

Focused high-resolution sonography of the suprascapular nerve: A simple surrogate marker for neuralgic amyotrophy?



Leonhard Gruber^{a,*}, Alexander Loizides^a, Wolfgang Löscher^b, Bernhard Glodny^a, Hannes Gruber^a

^a Department of Radiology, Medical University Innsbruck, Anichstraße 35, Innsbruck, Austria

^b Department of Neurology, Medical University Innsbruck, Anichstraße 35, Innsbruck, Austria

ARTICLE INFO

Article history:

Accepted 30 April 2017

Available online 19 May 2017

Keywords:

Neuralgic amyotrophy

Parsonage-Turner syndrome

Suprascapular nerve

High-resolution ultrasonography

Diagnostics

HIGHLIGHTS

- Sonography of the suprascapular nerve can serve as a surrogate marker for neuralgic amyotrophy.
- Patients with neuralgic amyotrophy showed swelling of the suprascapular nerve on the affected side.
- This examination algorithm is simple and can substantiate the suspicion of neuralgic amyotrophy.

ABSTRACT

Objectives: To define the diagnostic value of high-resolution ultrasound (HRUS) of the suprascapular nerve (SSN) in the diagnosis of neuralgic amyotrophy (NA).

Methods: The cross-section areas (CSA) of the SSN at the C5 root (CSA1) and the omohyoid muscle in the midclavicular line (CSA2) were assessed bilaterally in 15 healthy volunteers and 14 patients with clinically and electrophysiologically verified NA.

Receiver-operator-characteristics (ROC) curves were generated and cut-off values, sensitivity, specificity, positive (PPV) and negative predictive values (NPV), likelihood (LR) and odds ratios (OR) were calculated. **Results:** Patients with NA had significantly higher CSA2-values than controls (6.36 ± 2.75 vs. 2.79 ± 0.83 mm², $p < 0.0001$) and significantly higher ratios of SSN CSA2-values of the affected vs. contralateral side ($224.6 \pm 78.5\%$ vs. $127.7 \pm 51.1\%$, $p < 0.0001$). The ratios of SSN CSA2-values vs. CSA1-values ($146.7 \pm 74.5\%$ vs. $99.9 \pm 28.3\%$, $p = 0.008$) and CSA1-values were also significantly higher (4.70 ± 2.00 vs. 2.90 ± 0.90 mm², $p = 0.0028$) than in controls.

Beyond a CSA2 cut-off value of 4.2 mm², the ROC-AUC was 0.939 [0.861–1.00] when compared against healthy volunteers and 0.971 [0.901–1.00] when compared to patients with degenerative shoulder pain. Sensitivity was 85.7% [57.2–98.2%], specificity 96.7% [82.8–99.9%], PPV 92.3% [64.0–99.8%], NPV 93.5% [78.6–99.2%], OR 174.0 [14.4–2106.0] and LR 25.7 (95% confidence intervals in brackets).

Conclusion: SSN swelling in the lateral cervical region could be a supportive finding to identify NA patients.

Significance: This method allows for the rapid sonographic identification of NA.

© 2017 International Federation of Clinical Neurophysiology. Published by Elsevier Ireland Ltd. All rights reserved.

1. Introduction

Neuralgic amyotrophy (NA) is a rare, primarily idiopathic inflammatory condition with an incidence of 2–3/100,000 cases

Abbreviations: HRUS, high-resolution ultrasound; BP, brachial plexus; NA, neuralgic amyotrophy; EDx, electrophysiological testing; SSN, suprascapular nerve; CSA, cross-section area; PPV/NPV, positive/negative predictive value; LR, likelihood ratio; OR, odds ratio; AUC, area under the curve; ROC, receiver-operator characteristics; T2w, T2-weighted; GBS, Guillain-Barré syndrome.

* Corresponding author.

E-mail address: leonhard.gruber@i-med.ac.at (L. Gruber).

per year (van Alfen, 2011), although some authors suggest that the condition is far more common, yet often overlooked, with an incidence of up to 1/1,000 (van Alfen et al., 2015). Male patients are affected twice as often as female ones (Gonzalez Alegre et al., 2002). Patients usually present with atypical, motion-independent shoulder pain and patchy sensory distribution of hypaesthesia (van Alfen and van Engelen, 2006) and usually develop motor symptoms including muscle wasting and paresis over the course of two to eight weeks, while pain usually subsides (Gaskin and Helms, 2006; van Alfen and van Engelen, 2006; van Alfen, 2011). Motor symptoms usually improve (at least partially)

over the course of months to years, depending on disease subtype, but motor impairment often persists to some extent and can lead to severe disability in some cases (van Alfen, 2011). Although exact pathophysiological mechanisms of NA are not fully understood yet, autoimmune inflammatory mechanisms are suspected (van Alfen, 2011). Thus systemic corticosteroids or immunoglobulin administrations are used to reduce symptom severity and improve prognosis and outcome, yet with no clear data in favor of a positive effect (van Alfen et al., 2009a; Stutz, 2010). Potentially, delayed diagnosis and treatment may be causative for the apparent lack of success in many cases, though.

Electrophysiological testing (EDx) can be sensitive, yet unspecific (van Alfen, 2011) if not performed in a thorough fashion and results can depend on the time from onset (van Alfen et al., 2009b; Van Eijk et al., 2016). MRI can be equally unspecific and may reveal inflammation of the brachial plexus (BP) with T2w hyperintense changes of roots, trunks, fascicles and even the surrounding tissue (van Alfen and van Engelen, 2006; Lieba-Samal et al., 2016). Thus, clinical signs and symptoms remain the mainstay of diagnosis (van Alfen, 2011). Unfortunately, due to the lack of specificity of during initial presentation, symptoms are often misinterpreted as common degenerative shoulder pain or at most as unspecific radiculopathy (van Alfen, 2011); the correct final diagnosis of NA can thus be delayed and only established in a subacute stage of the disease with motor involvement.

The mixed sensorimotor suprascapular nerve (SSN), which originates from the superior trunk of the BP and dominantly receives fibers from C5 as well as C6, passes under the inferior belly of the omohyoid muscle in its further course (Blum et al., 2013). The SSN not only innervates the muscles of the shoulder rotator cuff – the supraspinatus and infraspinatus muscles – and also transmits sensory information from the glenohumeral and acromioclavicular joints, but also variably from skin areas of the overlying shoulder region (Boykin et al., 2011; Blum et al., 2013). Although authors have described an SSN involvement in up to 97% of cases with NA in an MRI study (Gaskin and Helms, 2006) with lower incidence rates of 71.1% in another clinical study (van Alfen and van Engelen, 2006), only unspecific oedematous changes have been described in case series so far (Gaskin and Helms, 2006; Scaff et al., 2007; Lieba-Samal et al., 2016); to our knowledge, no data exist on the role of diagnostic imaging in the timely diagnosis of NA.

In this study we assessed the diagnostic value of ultrasound examinations of the suprascapular nerve (SSN) as a surrogate parameter for the presence of NA.

2. Methods

2.1. Characteristics of patients and healthy controls

We retrospectively analyzed data from 14 patients that had been referred to our department from November 2015 to August 2016 for HRUS of the brachial plexus after diagnosis of NA. Diagnosis was based on typical history, clinical and electrophysiological assessment. In selected cases spinal MRI and/or lumbar puncture had been performed to exclude other causes. HRUS examinations were routinely performed to exclude masses or other restrictions of the plexus in a standardized manner, which always included the bilateral examination of the supra- and infraclavicular plexus segments. Two control groups were included: 15 healthy volunteers without any history of neural disease or musculoskeletal pain disorders and 10 patients with known degenerative shoulder pain (defined as typical pain episodes, clinical and imaging findings of omarthrosis) were also examined after consent on their study participation with HRUS in the manner mentioned above to serve as a control group. Information on affected side, patient age, gender, body height, and body weight was also collected.

Data were handled according to the World Medical Association Declaration of Helsinki (59th WMA Assembly, Seoul, 2008) in the prevailing version. An institutional review board approval (Ethikkommission, Medical University Innsbruck, 2009-02-20) was obtained in the form of a general waiver for studies with retrospective data analysis.

2.2. Technical prerequisites and HRUS examination of the SSN

All scans were performed with a Philips Epic Q7 (Philips; Bothell, Washington, USA) using an 18–5 MHz broadband linear array transducer and a predefined musculoskeletal preset as provided by the vendor. All images were stored in the institution's Agfa PACS (Agfa; Mortsel, Belgium).

All examinations were performed by two non-blinded readers with 15 and 8 years of experience in peripheral neural US. The SSN was always examined in a standardized fashion: The origin of the SSN was visualized after locating the dual tubercula of the lateral process of C6, then moving the transducer cranially to visualize the root of C5 as described before (Martinoli et al., 2002) and then following the C5 root laterally until the origin of the SSN was visible (CSA1, Figs. 1 and 2). Then the SSN, which runs ventrally of the middle scalene muscle, was followed laterally to the midclavicular line, where the omohyoid muscle was visualized (CSA2, Figs. 1 and 2); according measurements of CSA1 and CSA2 were always performed strictly perpendicular to the SSN's course by drawing a freehand region of interest (ROI) using the provided measurement module. Additionally, a longitudinal scan of the nerve was performed to assess the distribution pattern of nerve swelling and the presence of constrictions. All measurements were performed on the affected and unaffected side in 14 patients with verified unilateral NA. Bilateral measurements in the same fashion were also performed in 15 healthy volunteers. In the latter, bilateral SSN accessibility was defined over the entire nerve course from the root to the muscular branches for the supra- and infraspinatus muscles at the scapular spine. For a demonstration of the localization and assessment of SSN CSAs, please also refer to the exemplary video provided.

2.3. Statistical analysis

Statistical evaluation was performed in GraphPad Prism 6.0 (GraphPad Software Inc.; CA, USA) and SPSS version 22.0 (SPSS Inc., Chicago, Illinois, USA).

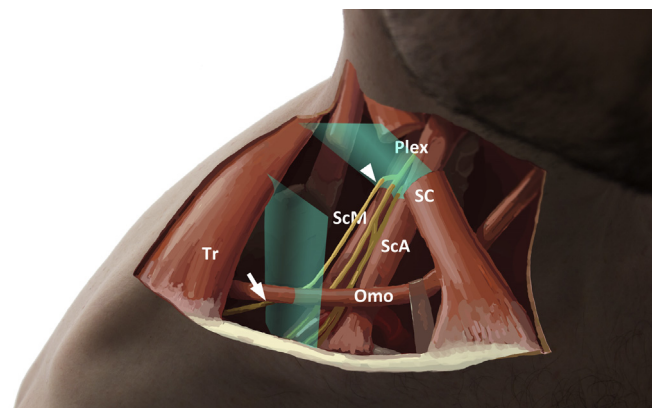


Fig. 1. Illustration of the course of the SSN and the two examination planes (cyan; CSA1: medial, CSA2: lateral). The root of C5 was first localized (see main text) and then followed laterally to identify the SSN origin between the scalene muscles (CSA1, white arrowhead). Then the transducer was moved laterally while always centring on the SSN until the midclavicular line was reached. Here the second CSA measurement was performed (CSA2, white arrow). Tr: trapezius muscle, ScA: anterior scalene muscle, ScM: middle scalene muscle, Plex: brachial plexus, SC: sternocleidomastoid muscle, Omo: omohyoid muscle.

Download English Version:

<https://daneshyari.com/en/article/5627621>

Download Persian Version:

<https://daneshyari.com/article/5627621>

[Daneshyari.com](https://daneshyari.com)