NEUROIMAGING OF CLASSIC NEURALGIC AMYOTROPHY

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ABSTRACT: *Introduction*: Neuralgic amyotrophy (NA) often imposes diagnostic problems. Recently, MRI and high-resolution ultrasound (HRUS) have proven useful in diagnosing peripheral nerve disorders. *Methods*: We performed a chart and imaging review of patients who were examined using neuroimaging and who were referred because of clinically diagnosed NA between March 1, 2014 and May 1, 2015. *Results*: Six patients were included. All underwent HRUS, and 5 underwent MRI. Time from onset to evaluation ranged from 2 weeks to 6 months. HRUS showed segmental swelling of all clinically affected nerves/trunks. Atrophy of muscles was detected in those assessed >1 month after onset. MRI showed T2-weighted hyperintensity in all clinically affected nerves, except for the long thoracic nerve, and denervation edema of muscles. *Conclusions*: HRUS and MRI are valuable diagnostic tools in NA. This could change the diagnostic approach from one now focused on excluding other disorders to confirming NA through imaging markers.

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Neuralgic amyotrophy (NA), Parsonage-Turner syndrome, and (brachial) plexus neuritis are different names for the same condition. It usually starts with acute onset of excruciating pain in the shoulder region, that, when it subsides often evolves into severe paresis that affects predominantly proximal muscles.[1]

Diagnosis is traditionally delayed,[2] as the intense, localized pain is often attributed to a musculoskeletal origin, and, only when atrophy becomes visible, patients make their way to the neurologist. Therefore, most patients are seen at a late stage, at which time therapy with corticosteroids is generally not considered successful, and rehabilitation is the only option. This is of interest, as NA leaves half to two-thirds of patients with disability and pain over the years[3,4] and its prevalence has been underestimated by 30- to 50-fold according to a recent study that found not 2–3 cases per 100,000 in the general population per year but 1 per 1,000.[5]

NA can affect almost every nerve, but most frequently involvement of the upper trunk, the long thoracic nerve (LTN), and the suprascapular nerve (SCN) are encountered.[2]

Diagnosis is usually based on medical history, clinical examination, and sometimes ancillary investigations, such as MRI of the cervical spine or lumbar puncture to exclude other causes. Electrodiagnostic studies can be of value, as they are the only examination that can support the clinical diagnosis. Neurography may, however, show subclinical sensory involvement with axonal damage in a small proportion of patients and, therefore, exclude radicular compression as the sole cause of symptoms.[6] Motor conduction studies can support clinical findings by demonstrating axonal damage, but only in severely paretic muscles. Electromyography can provide information about the extent of clinical lesions and confirm a multifocal distribution.[7] Currently, no standardized morphologic confirmatory test for NA has been proposed.

In the last several years, technical advances in MRI and ultrasound have enabled improved visualization and assessment of peripheral nerves. The introduction of higher field strength (3 Tesla [T]) MRI substantially improved the ability to detect nerve signal changes associated with various nerve pathologies, including inflammation. In some but not all cases, gadolinium enhancement helps to establish the diagnosis of neuritis. Moreover, MRI is particularly valuable for visualizing muscle denervation patterns associated with NA.[8,9] However, due to the limited field of view at a given resolution, only certain segments of the peripheral nerves can be depicted in detail, which can lead to false negative results. Ultrasound may be an alternative, because high-resolution probes of 15 MHz and above offer sufficient spatial resolution to judge morphology and, therefore, pathology of nerves as small as <1 mm diameter. This would include the nerves most frequently affected in NA.

Here, we present findings from HRUS and MRI in a series of patients with typical NA in the acute to subacute phase.

MATERIALS AND METHODS

Patients. The nerve ultrasound quality assurance database of the Department of Radiology was scanned for patients referred for suspicion of NA who were evaluated between March 1, 2014 and May 1, 2015.

Abbreviations: CSA, cross-sectional area; CSD, cross-sectional diameter; CSF, cerebrospinal fluid; EMG, electromyography; HRUS, high-resolution ultrasound; LTN, long thoracic nerve; NA, neuralgic amyotrophy; SCN, suprascapular nerve; TE, echo time; THRIVE, T1 high-resolution isotropic volume excitation; TR, repetition time

Key words: all pain; autoimmune diseases; brachial plexus neuritis; neuralgic amyotrophy; Parsonage-Turner syndrome; peripheral neuropathy; postinfectious; ultrasound

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Table 1. Clinical summary and MRI findings in 6 patients with a diagnosis of NA.

Patient no.	Age	Gender	Side	Duration (weeks)	Clinically affected nerve/plexus part	MRI
1	20	M	R	6	Long thoracic nerve	Nerve unremarkable, muscle not imaged
2	50	W	R	10	Long thoracic nerve	Nerve unremarkable, muscle not imaged
3	49	M	L	3	Suprascapular nerve	Hyperintense T2 signal and contrast enhancement
					Upper trunk	Contrast enhancement of C5 and C6, denervation edema of deltoid, infra- and supraspinatus muscles
4	34	M	L	2	Upper trunk	Hyperintense signal and contrast enhancement of C5 and C6 root /upper trunk, denervation edema of supraspinatus, infraspinatus, and deltoid muscles
					Fascicle of median nerve	Not imaged
					Fascicle of musculocutaneous nerve	Not imaged
			R	*	Long thoracic nerve	Not imaged
5	48	M	R	8	Deep branch of radial nerve	Not performed
6	49	W	L	26	Fascicle of median nerve	Hyperintense T2 signal of median nerve, muscles not imaged

**Not symptomatic at first assessment, became clinically symptomatic 2 weeks later.*

This study was approved by the local ethics committee, which waived the need for informed consent for this retrospective analysis of data.

Ultrasound. Ultrasound was performed by a radiologist with more than 20 years of experience (G.B.) and a neurologist with more than 3 years of experience (D.L.S.) in peripheral nerve high-resolution ultrasound (HRUS). Examinations were carried out with the GE logic E9 platform using probes with frequencies of 12 to 18 MHz. Examiners were not blinded to the patient diagnoses.

Evaluation typically began with assessment of the brachial plexus as a whole, then clinically affected nerves were examined in detail within their visible course, and finally corresponding muscles were assessed for atrophy based on a side-to-side comparison of diameter combined with increased echogenicity. Findings were documented in the transverse and longitudinal views on still images with video clips over altered nerve sections. Furthermore, the cross-sectional diameters (CSD) of the affected nerve segments or fascicles were assessed, and in the larger nerves of the upper extremity cross-sectional area (CSA) was also noted.

MRI. The 3T MRI scans (Philips Achieva, Best, The Netherlands) were performed in 5 of 6 patients using a standardized and dedicated brachial plexus imaging protocol. A neurovascular coil was used, and the patient was imaged in the supine position. The protocol consisted of a sagittal T2TSE sequence covering the cervical spine (repetition time [TR] = 2487, echo time [TE] = 120 reconstructed voxel size 0.49/0.49/3.5 mm), an axial STIR sequence covering both shoulders and the neck area (TR = 4718TE60, reconstructed voxel size 0.58/0.58/4.0 mm), and a paracoronal high-resolution STIR sequence (TR = 2802, TE = 180, reconstructed voxel size 0.56/0.56/2.70 mm) in the orientation of the ventral nerve roots of the brachial plexus (slice thickness 2.5 mm). In addition, a paracoronal T1-weighted sequence was acquired before and after administration of a gadolinium-based contrast agent. An axial THRIVE (T1 High-Resolution Isotropic Volume Excitation) sequence (with fat saturation) was acquired after administration of a gadolinium-based contrast agent in the axial plane (TR = 8.2, TE = 3.7; reconstructed voxel size 0.35/0.35/1 mm).

RESULTS

Six patients were identified. All fulfilled the criteria for a diagnosis of NA as suggested by the most recent and comprehensive study on NA incidence, namely (1) acute onset of very severe (numerical rating scale $\geq$ 7/10), analgesic-resistant shoulder and/or upper arm pain, often with worsening at night; and (2) multifocal peripheral nervous system symptoms and signs that could be bilateral but asymmetric.[5] Time from onset of symptoms to evaluation ranged from 2 weeks to 6 months. Two patients presented with a winged scapula, 2 with anterior interosseus nerve syndrome, 1 with weakness in finger extensors, and 1 with weakness in external rotation and arm elevation. To provide more detailed insight, we describe the patients individually rather than as a whole group. Please find descriptions below. An overview of clinical and neuroimaging findings is given in Tables 1 and 2.

Patient 1. A 20-year-old man experienced sudden onset intense pain during the night in the right shoulder and periscapular region. The pain subsided within 10 days under therapy with nonsteroidal anti-inflammatory drugs. He was seen 2 months later when he noticed persistent weakness in maximal elevation of the right arm and recurring pain in the right shoulder that was less intense than 2 months

Table 2. HRUS findings in 6 patients with a diagnosis of NA.

Patient no.	Clinically affected nerve/plexus part	CSD (mm) affected/ unaffected side	Muscle	CSD (mm) affected/ unaffected side
1	Long thoracic nerve	1.8 / 1.2	Serratus anterior	4 / 6
2	Long thoracic nerve	1.9 / 1.3	Serratus anterior	1 / 3
3	Suprascapular nerve	2.2 / 1.5	Unremarkable	
	Upper trunk	6.0 / 3.8	Unremarkable	
4	Upper trunk	7.0/3.5	Unremarkable	
	Fascicle of median nerve	3.1 / 0.8*	Unremarkable	
	Fascicle of musculocutaneous nerve	2.1 / 1.5*	Unremarkable	
	Long thoracic nerve†	3.5 / 1.5	Unremarkable	
5	Deep branch of radial nerve	2.5 /1.5*	Extensor muscles of the forearm	1.2 / 1.9
6	Fascicle of median nerve	3.4 / 1.4*	Pronator quadratus	2.9/4.1
			Deep finger flexor	5.4/7.5

Numbers given refer to maximal assessed diameter/proximal diameter of the affected fascicle.

†*Not symptomatic at first assessment, became clinically symptomatic 2 weeks later.*

previously. Clinical neurological examination showed only a winged scapula on the right with an abnormal movement pattern compatible with long thoracic neuropathy. This led to a suspected diagnosis of NA. No abnormal spontaneous activity could be detected in the serratus anterior muscle on electromyographic study.

On HRUS, the CSD of the right LTN was 1.8 mm compared with 1.2 mm on the left. The lower slips of the right serratus anterior muscle had increased echogenicity and a CSD of 4 mm compared with 6 mm on the left. MRI of the brachial plexus was unremarkable.

Patient 2. A 50-year-old woman had a respiratory infection followed by intense pain in the right periscapular region. She was treated with 3 injections with local anesthetics and started physiotherapy. As the pain did not subside after >1.5 months, she saw a neurologist who observed a winged scapula and suspected NA. Results of cerebrospinal fluid (CSF) examination were unremarkable. The patient refused to undergo electromyography (EMG) of the serratus anterior.

HRUS showed a CSD of 1.9 mm of the right LTN compared with 1.3 mm on the left. The CSD of the lower slips of the serratus anterior on the right were 1 mm compared with 3 mm on the left (Fig. 1). The affected muscle also exhibited increased echogenicity. MRI of the brachial plexus was unremarkable.

Patient 3. A 49-year-old man awoke with severe pain in the left periscapular region. The next day, he felt weakness when elevating the left upper extremity. He underwent MRI of the cervical spine, which was unremarkable, and HRUS was performed 2 weeks after onset. The scan showed swelling of the SCN in the supraclavicular fossa, with a CSD of 2.2 mm compared with 1.5 mm on the unaffected side. In addition, there was discrete swelling of the upper trunk on the left with a CSD of 6 mm compared with 3.8 mm on the right. Muscles did not show any alteration on sonography. On MRI of the brachial plexus, there was hyperintense T2 signal of the SCN and contrast enhancement of the SCN and the upper trunk (Fig. 2). The deltoid, infra-, and supraspinatus muscles showed denervation edema. EMG revealed spontaneous activity in the infraspinatus muscle, while the deltoid was unremarkable. CSF examination showed a minimal elevation of total protein (41.6 mg/dl, upper limit of normal: 40) and was normal otherwise.

Patient 4. A 37-year-old man was seen 2 weeks after the onset of symptoms. He described initial pain in the left shoulder and upper arm, and after some days, weakness when bending the tip of the left thumb and index finger. Clinical examination revealed anterior interosseus nerve syndrome on the left, and weakness during external rotation of the left shoulder.

Nerve conduction studies showed localized slowing of motor conduction velocity in the left median nerve in the upper arm. EMG revealed abnormal spontaneous activity in the median portion of the flexor digitorum profundus (the flexor pollicis longus was not examined). Results of the CSF examination were normal.

HRUS showed increased diameter of the upper trunk on the affected side and swelling of a single fascicle of the left median nerve within the medial bicipital groove, approximately 5 cm proximal to the antecubital fossa. Proximal to the swelling, the fascicle had a CSD of 0.8 mm, which increased to 3.1 mm. Maximum CSA of the median nerve was 24.6 mm^2 The connective tissue surrounding the nerve showed a semicircular hypoechogenic area, which was interpreted as an inflammatory exudate. Reactive lymph nodes were clustered along the nerve (Fig. 3). The left musculocutaneous nerve showed a similar

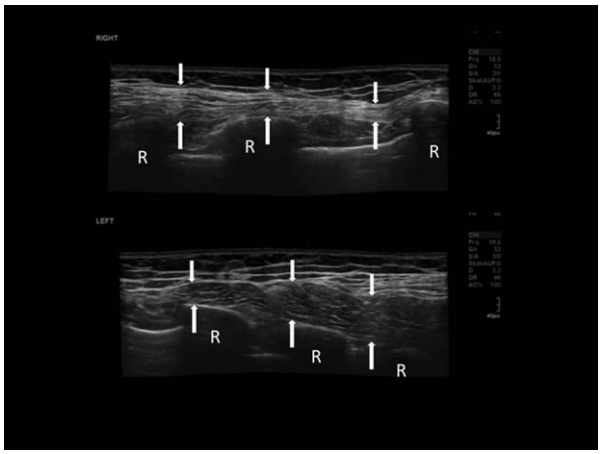

FIGURE 1. HRUS panoramic view of the lower slips of the affected (right) and unaffected (left) serratus anterior muscles (between white arrows) in patient 2. Note the increased echogenicity and reduced CSD of the affected right side (R = rib)

pattern; a single fascicle was noted that increased in CSD from 1.5 to 2.1 mm (CSA of the whole nerve max. 17.6 mm^2). At the time of the first ultrasound assessment, there was also a swelling of the right LTN (CSD of 3.5 mm on the right compared with 1.5 on the left). This was clinically asymptomatic at this time, but it became symptomatic with pain and a winged scapula approximately 2 weeks later (Fig. 4).

MRI of the left brachial plexus showed swelling and contrast enhancement of C5, C6, and the SCN and denervation edema of the supraspinatous, infraspinatous, and deltoid muscles. The right LTN was not visualized, as the initial symptomatic side was the left.

Patient 5. A 48-year-old man underwent surgery for stabilization of the scapula after NA that affected the left LTN and caused persistent painful malrotation. Two months after the surgery, he developed pain in the right shoulder followed by weakness during extension of the right wrist and fingers. He was seen by a neurologist who diagnosed a second episode of NA.

EMG showed abnormal spontaneous activity in the extensor muscles of the forearm. HRUS showed circumscribed swelling of the deep branch of the radial nerve on the right with a maximal CSD of 2.5 mm (CSA 4.3 mm^2) with 1.5 mm proximal to that segment. In addition, there was atrophy of the extensor muscles of the right forearm, with increased echogenicity and a CSD of 1.2 cm compared with 1.9 cm on the left. He did not undergo MRI.

Patient 6. A 49-year-old woman was seen because of unexplained weakness in the flexor muscles of the left lower arm. Six months previously she had a laryngeal infection which led to hospital admission for intravenous antibiotics. Fourteen days after this episode, a strong pain close to the antecubital fossa began, lasted another 14 days, and was followed by weakness when bending the tip of the left thumb and index finger. Her initial suspicion was that the median nerve was injured by the intravenous infusions.

HRUS showed swelling of a fascicle of the left median nerve just proximal to the antecubital fossa. This fascicle had a CSD of 1.4 mm, which increased to 3.1 mm maximum (Fig. 5). CSA of the whole median nerve was 17.5 mm^2 within the swelling. In addition, atrophy of the flexor pollicis longus and pronator quadratus muscles was noticed.

MRI showed increased T2 signal of the median nerve corresponding to the affected site seen on ultrasound.

Routine median nerve conduction studies recording from the abductor pollicis brevis muscle were unremarkable. EMG of the median portion of the flexor digitorum profundus muscles showed no spontaneous activity but a slightly increased rate of polyphasic motor unit action potentials as a sign of a subacute neurogenic lesion.

DISCUSSION

We describe a series of 6 patients with a clinical diagnosis of NA, all of whom showed abnormalities on HRUS of clinically affected nerves. These consisted of focal swelling or fascicular enlargement, and, in patients with longer duration disease, muscle atrophy as well. On MRI, denervation edema was visible at an early stage. However, as the standard plexus MRI examination covers exclusively the upper thoracic and shoulder regions, denervation edema of distal upper extremity muscles was not assessed. Moreover, MRI is limited by resolution, and the LTN, as a very small nerve, could not be visualized to a diagnostic extent using conventional coils within a reasonable examination time.

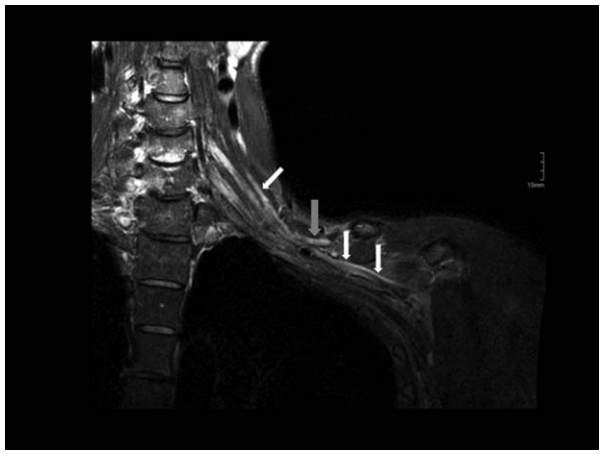

FIGURE 2. Oblique coronal STIR sequence showing hyperintense signal in the C5 root, the upper trunk of the brachial plexus (white arrows) and the suprascapular nerve (gray arrow) in patient 3

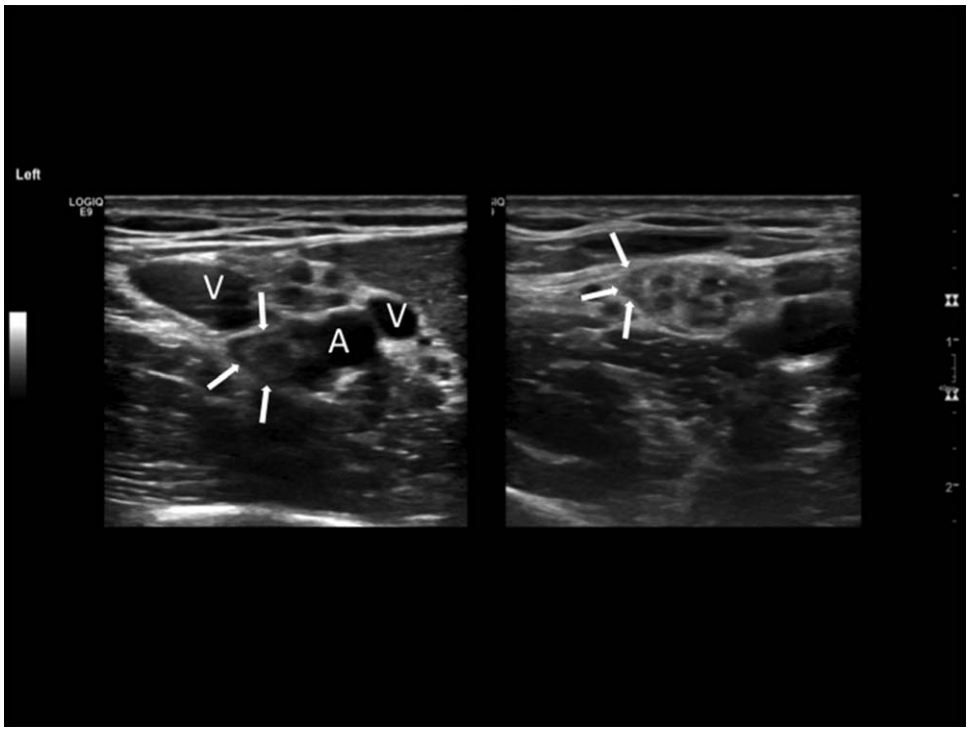

FIGURE 3. Left image: Reactive lymph node next to the median nerve in the bicipital sulcus in patient 4 (A = Brachial artery, V = vein) **Right image:** Median nerve with perineural edema (white arrows).

Concerning HRUS, focal swelling or fascicular enlargement of peripheral nerves is a well-known finding. Swelling is the hallmark of compression syndromes[10,11] before the nerve enters the actual site of compression, and both signs can be found in acute and chronic demyelinating disorders like Guillain-Barré syndrome[12,13] or chronic inflammatory demyelinating polyneuropathy.[14,15] However, in a syndrome in which acute axonal damage is the dominant pathomechanism, this is a new and

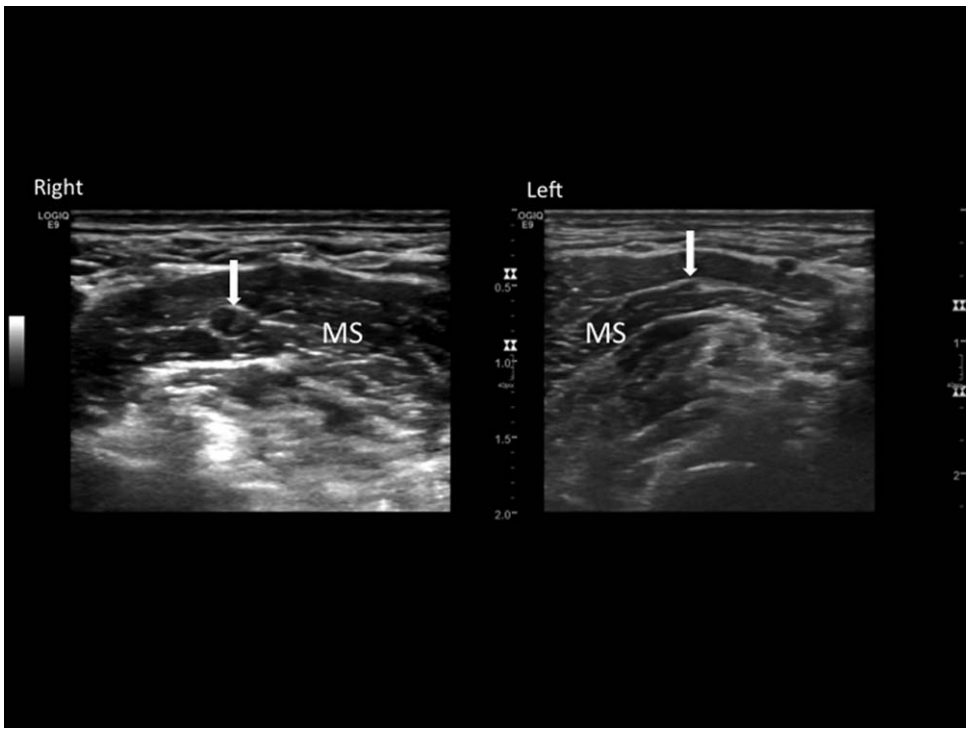

FIGURE 4. Side-to-side comparison of long thoracic nerve (white arrow) in transverse view within the middle scalene muscle (MS) in patient 4 shows enlargement of the nerve on the right.

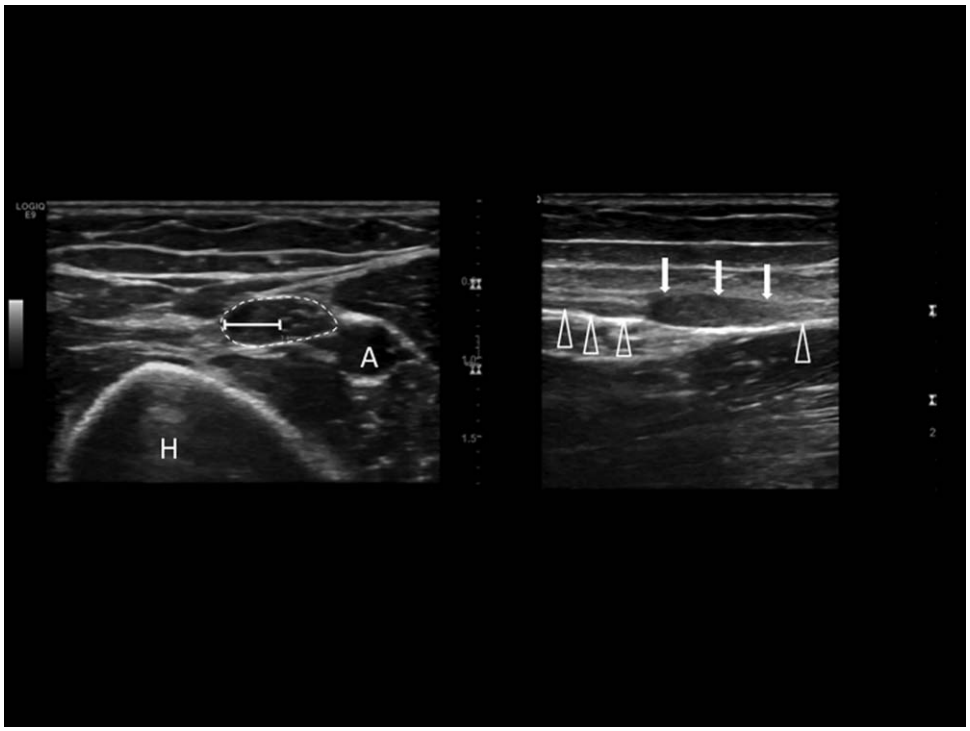

FIGURE 5. Left image: Swelling of a single fascicle (with white distance marker inside) of the median nerve (encircled with a white dotted line) in the transverse view (H = Humerus, A = Brachial artery). **Right image:** The focal nature of this swelling is shown in longitudinal view (white arrows: swollen part of the fascicle; arrowheads: normal caliber of the fascicle proximal and distal to swelling) in patient 6.

exciting finding. Of course it fits well with the concept of autoimmune inflammation, which is considered the underlying etiology of NA.[1]

Concerning fascicular versus diffuse enlargement we think that this is a matter of the resolution of commercially available probes. The affected nerves we encountered that would typically give the "honeycomb" appearance were the median and the musculocutaneous nerve, and these were the nerves in which we could also identify particularly enlarged fascicles (patients 4 and 6). The other affected nerves or roots typically appear as singular hypoechoic dots in the transverse view with HRUS and show generalized enlargement. Thus, there are 2 main possibilities: (1) in smaller nerves there is a greater likelihood that all fascicles are affected in an inflammatory process; or (2) if spatial resolution increases with technical progress, further differentiation of affected and unaffected fascicles will also be possible in these smaller nerves.

For some of the affected nerves, published reference values for CSA are available. These include the median nerve at the midhumerus level,[16] the musculocutaneous nerve,[16,17] and the posterior interosseus nerve.[16] The 2 affected median nerves clearly exceeded the upper limit of normal (12.2 mm^2)[16] with values of 24.6 and 17.5 mm^2 (patients 4 and 6). The same was true for the affected musculocutaneous nerve in patient 4 (17.6 mm^2, with a described upper limit of normal of 6.9[16] or 11.9[17]) and the affected posterior interosseus nerve in patient 6 (4.3 mm^2, with a described upper limit of normal of 2.95[16]). Thus, changes in these nerves were not subtle but were rather distinct.

The earliest time point at which a patient was seen was 2 weeks after onset of symptoms (patient 4). Swelling of affected nerves was easily detectable with both imaging modalities at this early stage. MRI, however, can detect denervation earlier, as it shows denervation edema within 24 h of onset,[18] while muscle ultrasound in our patients only demonstrated muscle atrophy, which develops after some weeks.

The swelling of single fascicles in the 2 patients with the anterior interosseus nerve syndrome (patients 4 and 6) closely resembles the findings of Pham et al., who performed MRI in patients with this condition.[19] We could speculate that our HRUS findings are equivalent and again highlight the predilection of NA for the distal upper arm.

The LTN, most commonly affected in NA, can only be assessed over a short distance and appeared swollen throughout its whole visible course (patients 1, 2, and 4). In 2 of these patients, CSD of the LTN on the affected side was within the range of values in healthy volunteers published by our group previously.[20] However, the minimum side-to-side difference of 0.6 mm (Table 1) in patients clearly exceeded the

normal limit of 0.4 mm found in volunteers. This, however, should be examined systematically in future studies.

A recent study by Aranyi et al.[21] also describes HRUS findings in patients with NA. In addition to noting fascicular swelling, they describe 10 of 14 patients with clinical NA, with involvement of the radial or posterior interosseus nerves. Six of their patients showed complete nerve constriction and an hourglass-like appearance, some of which were also confirmed surgically. We did not observe any of these findings in our sample, and this difference may be explained by first, differences in the clinical picture and second, the time of evaluation after onset, which was up 6 months in our sample and up to 6 years in the patients described by Aranyi et al. Nevertheless, we think that their findings are important in patients with a clinical history of NA that does not follow the classical distribution pattern, and, e.g., predominantly involves the radial nerve. However, we believe our sample, with patients who presented with the classical pattern of nerve involvement, may be more representative of classic NA.

Concerning MRI, a dedicated protocol covering the brachial plexus could not detect abnormalities in 2 of our patients, although they were clearly present on HRUS. In both cases, the LTN was the only affected nerve; the caudal portions of the serratus anterior were the only affected muscles, which are located outside the standard plexus MRI field of view. There were no signal alterations on T2-weighted or STIR sequences, and, there was no enhancement in the 1 patient who also received contrast agent. The LTN is, of course, a smaller nerve compared with the SCN or the cervical roots. Currently, the limited spatial resolution of a standard 3T plexus MRI using conventional coil systems and the limitation of breathing motion during image acquisition, do not allow assessment of this nerve within a reasonable MR examination time. As the LTN is the most frequently affected nerve in NA, this may explain the high rate of false-negative MRI findings and the frequent mismatch with clinical signs.[2] Thus, if considering MRI as the diagnostic imaging modality for NA, one has to be aware of these shortcomings, especially when a winged scapula is the only clinical sign.

Our study has several limitations. It is retrospective in design, uncontrolled, the examiners were not blinded to the patient's suspected diagnosis, and assessment was not standardized but oriented toward clinical symptoms. Despite these limitations, we think that these findings provide an initial impression of the usefulness of HRUS and MRI in patients with typical NA. Additional prospective, controlled studies to assess the diagnostic value of imaging in NA should be performed with the goal of changing the diagnostic approach from one of exclusion of other disorders to confirming NA through HRUS or MRI.

Dr. Lieba-Samal, Dr. Jengogan, and Dr. Kasprian report no disclosures. Dr. Wöber received honoraria from Allergan and Pfizer, is a consultant to Curelator. Dr. Bodner received honoraria from GE.

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