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Isolated mononeuropathy of the suprascapular nerve: traumatic traction injury as an important differential diagnosis to the entrapment syndrome

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Introduction

The suprascapular nerve (SSN) is a mixed motor and sensory nerve that innervates the supraspinatus (SSP) and infraspinatus (ISP) muscles as well as the capsule of the glenohumeral joint.[3] It surpasses the transverse scapular ligament and, at this point, is susceptible to compression, entrapment, or traction.[9] SSN compression syndrome is the second most common cause of atrophy of the SSP and ISP after rotator cuff injuries.[3] The most common site of compression is the suprascapular notch, and arthroscopic decompression of the nerve is an established therapeutic procedure.[13] Mononeuropathic traction damage is a very rare but important differential diagnosis. This case report describes a detailed setup to detect such damage to the nerve via magnetic resonance imaging (MRI).

Case report

A 40-year-old male patient reported a strong pain in his left shoulder with marked muscle cramps after lifting his child. Clinical examination showed full range of motion. Jobe and Patte tests were not painful, although with a distinct strength reduction in side comparison. The patient presented a Quick Disabilities of Arm, Shoulder and Hand questionnaire score of 16 and a Constant score of 83 points for the left shoulder.

Electromyography showed a distinct rarefaction of the discharge pattern (Fig. 1, *a*). Electroneurography revealed a significant latency delay of the SSP and ISP. A subsequent MRI of the left shoulder showed edematous changes of the SSP and ISP (Fig. 2, *a*). At this time, a compression syndrome of the SSN was most likely. In the case of compression, the arthroscopic decompression of the SSN in the suprascapular notch would be recommended. However, to rule out differential diagnosis, that is, a traction injury or an idiopathic neuritis, an MR neurography of the brachial plexus was performed.

MR neurography confirmed the denervation edema in the SSP and ISP muscles (Fig. 3, *c*) and showed normal nerve signal in the proximal cross section of the brachial plexus and the SSN exiting from the superior trunk (TS) (Fig. 3, *a*). Tracing the further course of the SSN reveals a short increase of caliber and T2 signal intensity of the SSN proximal to the suprascapular notch but preserved continuity of the nerve sheath (Fig. 3, *b*). No sign of entrapment within the suprascapular notch by the transverse scapular ligament could be detected. In accordance with the described injury mechanism and the immediate onset of pain, this monofocal nerve lesion indicated a shear or traction injury of the SSN.

As the lesion was associated with just a moderate-caliber increase of the SSN, the findings suggested a sufficient spontaneous regeneration throughout. The suspected compression syndrome could be disproved. We withdrew indication for

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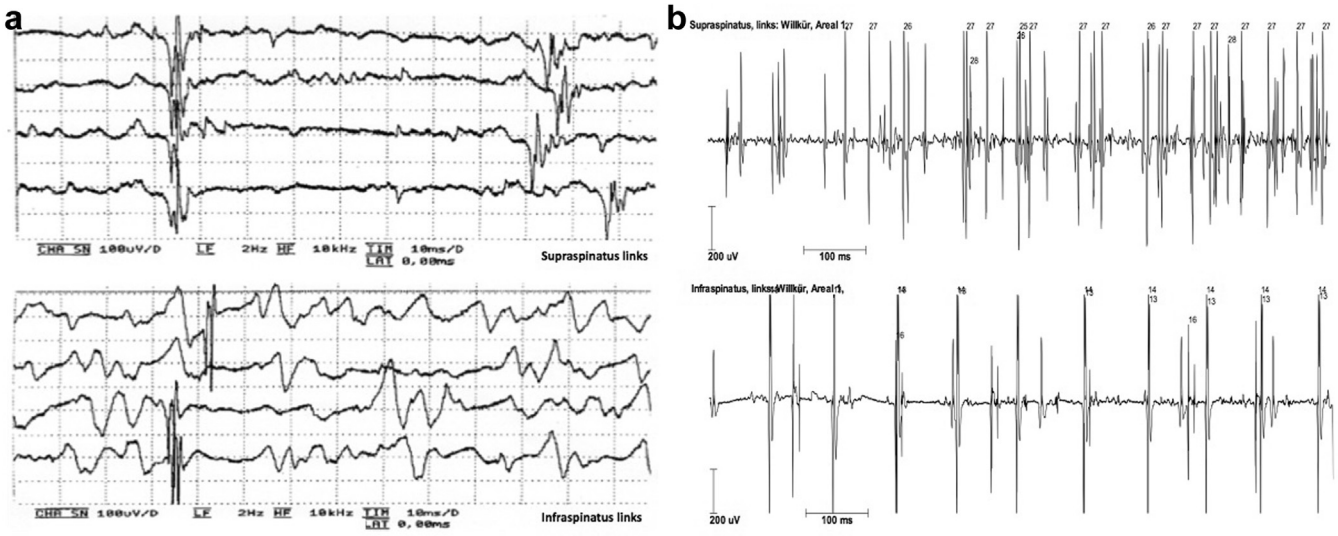

Figure 1 (**a**) Initial electromyography (EMG) with a distinct rarefaction of the discharge pattern and signs of a neurogenic remodeling. (**b**) EMG 1 year after trauma with a normalization of the discharge pattern and only minor chronic neurogenic changes.

decompression surgery. Instead, we went for symptomatic treatment, that is, manual therapy and physiotherapy among demand-oriented analgesia.

Follow-up MRIs at 3 months and subsequently 1 year after injury revealed waning muscular edema of the SSP and ISP with minor residual findings after 3 months and remission after 1 year (Fig. 2, *b*). Electromyography showed a normalization of the discharge pattern, with only minor chronic neurogenic changes 1 year post-trauma (Fig. 1, *b*). Moreover, clinical examination showed an improvement in abduction and external rotation strength. Quick Disabilities of Arm, Shoulder and Hand questionnaire score revealed an improvement to 7.5 points, and Constant score showed 97 points for the left shoulder. A follow-up MR neurography after 1 year showed continued signal enhancement of the suprascapular nerve and residual signal alterations in the primarily heavily affected SSP and ISP muscles (Fig. 3, *d*), underlining the profound functional improvement with a slight residual deficit.

Discussion

Although the number of articles referring to suprascapular neuropathy has increased during the recent past, the incidence and prevalence of suprascapular neuropathy remain widely unknown.[3] The most recent meta-analysis, reviewing the literature between 1959 and 2001, found only 88 cases with suprascapular neuropathy.[15]

Common causes for a suprascapular neuropathy are repetitive overhead activities, traction from a rotator cuff tear, and compression from space-occupying lesions at the spinoglenoidal and the suprascapular notch (Table I).[3,6] Repetitive microtraumata may cause a direct injury to the nerve or an indirect injury by affecting the vascular bundle to the nerve.[12] However, isolated injuries of the SSN can also occur with fractures of the proximal humerus, scapula, and in shoulder dislocations.[12]

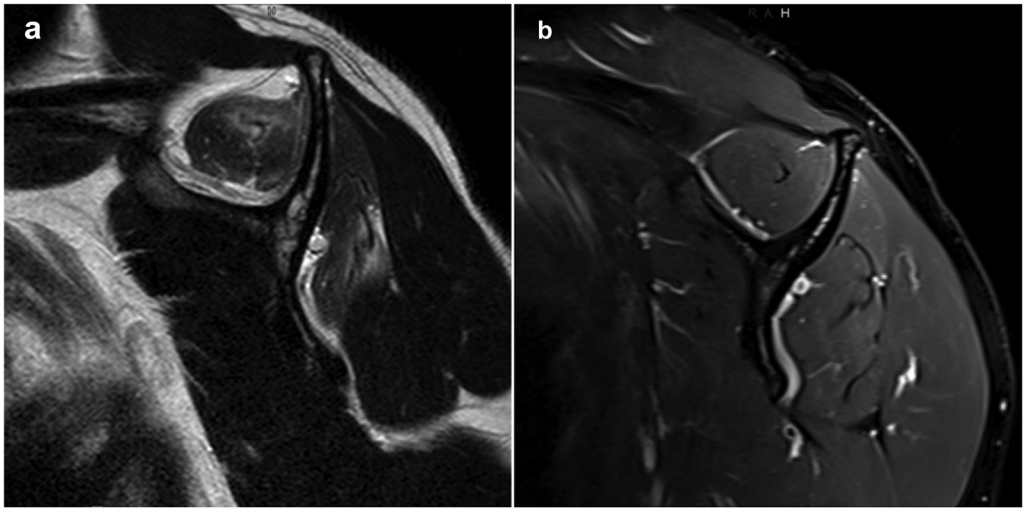

Figure 2 Magnetic resonance imaging (MRI), T2 turbo-spin echo sagittal (**a**) without fat suppression; edematous changes of the supraspinatus (SSP) and infraspinatus (ISP) muscle (**b**) with fat suppression. One year after trauma, the edematous changes dissipated.

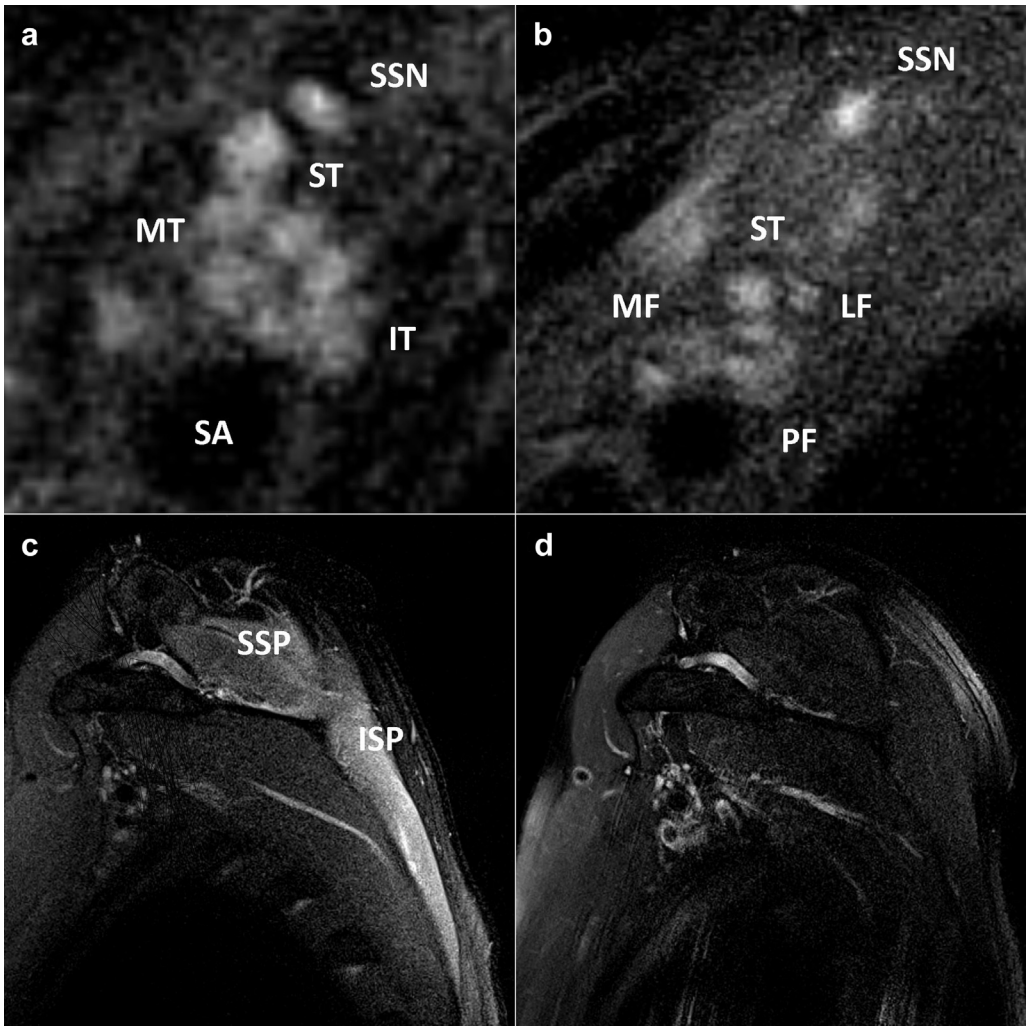

Figure 3 Magnetic resonance (MR) neurography fat-saturated sagittal oblique T2 turbo-spin echo sequences (repetition time/echo time 5300/54 ms slice thickness 4 mm) of the brachial plexus. (**a**) Regular nerve-signal in the supraclavicular section of the brachial plexus; the anterior and posterior divisions of superior, median, and inferior trunci (ST, MT, IT); subclavian artery (SA); and the exit of the suprascapular nerve (SSN) from the ST. (**b**) T2-hyperintense lesion site in SSN in the distal slab. The regular signal of the plexus further distally depicts the transition of the trunci into the median, lateral, and posterior fascicles (MF, LF, PF). (**c, d**) Imaging of the supraspinatus (SSP) and infraspinatus (ISP) muscle (**c**) initially and in (**d**) follow-up after 1 year shows a complete remission of denervation edema.

Injury mechanism

Reports of SSN mononeuropathies are rare in literature. Kowalczuk et al[8] reported a case of suprascapular mononeuropathy caused by a nondisplaced scapular fracture. A football player suffered a nondisplaced fracture of the scapular body and spine extending into the base of the suprascapular notch that resulted in an isolated mononeuropathy of the SSN proximal to the suprascapular ligament.[8] In contrast, the neuropathy in the present case was caused by an indirect trauma. It is, to our knowledge, the first report to describe an isolated mononeuropathy of the SSN by a traction injury mechanism.

Imaging technique

MR neurography is a specialized MRI technique, mainly based on high-resolution fat-saturated T2- or proton density–weighted sequences,[7] that reveals nerve lesions down to the fascicular level as T2 signal enhancement at a magnetic field strength of 3 tesla. It allows a spatial resolution down to 200 μm[5] and reveals muscular denervation signs as early as 24 hours after nerve trauma.[2] This enables the radiologist to detect peripheral nerve lesions with a high sensitivity.[4]

The lesion pattern shown by MR neurography is especially helpful in the differential diagnostics of traumatic nerve injury,

Table I
Overview of the different locations and pathogenesis of nerve compression of the SSN and its branches[13]

Topography	Cause	Atrophy
Suprascapular notch	Chronic compression due to a hypertrophic transverse scapular ligament or bony narrowness	SSP and ISP
Spinoglenoidal notch	Repetitive traction or chronic compression due to a ganglion or hypertrophic spinoglenoid ligament	ISP
Scapular body	Compression/infiltration due to a tumor	SSP and/or ISP
Scapular body	Compression due to a ganglion	ISP
Inferior glenoid	Bennett lesion	ISP

SSN, Suprascapular nerve; *SSP*, supraspinatus; *ISP*, infraspinatus.

nerve compression syndromes, and immunogenic or metabolically associated nerve damage,[1,10,11] which, as in the present case, is crucial in therapeutic decision making.[14]

In most cases, a careful medical history, clinical examination, and standard MRI are sufficient to determine diagnosis. However, in case of doubtful findings in relation to neurologic pathologies, especially before indicating nerve decompression surgery, MR neurography may offer crucial information to confirm or withdraw the diagnosis. We recommend the implementation of an MR neurography only in cases of a doubtful medical history in the context of an expedient and economic approach.

Conclusion

Before an arthroscopic decompression at the suprascapular notch for treatment of a compression syndrome of the SSN, a traction-related neuropathy of the nerve should be excluded in differential diagnosis. In the case of a doubtful medical history, the presented MR neurography is recommended.

Disclaimer

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