement. Currently, no hypothesis has been definitively adopted to explain this condition. Hyperemic phenomena may be suggested as a possible explanation.
Muscle atrophy

In their study, Küllmer et al. determined that muscle atrophy could be observed as muscular edema at the acute or subacute phase of neuropathy with a decrease in muscle diameter (ultrasonography and MRI) starting on day 21 post denervation \[35\]. In the same way, we noticed an infraspinatus atrophy in one patient who underwent shoulder MRI on days 15 and 45 after the onset of symptoms. On day 15, muscle atrophy was not detectable but was visible on day 45; however, the delay between exams (EMG and MRI) and the onset of symptomatology (15 days to 2 years) was too irregular to confirm a definite relationship between nerve entrapment and the early onset of muscle atrophy.

The topographic value of muscle atrophy to determine the nerve injury site remains similar to that of muscular edema. Moreover, muscle atrophy in acute or subacute forms of neuropathy does not necessarily evolve into fatty muscular replacement \[35\]. Indeed, MRI demonstrated muscle trophicity in 6 of our patients with recovery or stability at 7 and 11 months, respectively, without fatty replacement; however, muscular trophicity evaluation remains a subjective but very practical tool on everyday basis in as much as it does not require more elaborated techniques for muscular surface or volume measurements \[6\].

Fatty muscular changes

We applied Goutallier et al.’s CT classification \[8\] to our MRI studies, assuming that CT fatty changes can be superposed on MRI fatty muscular replacement \[6\],[7]. We simplified Goutallier et al.’s classification from five grades to three as explained in Fuchs et al.’s study \[6\]. Our results showed only 19% of muscles with minor fatty changes (grade 2) and fewer (6%) with severe fatty changes (grades 3 or 4). In accordance to Fleckenstein et al.’s report \[27\], we noticed irreversible fatty changes in patients with chronic neuropathy as noted by several authors \[12,26,37\]. Indeed, among our 5 patients with fatty changes, 4 remained stable and 1 went from grade 1 to grade 3 over a 6-month period.

The topographic value of muscular fatty changes to determine the nerve injury site remains similar to that of muscular edema and muscle atrophy; however, sensitivity of muscular fatty changes is low (25%), offering little help in the detection of suprascapular neuropathy.

Study limitations

This study presents some limitations due to the nature of the pathology and to the small population in our series. Non-consecutive exams, the retrospective aspect of our work resulting in an involuntary selection bias, did not allow conclusive establishment of the specificity of muscular edema, muscle atrophy, and muscular fatty changes. Moreover, the absence of muscular edema in the control group prevented determination of the specificity of this MR imaging criterion. In addition, it should be noted that muscle trophicity evaluation was subjective and that MRI may not accurately reflect fatty changes of the supraspinatus and infraspinatus muscles since no histological study was performed on muscular biopsy. Finally, some patients did not have an EMG follow-up, altering the evaluation of imaging criteria.

Further studies are necessary in order to assess whether or not muscular edema occurs in chronic suprascapular neuropathy, in order to understand muscle trophicity evolution. Finally, we must determine if muscular fatty changes can deteriorate as observed in rotator cuff tears and whether MRI evaluation truly reflects fatty degeneration.
Conclusion

Magnetic resonance imaging results demonstrated that muscular edema seems to be a more sensitive sign in the diagnosis of suprascapular neuropathy than muscle atrophy and fatty changes, when compared with EMG results. Thereby, MRI can reach a positive, topographic, and etiologic diagnosis of suprascapular neuropathy.

References


