Clinical Neurophysiology 126 (2015) 2390–2396

Contents lists available at ScienceDirect

Clinical Neurophysiology

journal homepage: www.elsevier.com/locate/clinph

Precise localization of ulnar neuropathy at the elbow

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ARTICLE INFO

Article history:
Accepted 26 January 2015
Available online 14 February 2015

Keywords:
Nerve entrapment
Electromyography
Peripheral neuropathy
Ulnar nerve
Ultrasonography

HIGHLIGHTS

- Combination of electrodiagnostic and ultrasonographic studies precisely localized ulnar neuropathy at the elbow (UNE) in 93% of arms.
- Short segment nerve conduction studies (71%) and ultrasonography (72%) were similarly efficient in precise UNE localization.
- Nerve constriction at the site of ulnar nerve entrapment in the cubital tunnel is reported for the first time.

ABSTRACT

Objective: To report the utility of short-segment nerve conduction studies (SSNCSs) and ultrasonography (US) in the precise localization of ulnar neuropathy at the elbow (UNE) and differentiation between lesions in the retroepicondylar (RTC) groove and under the humeroulnar aponeurotic arcade (HUA; i.e., cubital tunnel).
Methods: In a group of prospectively recruited patients with suspected UNE, four blinded examiners took a history and performed neurologic, electrodiagnostic (EDx) and ultrasonographic (US) examinations. Precise UNE localization was determined by SSNCSs criteria (conduction slowing and conduction block), and by US criteria (changes in cross-sectional area – CSA). Localizations obtained by EDx and US studies were compared.
Results: We included 83 patients (86 arms) with SSNCSs or US diagnosis of UNE. US confirmed the SSNCSs localization in 45%, provided localization alone in 24%, and was unable to confirm SSNCSs localization in 23% of arms. Lesions in RTC (76%) were mainly demyelinating (63%), and localized at the medial epicondyle (29%) or 2 cm proximal to it (69%). By contrast, lesions at HUA (17%) were mainly axonal (73%), and localized 2 cm (57%) or 3 cm (43%) distal to the medial epicondyle.
Conclusion: SSNCSs and US are able to precisely localize UNE in the majority (93%) of arms with pathologic SSNCSs or US. UNE in RTC are predominantly demyelinating, and approx. 5-times more common than UNE at HUA that are more commonly axonal.
Significance: SSNCSs and US are of similar utility and complement each other in precise UNE localization.

1. Introduction

The most common locations of ulnar neuropathy at the elbow (UNE) are in the retroepicondylar (RTC) groove and under the humeroulnar aponeurotic arcade (HUA; i.e., in the cubital tunnel) (Campbell et al., 1988, 1992). Differentiation between ulnar nerve lesion at these two locations is potentially useful for a rational treatment approach. Ulnar nerve compression or traction in the RTC is presumed to be (at least initially) best treated conservatively, while entrapment under the HUA requires early surgical release (i.e., cubital tunnel decompression) (Bolster et al., 2013). Furthermore, when conservative treatment of UNE due to UNE in the RTC fails, theoretically anterior transposition of the ulnar nerve would be best tried (Zarezadeh et al., 2012). However, currently no reliable data to support this are available, probably also due to inadequate UNE localization.

Although standard 10-cm segment nerve conduction studies (NCSs) localize ulnar neuropathy to the elbow in 37–86%

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http://dx.doi.org/10.1016/j.clinph.2015.01.023
1388-2457/

(American Association of Electrodiagnostic Medicine and Campbell, 1999; Omejec et al., 2015), they cannot differentiate between a lesion in RTC and under the HUA. Therefore, in order to precisely localize the lesion short-segment NCSs (SSNCSs) should be used (Azrieli et al., 2003; Campbell et al., 1992; Herrmann et al., 2001; Visser et al., 2005). Although magnetic resonance imaging (MRI) (Britz et al., 1996; Vucic et al., 2006) and near-nerve NCSs (Odabasi et al., 1999) have also been used for this purpose, they are less practical, and near-nerve NCSs are also invasive. Ultrasonography (US) has proved to be helpful in the diagnosis of UNE by demonstrating ulnar nerve thickening and occasionally structures compressing the ulnar nerve (Beekman et al., 2004, 2011; Omejec et al., 2015). However, to date, the ability of US to differentiate ulnar nerve lesions at RTC and HUA has not been established. Furthermore, the correlation between SSNCSs and US localization has not been studied yet in individual UNE patients (Park et al., 2004; Simon et al., 2015).

In the present study, we aimed to determine the ability of SSNCSs and US to differentiate between lesions in the RTC and under the HUA in patients with SSNCSs or US diagnosis of UNE.

2. Materials and methods

2.1. Patients and controls

We prospectively recruited patients with suspected UNE in a secondary referral center (i.e., the Institute of Clinical Neurophysiology, University Medical Center Ljubljana, Slovenia) as described previously (Omejec et al., 2015). Inclusion criteria was at least one of the following presenting symptoms typical for UNE: (1) continuous numbness or paresthesias in the 5th and in the ulnar half of the 4th finger; *or* (2) feeling of weakness or clumsiness of the ulnar-innervated muscles; *or* (3) pain on the medial aspect of the elbow radiating to the forearm or hand (Beekman et al., 2004). Exclusion criteria were: (1) previous elbow fracture or surgery; *or* (2) known polyneuropathy, symptoms of polyneuropathy, all conditions causing polyneuropathy (e.g., diabetes), hereditary neuropathy with liability to pressure palsies and multifocal motor neuropathy with conduction block (MMN); *or* (3) motor neuron disorders (e.g., monomelic amyotrophy, amyotrophic lateral sclerosis – ALS). Each of four blinded examiners performed one part of diagnostic evaluation that included the history, neurologic examination, electrodiagnostic (EDx) and US studies. We defined abnormality according to our own reference intervals obtained in a previously reported group of 49 controls (29 men, aged 23–81 years) with demographic characteristics similar to the included patients (Omejec and Podnar, 2015). The study was approved by the National Ethics Committee of Slovenia, and signed written informed consent was obtained from all subjects prior to the investigation.

2.2. History and clinical examination

The first examiner (AD, EDx technician > 20 years) took a short history and collected demographic and clinical data using a focused questionnaire (Mondelli et al., 2006). The second examiner (TŽ, neurologist > 25 years) graded muscle wasting, estimated muscle strength using Medical Research Council (MRC) scale (Florence et al., 1992), and tested light touch and pin prick in both hands.

2.3. Electrodiagnosis (EDx)

The third examiner (SP, clinical neurophysiologist > 20 years) performed ulnar NCSs across the elbow with the subject supine, using a standard EMG system (Nicolet Synergy, Natus Medical Incorporated, San Carlos, USA). With the elbow flexed at 90°, markers were placed at the medial epicondyle (ME), 2 and 4 cm distal (D2, D4), and 2, 4 and 6 cm proximal (P2, P4, and P6) along the course of the ulnar nerve (Kanakamedala et al., 1988). Compound muscle action potentials (CMAPs) were recorded separately from the ADM and the FDI muscles. Ulnar and median antidromic sensory nerve action potentials (SNAPs) from the 4th finger and ulnar SNAPs from the 5th finger were recorded. In all patients, the examiner performed concentric needle electromyography (EMG) of the forearm and hand muscles, as described previously (Omejec et al., 2015).

Using SSNCSs, UNE was diagnosed in nerves with: (1) motor nerve conduction velocity (MNCV) below the lower normative limit for the appropriate short-segment (e.g., <31 m/s in the most critical ME/P2 segment); *or* (2) the CMAP amplitude dropped above the upper normative limit (>12%, Fig. 1) (Omejec and Podnar, 2015). In arms with a disagreement between the ADM and FDI findings, the final EDx diagnosis was reached by also considering sensory NCSs and concentric needle EMG results. UNE was localized to 2-cm segment with maximal MNCV slowing (MNCV < our normative limit) (Omejec and Podnar, 2015). In patients with conduction block, UNE was localized to 2-cm segment with an abnormal CMAP amplitude drop (CMAP amplitude drop > our normative limit) (Omejec and Podnar, 2015). We defined EDx lesion length by summation of 2-cm segments with abnormal MNCV slowing or CMAP amplitude drop.

2.4. Ultrasonography

During US examination, the fourth examiner (GO, US technician with >500 US examinations) measured the ulnar nerve

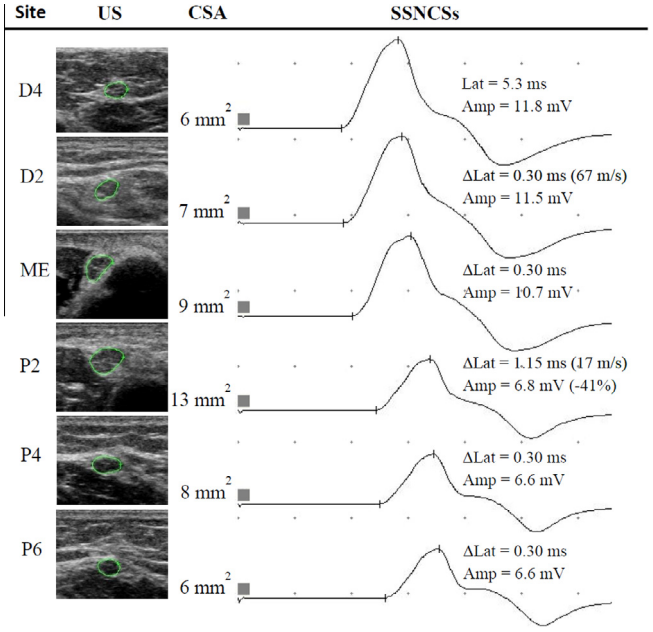

Fig. 1. Ultrasonography (US) and short-segment nerve conduction study (SSNCS) findings in a 35-years old woman included in the present study. Ulnar nerve cross sectional areas (CSAs) were measured and ulnar nerve stimulated on the following sites: D4, D2 (4 and 2 cm distal to the medial epicondyle (ME) respectively), ME; P2, P4, P6 (2, 4 and 6 cm proximal to ME respectively; see left column). Shown compound motor action potentials (CMAPs) were recorded from the abductor digiti minimi muscle. For D4 stimulation CMAP latency (Lat) and for other sites increase in latency compared to 2-cm distal sites (latency difference: ΔLat) are shown. Ulnar nerve US cross sectional views, CSAs and CMAP amplitudes (Amp) are also shown. US (CSA > 10 mm^2) and SSNCSs (ΔLat > 0.65 ms and Amp drop > 12% (Omejec and Podnar, 2015)), localized the ulnar nerve lesion in this arm to the P2 site (US) and the ME/P2 segment (SSNCSs). Findings are characteristic for an ulnar neuropathy located in the retroepicondylar (RTC) groove.