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MUSCULOSKELETAL

Diagnostic Signs of Motor Neuropathy in MR Neurography: Nerve Lesions and Muscle Denervation

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Abstract
Objective To investigate the diagnostic contribution of T2-w nerve lesions and of muscle denervation in peripheral motor neuropathies by magnetic resonance neurography (MRN).
Methods Fifty-one patients with peripheral motor neuropathies underwent high-resolution MRN by large coverage axial T2-w sequences of the upper arm, elbow, and forearm. Images were evaluated by two blinded readers for T2-w signal alterations of median, ulnar, and radial nerves, and for denervation in respective target muscle groups.
Results All 51 patients displayed nerve lesions in at least one of three nerves, and 43 out of 51 patients showed denervation in at least one target muscle group of these nerves. In 21 out of 51 patients, the number of affected nerves matched the number of affected target muscle groups. In the remaining 30 patients, T2-w lesions were encountered more frequently than target muscle group denervation. In 153 nerve-muscle pairs, 72 showed denervation, but only one had increased muscle signal without a lesion in the corresponding nerve.
Conclusions MRN-based diagnosis of peripheral motor neuropathies is more likely by visualization of peripheral nerve lesions than by denervation in corresponding target muscles. Increased muscular T2-w signal without concomitant nerve lesions should raise suspicion of an etiology other than peripheral neuropathy.
Key Points
- *In peripheral neuropathy, T2-w nerve lesions are more frequent than muscle denervation.*
- *Muscle denervation almost never occurs without detectable lesions in corresponding nerves.*
- *MRN-aided diagnosis of peripheral motor neuropathy should focus primarily on nerve lesions.*
- *Increased muscular T2-w signal intensity without concomitant nerve lesions indicates other aetiology.*

Keywords MR neurography · Polyneuropathy · MRI · Muscle denervation · Nerve lesion

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Introduction

In peripheral neuropathies with involvement of motor fascicles, denervation of affected muscles is displayed by a pathological increase in the T2-weighted (T2-w) signal intensity [1], which is attributed to increased levels of extracellular fluid [2, 3]. The pattern of muscle denervation has long been used as an indirect sign for the diagnosis of peripheral motor neuropathy by magnetic resonance imaging (MRI) [4–7]. High-resolution magnetic resonance neurography (MRN) now allows for direct depiction of nerve lesions [8–12]. These lesions of different aetiologies all present with an increased T2-w signal as a sensitive albeit unspecific sign of neuropathy,

and can be detected with high precision by visual assessment of T2-w images [8, 10, 13–16].

Patients with an initial diagnosis of peripheral neuropathy with involvement of motor fascicles and muscle paresis are frequently referred for MRN examination for diagnostic confirmation by imaging. This procedure can be a useful complement to clinical examination and electrophysiological testing, as MRN may better determine the localization and extent of nerve lesions [16] and may also detect lesions in clinically unaffected nerves [17]. Further, cross-sections of forearm musculature contain the majority of muscles innervated by median, ulnar, and radial nerves, whereas electromyography (EMG) is invasive and can examine only a limited number of muscles.

If muscle denervation is due to peripheral neuropathy, lesions should be expected to occur in a nerve before the resulting denervation in its respective target muscles. Therefore, nerve lesions on MRN might be expected to be a more sensitive sign of peripheral neuropathy with motor involvement. In electrophysiological studies, however, the contrary is the case: EMG is often a more sensitive parameter than nerve conduction studies (NCS) [18–21]. In denervated muscle, EMG findings closely correspond to the increased T2-w signal on MRI [22]. By analogy, it may also be possible in MRN that muscle denervation represents a more sensitive sign of motor neuropathy than the presence of nerve lesions.

In this study, we aimed to address this question by testing whether nerve lesions or muscle denervation patterns were more frequently detected in patients with peripheral neuropathy with involvement of motor axons.

Materials and methods

Clinical and demographic patient data

The study was approved by the institutional ethics board (University of Heidelberg Ethics Committee; S-057/2009), and written informed consent was obtained from all participants. Patients were examined at the Department of Neuroradiology of Heidelberg University Hospital in Germany between January 2012 and April 2013, at least four weeks after onset of symptoms. Patients were included in our study based on a clinical and electrophysiological diagnosis of peripheral motor neuropathy, i.e., clinically diagnosed weakness, atrophy, and/or positive electrophysiological findings suspicious of motor neuropathy. Exclusion criteria were (previous or subsequent) diagnosis of a disease other than peripheral neuropathy as a possible cause of muscle weakness, e.g., spinal canal stenosis, myopathy, or motor neuron disease. Furthermore, the typical compressive neuropathies of carpal tunnel syndrome and ulnar neuropathy at the elbow were excluded, as the protocol did not systematically include coverage of hand muscles. Sensory co-involvement was not an exclusion criterion, since most peripheral neuropathies affect both motor and sensory fascicles. A total of 51 patients fulfilled the criteria and were included in the study.

MRN imaging

Examinations were conducted using a 3 Tesla unit (MAGNETOM Verio, Siemens AG, Erlangen, Germany). Subjects were examined in the prone position with the arm extended at the upper arm, the elbow, and the forearm, placed in a knee 8-channel phased array coil. Coverage comprised the upper arm, the elbow region, and the forearm in two image slabs. A third image slab was acquired in 24 of 51 cases because the forearm musculature was not sufficiently covered by the second image slab. If the brachial plexus, distal forearm, or wrist were also imaged as part of the clinical exam protocol, these additional images were not included in our analysis. To avoid any significant artificial signal increase in a T2-w sequence related to the so-called magic angle effect at the elbow [23], the longitudinal axis of the upper arm was aligned at an angle of ≤10° relative to the B0 field direction.

The sequence parameters were as follows: transversal T2-w turbo spin-echo TR/TE 7,020/52 ms, spectral fat saturation, parallel imaging (GRAPPA 2, reference lines PE 32), slice thickness 3.0 mm, number of slices 45, interslice gap 0.3 mm, FoV 130×130 mm^2, acquisition matrix 512×358, pixel spacing 0.254×0.254 mm^2, number of excitations=3, acquisition time 7:17 min each sequence, resulting in a total acquisition time of 21:51 min.

Image analysis

Qualitative evaluation in a proximodistal direction of nerve and muscle T2-w signal from patients was performed independently by two neuroradiologists (DS, PB), who were blinded to the patients' clinical data. Standardized rating was performed in a dichotomous fashion, either as "affected" or as "non-affected," with regard to the following items:

1. Presence of peripheral nerve lesions in the a) median nerve, b) ulnar nerve, and c) radial nerve at the upper arm and elbow level.
2. Presence of muscle denervation in any muscle within the target muscle groups (according to [4]) of a) median nerve (Mm. pronator teres, flexor carpi radialis, palmaris longus, flexor digitorum superficialis, flexor digitorum profundus 2nd and 3rd, flexor pollicis longus and pronator quadratus), b) ulnar nerve (Mm. flexor carpi ulnaris and flexor digitorum profundus 4th and 5th), and c) radial nerve (Mm. triceps brachii, anconeus, brachioradialis, extensor carpi radialis longus and brevis, supinator, extensor carpi ulnaris, extensor digitorum, extensor digiti

minimi, abductor pollicis longus, extensor pollicis longus and brevis, and extensor indicis).

Rating was performed from proximal to distal (Fig. 1). Cohen's κ, a statistical measure of inter-rater agreement, was calculated for the evaluation of inter-rater agreement. For further statistical analysis of the results of the ratings, final classification in the case of disagreement between the raters was achieved by a second, consensus-based evaluation.

Additional quantitative analysis

In order to objectify the qualitative ratings of nerves, quantitative analysis of T2-w signals from nerves was performed by evaluation of the mean T2-w signal from a representative intraneural region-of-interest (nROI) of the three nerves (median, ulnar, radial). For nerves with qualitatively detectable nerve lesions, this was placed at a location representative for the lesion. For normal-appearing nerves, it was placed in a standard position of the unaffected nerve in the mid-portion of the upper arm (12 cm proximal to the humeroradial joint). Additional regions of interest were placed within adjacent non-denervated muscle (mROI), and the mean signal intensities were read out in order to calculate the normalized T2-w signal intensity, abbreviated normT2 (normT2=normROI /mROI).

Statistical analysis

Based on qualitative ratings, two different categorizations were performed.

1. Patients were grouped into one of the following three categories (Fig. 2):
    1. Patients with a greater number of affected nerves than target muscle groups
    2. Patients with the same number of affected nerves and target muscle groups
    3. Patients with a greater number of affected target muscle groups than nerves
2. Classification of all three evaluated arm nerves and their corresponding target muscle groups in all 51 patients, resulting in 153 nerve-muscle pairs. Every nerve-muscle pair was likewise grouped into one of three categories:
    1) Nerve lesion without concomitant muscle denervation in its target muscle group
    2) Nerve lesion with concomitant muscle denervation in its target muscle group
    3) No nerve lesion present but presence of muscle denervation in its target muscle group

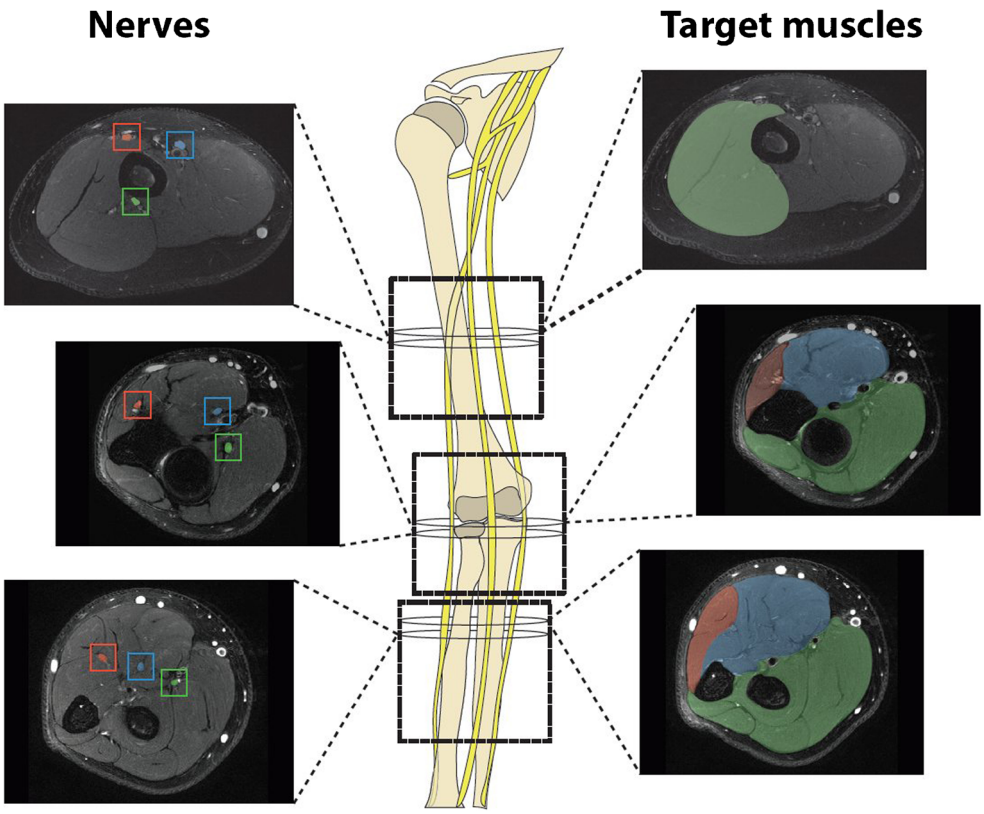

Fig. 1 Schematic of the imaging rationale. The left column shows a representative section of each image slab and the three assessed peripheral nerves of the upper extremity, coloured according to their identity (*blue=median*, *red=ulnar*, *green=radial*). Similarly, the right column shows the target muscle groups of each of the three nerves (*same colour code*). The location of the displayed images is indicated on the schematic of the upper extremity and the three nerves in the middle column

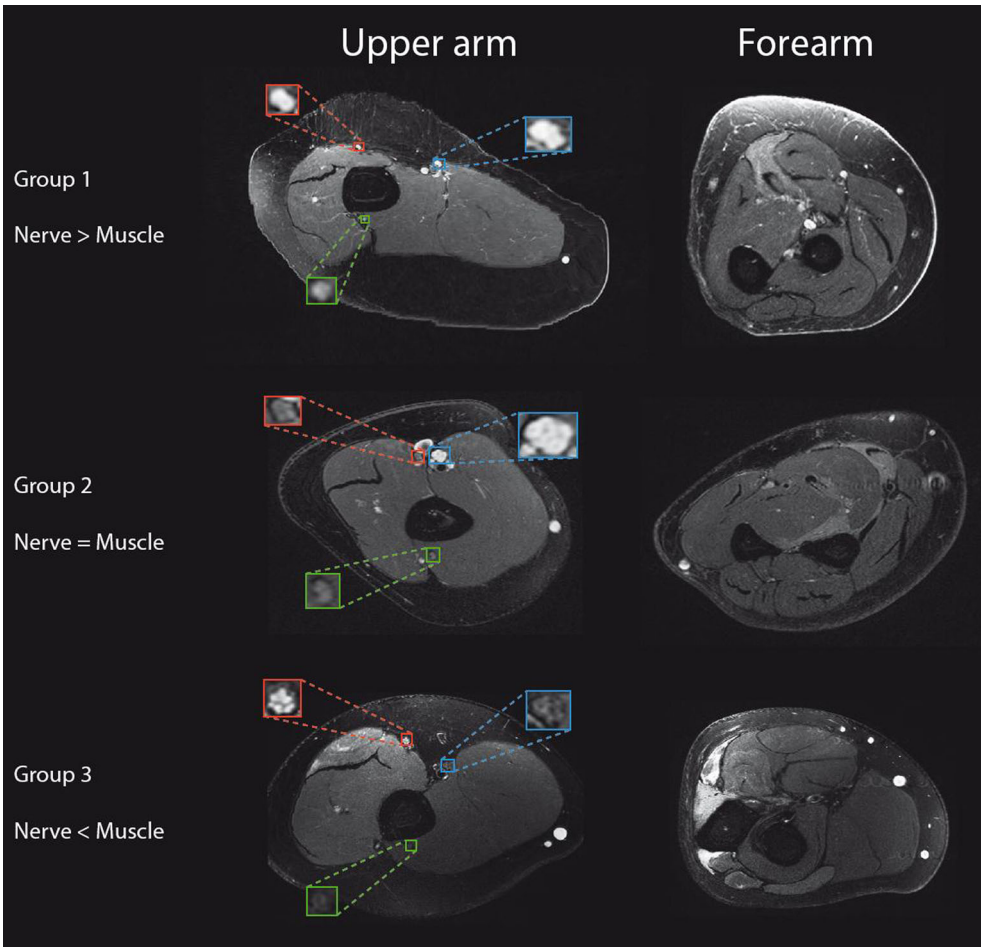

Fig. 2 Classification of MRN findings. The upper panel shows a representative finding of an imaging case from group 1 exhibiting pathologic lesions in all three nerves (blue=median, red=ulnar, green=radial) while denervation only becomes evident in the median innervation territory (M. flexor digitorum superficialis). In the middle panel, a typical case from group 2 with pathologic T2-weighted signal elevation in the median nerve (*blue*) and the median-dependent target muscle group (Mm. flexor digitorum profundus and superficialis, flexor pollicis longus, flexor carpi radialis and palmaris longus). A case exhibiting affection of more target muscle groups than nerves (group 3) is shown in the lower panel. Here, pathologic T2-weigthed signal alterations are visible in all three target muscle groups, while nerve lesions can only be detected in the ulnar nerve. This patient was excluded, however, because he was later diagnosed with spinal canal stenosis

Results of the quantitative analysis were plotted by graphs mapping normT2 values comparing affected and non-affected using MATLAB (MathWorks, release 2013a).

Results

All 51 patients exhibited nerve T2-w lesions in at least one of the three nerves evaluated. In contrast, muscle denervation at the upper arm or forearm level was detected in only 43 of 51 patients (84.3 %). Inter-rater agreement was high, with an overall Cohen's κ of 0.92 (Table 1).

In 30 of 51 patients (58.8 %), T2-w signal alterations were detectable in a greater number of nerves than target muscle groups. In the other 21 patients (41.2 %), the number of affected nerves matched the number of target muscle groups with denervation (Fig. 3). There were no cases of a lower number of affected nerves than target muscle groups containing denervated muscles.

Similar results were obtained when comparing individual nerves and their respective muscular innervation territory. Lesions of the median, ulnar, and radial nerves were found in 44, 45, and 29 cases, respectively, while muscle denervation in target muscle groups could only be detected in 30, 21, and 20 cases (Fig. 4). Of these total 153 nerve-muscle pairs, 47 (30.7 %) showed nerve lesions but no concomitant muscle denervation, 71 (46.4 %) exhibited both nerve lesions and corresponding muscle denervation, and only one pair (0.7 %) was found to have an increased T2-w muscular signal without a lesion in the corresponding nerve. In this case, a more proximal nerve lesion was found in the plexus, outside the study protocol. The remaining 34 pairs (22.2 %) had both normal nerve and muscle appearance.

To strengthen the qualitative assessment of nerve lesions, normalized T2-w signal intensity values from representative regions of interest of "affected" and "non-affected" arm nerves were compared (Fig. 5). A separation between pathological and normal nerves was observed at normT2 values of 1.69 for the median nerve, 1.79 for the ulnar nerve, and 1.80 for the radial nerve.

Table 1 Rater performance and inter-rater variability. The upper panel shows the absolute number of ratings (either positive or negative) among all 51 patients. The first three columns indicate the numbers individually for the three nerves of the upper extremity, and the next three columns show the numbers for the individual target muscle groups. The lower panel shows the two individual components for the calculation of Cohen's κ: Pr(α), the observed percentage agreement; and Pr(ε), the probability of random agreement. The last row gives the values of Cohen's κ for the three nerves and the three target muscle groups separately and combined

	Median	Ulnar	Radial	Median_musc	Ulnar_musc	Radial_musc	Total
Rater 1 positive	36	41	24	25	17	18	
Rater 2 positive	39	39	24	25	19	18	
Rater 1 negative	10	5	22	21	29	28	
Rater 2 negative	7	7	22	21	27	28	
Pr(α)	0.90	0.96	0.96	1.00	0.96	1.00	0.96
Pr(ε)	0.72	0.79	0.50	0.52	0.52	0.52	0.52
Cohen's κ	0.65	0.81	0.92	1.00	0.92	1.00	0.92

Discussion

Diagnosis of peripheral neuropathy on MRN is based on signal alterations in affected nerves and muscles. In the present study, we showed that in patients with a clinical diagnosis of peripheral motor neuropathy, abnormal signal intensity on T2-w imaging was almost always found in at least one peripheral nerve, whereas corresponding muscle denervation was found less frequently.

It has long been the general perception that specific patterns of muscular denervation are the major hallmark for MRI diagnosis of peripheral motor neuropathies [4–7, 24]. Similarly, in electrophysiology, EMG is often more sensitive than NCS in demonstrating abnormalities in peripheral motor disease, due to technical reasons [18–21]. Our results for MR image analysis help to shift the diagnostic focus from muscular denervation to nerve lesions, and thereby have relevant implications for both MRN protocols and image evaluation.

As a consequence, for sequence protocols, MRN exams for clinically suspected peripheral neuropathy should focus primarily on direct detection of nerve lesions. Detection is more likely in regions where nerve trunks still contain all or most of their fascicles, i.e., the upper arm rather than the forearm [16, 17]. Determining the extent of peripheral nerves affected by a neuropathy is highly relevant in the classification and subsequent treatment of neuropathic disorders [25]. Visualizing denervation in target muscle groups confirms clinical motor affection but may not necessarily reflect the actual extent of peripheral nerve involvement.

A fundamental implication for image evaluation is that increased muscle T2-w signal intensity indicative of

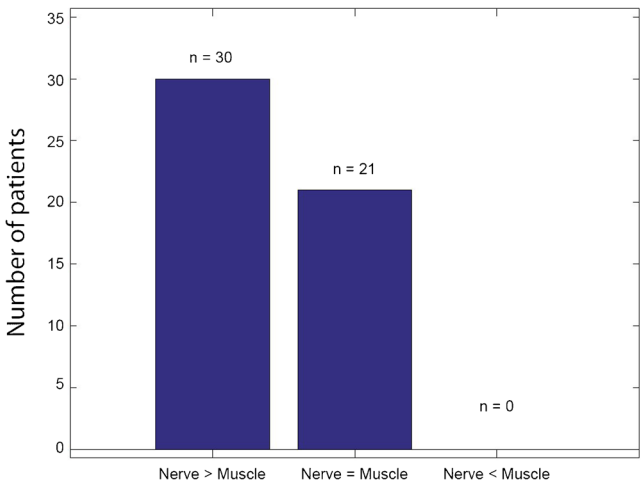

Fig. 3 Qualitative assessment of pathological MRN findings. Diagram illustrating the number of patients in each of the three groups. In the majority of cases, affection of nerves was encountered more frequently than denervation in the corresponding target muscle groups. There were no cases showing affection of target muscles without an appreciable lesion in the nerve

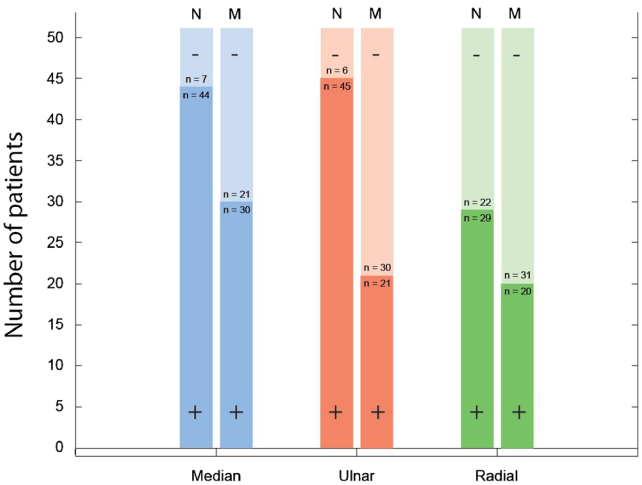

Fig. 4 Qualitative assessment of individual nerves and their respective target muscle groups. Separate assessment of the three nerves (N) and their respective target muscle groups (M) shows that detection of nerve lesions exceeds positive image findings in the target muscle groups. Among the three nerves of the upper extremity, the discrepancy between findings in nerve and target muscles is most pronounced for the ulnar nerve

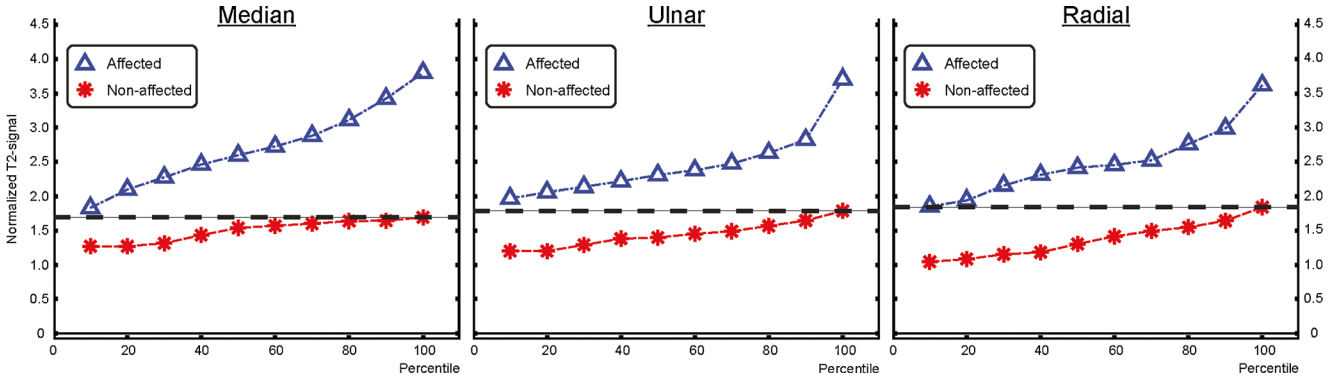

Fig. 5 Quantitative evaluation of qualitatively assessed nerve lesions. Normalized T2-weighted signal values (normT2) were calculated for each of the three nerves and plotted in a percentage-wise fashion from lowest to highest signal intensity. Consistent with our qualitative assessment, normT2 values differed between "affected" and "non-affected" nerves. Most interestingly, we found similar cutoff values distinguishing the "affected" and "non-affected" categories for all three nerves: median=1.69, ulnar=1.79, radial=1.8

denervation is almost always associated with T2-w nerve lesions in peripheral neuropathies with involvement of motor fascicles. If such corresponding peripheral nerve lesions cannot be detected but pathologically increased T2-w signal in the musculature is present, underlying pathologies other than a peripheral neuropathic aetiology should be considered and carefully excluded.

How can the observed discrepancy between neural and muscular T2-w signal alterations be explained? One important aspect is that MR morphologic evidence of muscular denervation may represent a threshold phenomenon that is dependent on sufficiently severe axonal damage. In contrast, in the early stages of motor dominant neuropathy, T2-w hyperintense nerve signals may be caused simply by demyelination, and not necessarily accompanied by axonal damage. Further, the development of T2-w signal alterations is likely to follow a different time course in nerves versus muscles. While MRN exams in our study were all performed after clinical and electrophysiological examination, it may still have been too early in some cases for the development of muscular denervation in the innervation territory of all pathologic nerves.

A previous study examined the time course of muscular T2-w signal patterns after traumatic nerve lesions, and found good correlation with clinical recovery [26]. For less acute neuropathies, further investigation is needed with regard to the exact onset of symptoms and correlation with electrophysiology, although a study in the lower extremity found that the sensitivity of increased muscular T2-w signal intensity for denervation remained high regardless of duration, location, or severity of the neuropathy [27].

A number of limitations apply to this study. Image acquisition was not systematically paralleled by electrophysiological testing objectifying MRN images. However, studies have abundantly demonstrated the correlation of T2-w nerve lesions and pathological findings in electrophysiological exams. Second, the majority of patients included in the study were diagnosed with an inflammatory neuropathy, so the group is not completely representative of all patients examined by MRN. However, this is the group for whom these findings are most relevant, since assessment of the extent of disease is more difficult here than in neuropathies of compressive or neoplastic aetiology. Another limiting factor was that the hand region containing the distal target muscles of the median and ulnar nerves [4] was not routinely included in the standard analysis in our protocol. To minimize the effect of systematically underestimating the proportion of affected target muscle groups, we explicitly excluded cases with compressive neuropathies, as their denervation occurs in distal target muscles (e.g., carpal tunnel syndrome and ulnar neuropathy at the elbow). More importantly, for the radial nerve, the entire muscular innervation territory was systematically covered by our examination protocol, and radial nerve lesions were still found more frequently than radial nerve denervation. Finally, we used transversal 2D T2-w sequences for large coverage. The use of 3D T2-w, with its three-dimensional reconstruction possibilities, may further improve diagnostic accuracy for both nerves and muscles [28].

Taken together, our results suggest that peripheral neuropathy with involvement of motor fascicles is more frequently confirmed by detection of peripheral nerve lesions than denervation in the corresponding target muscle groups. Further, pathological muscle signals without concomitant nerve lesions should raise suspicion of an aetiology other than peripheral neuropathy.

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