Clinical Neurophysiology 127 (2016) 3259–3264

Contents lists available at ScienceDirect

Clinical Neurophysiology

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

Can neurologic examination predict pathophysiology of ulnar neuropathy at the elbow? ☆

Gregor Omejec, Tomaž Žgur, Simon Podnar *

Institute of Clinical Neurophysiology, Division of Neurology, University Medical Center Ljubljana, Slovenia

ARTICLE INFO

Article history:
Accepted 2 August 2016
Available online 9 August 2016

Keywords:
Axonal neuropathy
Nerve conduction block
Nerve conduction studies
Nerve conduction slowing
Neurologic examination
Pathophysiology of peripheral neuropathy
Ulnar neuropathy at the elbow

HIGHLIGHTS

- This study examined to what extent pathophysiology of ulnar neuropathy at the elbow (UNE) can be predicted by neurological examination.
- Correspondence of UNE pathophysiology with muscle atrophy and weakness was confirmed.
- Neurologic examination, in contrast to nerve conduction studies, did not reliably predict UNE pathophysiology in individual arms.

ABSTRACT

Objective: To explore the utility of neurologic examination to predict the pathophysiology of ulnar nerve lesions in patients with ulnar neuropathies at the elbow (UNE).
Methods: We prospectively recruited consecutive patients with suspected UNE. Four blinded investigators took a history and performed neurologic, electrodiagnostic (EDx) and ultrasonographic (US) examinations. In patients with axonal UNE, conduction block and conduction slowing, the pathophysiologies of UNE and neurologic examination findings were compared.
Results: We found significant differences in muscle bulk and strength of the ulnar hand muscles between 96 arms with axonal UNE, 34 with conduction block, and 45 with isolated conduction slowing. Severe muscle atrophy and weakness (0–3/5 on MRC) predicted axonal UNE, and moderate weakness (−4/5 on MRC) with normal muscle bulk predicted UNE with conduction block. Using more restrictive criteria for axonal and conduction block UNE, muscle strength of 4–5/5 on MRC was predictive of isolated conduction slowing.
Conclusion: Although we found significant differences in patterns of muscle bulk and strength between groups of UNE patients with different UNE pathophysiologies, in the majority of arms, neurologic examination could not reliably predict UNE pathophysiology.
Significance: Results confirm that nerve conduction studies are essential for determination of the pathophysiology of ulnar neuropathy at the elbow.

1. Introduction

Two main pathophysiologic mechanisms underlie peripheral neuropathies: (1) damage to the myelin (i.e., myelinopathy), and (2) damage to the axons (i.e., axonopathy). Discrimination between the two mechanisms substantially narrows the differential diagnosis of polyneuropathies (Tankisi et al., 2007; Bromberg, 2013). The underlying pathophysiology is also a major predictor of the speed of recovery, and often also of the final outcome in neuropathies (Robinson, 2015). The gold standard for the determination of pathophysiology in peripheral neuropathies is electrodiagnostic (EDx) studies. They reveal reduced amplitudes of the motor and sensory responses in axonal lesions, and conduction slowing with or without conduction block in myelin damage (Falck and

☆ Presented in part at the 2nd Congress of the European Academy of Neurology, Copenhagen, May 2016.

* Corresponding author at: Institute of Clinical Neurophysiology, University Medical Center Ljubljana, Zaloška cesta 7, SI-1525 Ljubljana, Slovenia. Fax: +386 1 522 1533.
E-mail addresses: gregor.omejec@kclj.si (G. Omejec), tomaz.zgur@kclj.si (T. Žgur), simon.podnar@kclj.si (S. Podnar).

http://dx.doi.org/10.1016/j.clinph.2016.08.002
1388-2457/

Stålberg, 1995). This is regarded as one of the main advantages of EDx studies compared to neurologic examination.

Nevertheless, at least theoretically, neurologic examination can also provide information on the pathophysiology of peripheral neuropathies. Examination of the motor system is thought to be particularly informative, as it is expected to demonstrate: (1) muscle strength reduction with muscle atrophy in axonal lesions, (2) muscle strength reduction without muscle atrophy in demyelinating nerve lesions with conduction block, and (3) normal muscle strength without muscle atrophy in demyelinating lesions with isolated conduction slowing (Kimura, 2001). However, to our knowledge, there has been no empirical validation of how well these general rules actually work in clinical practice.

Therefore, we compared neurologic examination findings in our group of patients with ulnar neuropathies at the elbow (UNE) with predominant axonal loss, conduction block, or conduction slowing. We aimed to establish the usefulness of individual patterns found on neurologic examination for prediction of UNE pathophysiology. We also compared neurologic findings in UNE under the humeroulnar aponeurotic arcade (HUA), and in the retroepicondylar (RTC) groove (Omejec and Podnar, 2015b).

2. Materials and methods

2.1. Patients and controls

We prospectively recruited a cohort of consecutive patients with suspected UNE that was reported also in our previous studies (Omejec and Podnar 2015a,b; Omejec et al., 2015; Omejec and Podnar, 2016). We included patients with at least one of the following symptoms (i.e., the inclusion criteria): (1) continuous 4th and 5th finger numbness or paresthesia; (2) the ulnar-innervated muscles weakness; or (3) loss of hand dexterity. We excluded all patients with (i.e., the exclusion criteria): (1) previous elbow fracture or surgery; (2) polyneuropathy; or (3) motor neuron disorders.

Four different investigators obtained the history, and performed the clinical neurologic, EDx, and ultrasonographic (US) examinations. They were blinded to the findings of the other parts of the evaluation.

The study was approved by the National Ethics Committee of Slovenia, and prior to the investigation all participating patients provided written informed consent.

2.2. History and clinical neurologic examination

The first investigator obtained a short history, and used a focused questionnaire for collection of demographic and clinical data (Mondelli et al., 2006). The second investigator performed a clinical neurologic examination of both upper limbs. He graded muscle atrophy as 1 – severe, 2 – moderate, 3 – mild, or 4 – normal muscle bulk, and estimated muscle strength according to the Medical Research Council (MRC) scale (O'Brien, 2010). He also graded light touch and pinprick in both arms as 0 – absent, 1 – reduced, or 2 – normal.

2.3. Electrodiagnostic studies (EDx)

The third investigator performed nerve conduction studies (NCSs) using a standard EMG equipment (Nicolet Synergy, Natus Medical Incorporated, San Carlos, USA). He stimulated the ulnar nerve at the wrist, and at six positions separated by 2-cm from 4 cm distal (D4) to 6 cm proximal (P6) to the medial epicondyle (ME) of the elbow (i.e., short-segment NCSs – SSNCSs). Compound muscle action potentials (CMAPs) were recorded separately from the abductor digiti minimi (ADM) and the first dorsal interosseus (FDI) muscles, as described in detail elsewhere (Omejec and Podnar, 2015b). He also recorded the ulnar sensory nerve action potentials (SNAPs) on stimulation at the wrist and recording from the 5th finger (i.e., antidromic), and performed concentric needle electromyography (EMG) of the selected hand and forearm muscles (Omejec et al., 2015).

We diagnosed and localized UNE to a 2-cm segment with the most pronounced: (1) motor nerve conduction velocity (MNCV) slowing (i.e., below the lower normative limit, <31 m/s); or (2) CMAP amplitude drop in the elbow area (i.e., above the upper normative limit, >12%) (Omejec and Podnar, 2015a,b). We also used NCSs to determine the pathophysiology of UNE: (1) the CMAP amplitude on stimulation 4-cm distal to ME (D4) below the lower normative limit (i.e., 6.5/6.6 mV on ADM/FDI muscle recording) = axonal UNE; (2) the CMAP amplitude drop in the elbow segment above the upper normative limit (>12%) = UNE with conduction block; and (3) the MNCV in the elbow area below the lower normative limit (<31 m/s) = conduction slowing (Omejec and Podnar, 2015a,b). In arms with incongruent CMAP recordings from the ADM and FDI, UNE pathophysiology was determined also using the 5th finger SNAPs (the lower normative limit <13 μV (Omejec and Podnar, 2015a)) and needle EMG findings. We diagnosed axonal pathophysiology also when additional conduction block or conduction slowing, and conduction block pathophysiology when additional conduction slowing was present (Omejec and Podnar, 2015b).

2.4. Ultrasonography (US)

The fourth investigator measured cross-sectional areas (CSAs) of the ulnar nerve at the wrist and at six markers across the elbow. He used a standard US equipment (ProSound Alpha 7, Hitachi Aloka Medical, Ltd, Tokyo, Japan), and a 4–13 MHz linear array transducer. For CSA measurements he employed a trace method excluding the hyperechoic rim (Omejec and Podnar, 2015b).

We diagnosed and localized UNE: (1) to the ulnar nerve constriction (i.e., CSA just proximal and distal $\geqslant 2$ mm^2 larger); (2) in ulnar nerves with maximal CSA (CSAmax) distal to ME: to the first marker distal to the ulnar nerve with CSA > our normative limit; or (3) in ulnar nerves with CSAmax at or proximal to ME: to the CSAmax (Omejec and Podnar, 2015b).

We diagnosed UNE under the HUA, if the lesion was localized distal to ME, and UNE in the RTC groove, if the lesion was localized at ME or proximal to ME. We classified as non-localized all UNE with unclear exact localization (Omejec and Podnar, 2015b).

2.5. Statistics

After exclusion of patients with: (1) alternative diagnoses (e.g., ulnar neuropathy at the wrist, C8 radiculopathy, etc.); (2) normal neurologic examination; and (3) normal SSNCSs and US studies, we included in further analyses patients with UNE diagnosis, confirmed by SSNCSs or US. From comparison of UNE under the HUA and in the RTC we also excluded all non-localized UNE.

We described continuous variables as the median, 5th and 95th percentiles, range, or as the mean and standard deviations (SDs). We compared two groups by the Mann–Whitney *U*-test, and three groups by the Kruskal–Wallis test. The effect size of the difference between each group pair was calculated as $Z/\sqrt{N}$; values of 0.1–0.3 were considered small, 0.3–0.5 medium, and >0.5 large (Fritz et al., 2012). Correlations between variables were shown by the Spearman correlation coefficient. For the purpose of statistical analyses, the MRC scale was transformed to seven grades, with values of 1–7. All tests were performed at a significance level of $\alpha = 0.05$ (two-sided).