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Ulnar neuropathy with abnormal non-localizing electrophysiology: Clinical, electrophysiological and ultrasound findings

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HIGHLIGHTS

- A quarter of ulnar neuropathies with abnormal electrophysiology were axonal non-localizing (NL-UN).
- Most NL-UNs (87%) were in males with severe or moderate clinical and electrophysiological ratings.
- Ultrasound had a critical role in localization and classification that facilitated management.

ABSTRACT

Objective: To systematically study demographic, clinical, electrophysiological and nerve ultrasound characteristics of ulnar neuropathy with abnormal non-localizing electrophysiology (NL-UN) and further define the utility of ultrasound over and above the conventional electro-diagnostic approach.
Method: NL-UNs were prospectively identified from 113 consecutive referrals with suspected ulnar neuropathy. All received electro-diagnostic tests and ulnar nerve ultrasound. NL-UN severity was graded using clinical and electrophysiological scales.
Results: In 64 of 113 referrals, an ulnar mono- neuropathy was confirmed by electrophysiology. Sixteen of these 64 (25%) had NL-UN, predominantly males (14 out of 16 patients) with severe or moderate clinical and electrophysiological ratings. Ultrasound showed focal ulnar neuropathy at the elbow in 13 out of 16, and diffuse ulnar nerve abnormality in three, and identified a likely or possible causative mechanism in 11.
Conclusion: A significant proportion (a quarter) of ulnar neuropathies with abnormal electrophysiology were NL-UN, of heterogeneous etiology; the majority were males with significant disability and axonal loss. Ultrasound had a significant role in localization and classification that facilitated management.
Significance: To our knowledge, this is the first systematic prospective study that analyzes the demographic, clinical, electrophysiological and ultrasound characteristics of NL-UN in a routine clinical neurophysiology setting.

1. Introduction

Ulnar neuropathy, the second most common mono-neuropathy, is usually due to focal nerve pathology at the elbow, with an estimated incidence of 24.7/10^5/year (Mondelli et al., 2005). It typically presents with paraesthesia or sensory loss in the little and ring fingers and weakness of ulnar innervated muscles. Diagnosis is usually based on clinical findings and abnormal electrophysiology. Electrodiagnostic tests can localize the lesion by demonstrating focal conduction slowing, with or without temporal dispersion

Abbreviations: APB, abductor pollicis brevis; ADM, abductor digiti minimi; CMAP, compound muscle action potential; CSA, cross sectional area; CSA Max, maximal cross sectional area; EDX, electrophysiology; EMG, electromyography; FDI, first dorsal interosseous; MRC, medical research council; NL-UN, non-localizing ulnar neuropathy; SNAP, sensory nerve action potential.

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and conduction block across the elbow (AAEM, 1999a), with a sensitivity varying from 38 to 89% (AAEM,1999b). In some ulnar neuropathies however, electrophysiology is abnormal, showing findings of axonal degeneration, but non-localizing (Jabre and Wilbourn, 1979; Schady et al., 1998; Wilbourn, 1987). From now on we will refer to these subgroup of ulnar neuropathies as 'Non Localising Ulnar Neuropathy (NL-UN). NL-UN is often mentioned in the literature, but a systematic analysis of its incidence, demographic, severity and distinctive pathophysiology is not available. NL-UN may be associated with important impairment and disability, but management can be difficult in the absence of anatomically defining studies. It is unclear if NL-UN is indeed non-localized, or if it may be an undetected focal ulnar neuropathy that might benefit from decompression. This could be particularly difficult in patients with diabetes or other systemic disorder, in whom the ulnar mononeuropathy could have a metabolic or ischemic basis, rather than being secondary to focal nerve injury.

In recent years, high-resolution nerve ultrasound has emerged as a reliable and sensitive technique to complement electrophysiological investigation of mono-neuropathies, and previous papers have also included data on utility of nerve ultrasound in electrodiagnostically non-localizable ulnar neuropathy at the elbow (Beekman et al., 2004a, 2004b, 2011; Omejec and Podnar, 2015).

The purpose of this study was to investigate frequency, demographic, clinical and electrophysiological characteristics of NL-UN and use ultrasound in order to assist with classification and to examine the utility of ultrasound over and above the conventional electro-diagnostic approach.

2. Methods

2.1. Patients

In this prospective study, NL-UN was identified from all consecutive referrals with suspected ulnar neuropathy to our neurophysiology clinic, from May 2014 to January 2016.

The procedures followed were in accordance with the Helsinki Declaration of 1975 and were all part of the routine procedures for investigation of ulnar neuropathy at our Institution.

Clinical inclusion criteria were: numbness and/or paresthesia in the little and ring fingers with or without weakness and atrophy of ulnar-innervated hand muscles and medial elbow pain. Patients with previous ulnar nerve decompression, medial cord/lower trunk plexopathy or C8/T1 radiculopathy confirmed by electrophysiological tests (please see Section 2.2 for electrophysiology protocol) were excluded. Patients with possible underlying peripheral neuropathy in association with systemic conditions were included if the ulnar nerve was disproportionately affected, making it unclear whether this was ulnar neuropathy at the elbow or a complication of the systemic condition.

2.2. Electrodiagnostic tests

American Association of Electrodiagnostic Medicine recommendations were followed for electrophysiological evaluation and diagnosis of ulnar neuropathy at the elbow and exclusion of mimicking conditions such as C8/T1 radiculopathy and medial cord/lower trunk plexopathy (AAEM, 1999a). A single investigator (LP, clinical neurophysiologist >25 years) performed the electrophysiological studies using the Nicolet Synergy EDX EMG System, Natus Medical Incorporated, San Carlos, USA. Sensory and motor nerve conduction studies were performed with surface stimulation and recording under controlled limb temperature (>31 °C). The ulnar antidromic sensory nerve action potential (SNAP) was recorded from the little finger with stimulation at the wrist. The ulnar compound muscle action potential (CMAP) was recorded from the abductor digiti minimi (ADM) and first dorsal interosseous (FDI) with stimulation at the wrist, below elbow (BE) (3 cm distal to the medial epicondyle), above elbow (AE) (10 cm proximal to BE) and axilla. Abnormality was defined (from prior values obtained in our laboratory) by sensory baseline-to-peak amplitude <10 uV, sensory peak latency >3.1 ms (distance 11 cm), CMAP amplitude <5.0 mV for ADM and <6.5 mV for FDI, and motor conduction velocity (MCV) <50 m/s. In addition, for the electrophysiology grading of severity, amplitudes were considered 'markedly reduced' when <20% of the above lower normal limit for SNAP (i.e. SNAP < 2 uV) and < 30% of lower normal limit for CMAP (i.e. <1.5 mV for ADM and <1.9 for FDI). Motor studies were performed with the elbow flexed 90–70 (i.e. 20° less flexed than 90°).

According to the AAEM criteria, the following suggested a focal ulnar nerve lesion at the elbow (UNE): absolute MCV from AE to BE <50 m/s; an AE-to-BE segment >10 m/s slower than BE-to-wrist MCV; a decrease in CMAP negative peak amplitude from BE to AE >20%, suggestive of conduction block or temporal dispersion (assuming that anomalies of innervation i.e. Martin-Gruber anastomosis were not present); a significant change in CMAP configuration at the AE site compared to the BE site (assuming that anomalies of innervation i.e. Martin-Gruber anastomosis were not present).

In cases of severe neuropathy associated with conduction slowing over the BE-to-Wrist segment – presumably secondary to Wallerian degeneration – a comparison of the MCVs over the AE-to-BE and axilla-to-AE segments were included.

When motor conduction studies with the above protocol provided inconclusive evidence for a focal lesion at the elbow, an inching study was also obtained, looking for abnormal changes in the CMAP amplitude, area or configuration or abnormal changes in latency over 2 cm increments. We used a 2 cm × 5 protocol with two stimulations below the epicondyle, two above, and one at the epicondyle. However, accurate measurements of the onset (or peak) latency of the CMAP over short segments were not always possible when the CMAP amplitude was markedly reduced and the CMAP morphology was abnormal. In these situations, the inching study was deemed unreliable and the electrophysiological study classified as 'non-localizing'.

In all NL-UNs, electromyography (EMG) of FDI and/or ADM, flexor carpi ulnaris (FCU) and, where appropriate flexor digitorum profundus (FDP 4–5), was done to assess severity of denervation. Additional non-ulnar muscles (flexor pollicis longus and extensor indicis proprius and, if required, cervical paraspinal) were also examined to exclude C8/T1 radiculopathy and medial cord/lower trunk plexopathy.

2.3. Diagnostic criteria, clinical severity and electrophysiological severity of NL-UN

NL-UN was diagnosed when electrophysiology showed axonal pathology with absent/reduced SNAP and/or CMAPs, without evidence (per the criteria outlined in Section 2.2) of focal abnormality at the elbow.

Clinical severity of NL-UN was graded (scale adapted from previous classifications (Bartels et al., 1998; McGowan, 1950) as *Mild*: sensation reduced (light touch and pin-prick), motor function either normal or mild weakness of abductor digiti minimi (ADM) and/or first dorsal interosseous (FDI), Medical Research Council (MRC) >4; *Moderate*: ADM/FDI atrophy and weakness, MRC 4, and *Severe*: ADM/FDI atrophy and weakness, MRC $\leq$ 3.

Electrophysiological severity was graded as *Mild*: reduced SNAP-with normal CMAPs, *Moderate*: reduced SNAP *and* CMAPs or absent SNAP and normal or mildly reduced CMAP with abnormal EMG,