

## CLINICAL AND NEUROLOGICAL MANIFESTATIONS OF ULNAR NERVE INJURY

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### Abstract

**Objective:** To analyze the clinical and neurological manifestations of ulnar nerve injury (UNI) of various etiologies, evaluate the relationship between the level of injury, degree of neurological deficit, and functional impairment, and assess the diagnostic value of neurophysiological and imaging methods. Ninety-four patients aged 17–69 years were examined. Traumatic lesions were found in 44 patients (47%), compressive–ischemic injuries in 33 (35%), and iatrogenic injuries in 17 cases (18%). Clinical and neurophysiological assessments included strength testing of intrinsic hand muscles, sensory testing in the ulnar distribution, evaluation of neuropathic pain, electromyography (EMG), high-resolution ultrasound, MRI, and standardized hand function tests. The main manifestations included interosseous muscle weakness, hypothenar atrophy, Froment’s sign, claw-hand deformity, paresthesia in digits IV–V, and neuropathic pain. Injury severity correlated with lesion level, extent of axonal degeneration, and duration of symptoms. The results emphasize the importance of early diagnosis, comprehensive evaluation, and timely rehabilitation.

**Keywords:** ulnar nerve; neuropathy; cubital tunnel syndrome; nerve trauma; EMG; axonal degeneration; hand motor deficit.

### Introduction

Ulnar nerve injury (UNI) is one of the most common focal mononeuropathies of the upper limb, accounting for up to 28% of all peripheral nerve compressive syndromes (Kozin, 2020). The ulnar nerve is vulnerable at several anatomical sites, particularly at the elbow (cubital tunnel) and the wrist (Guyon’s canal), due to its superficial course and susceptibility to external pressure.

Clinical manifestations typically include weakness of intrinsic hand muscles, impaired finger abduction and adduction, decreased pinch strength, sensory deficits in the ulnar digits (IV–V), and neuropathic pain. In long-standing cases, characteristic deformities such as clawing of the fourth and fifth fingers may occur (Aszmann, 2019).

Understanding the correlation between lesion site, degree of motor–sensory deficit, and electrophysiological abnormalities is crucial for accurate diagnosis, prognosis, and treatment planning.

## Materials and Methods

### Study population

A total of **94 patients** were enrolled:

- **Men:** 58 (62%)
- **Women:** 36 (38%)
- **Age:** 17–69 years (mean  $41.7 \pm 13.2$  years)
- **Symptom duration:** 3 weeks to 18 months

### Etiology:

- traumatic injury — 44 (47%),
- compressive neuropathy — 33 (35%),
- iatrogenic — 17 (18%).

### Clinical examination

Evaluated parameters included:

- muscle strength (MRC scale),
- interosseous muscle function,
- Froment's sign, Wartenberg's sign,
- claw-hand deformity,
- sensory testing (light touch, pinprick, vibration),
- neuropathic pain (DN4 questionnaire),
- hand function tests (Nine-Hole Peg Test, Purdue Pegboard Test).

### Electrophysiological studies (EMG)

Assessed:

- motor conduction velocity (MCV),
- sensory conduction velocity (SCV),
- compound muscle action potential (CMAP),
- axonal vs. demyelinating patterns,
- conduction block,
- identification of lesion level.

### High-resolution ultrasound & MRI

Ultrasound parameters:

- cross-sectional area (CSA),
- nerve echogenicity,
- signs of compression or fibrosis.

MRI was used to detect:

- edema,
- perineural scarring,
- space-occupying lesions.

### Statistical analysis

SPSS 26.0; ANOVA, Mann–Whitney U-test,  $\chi^2$  test, Pearson correlations.

Significance  $p < 0.05$ .

**Results****Clinical manifestations****Table 1.****Frequency of symptoms**

<b>Symptom</b>	<b>n</b>	<b>%</b>
Interosseous muscle weakness	71	76%
Hypothenar atrophy	63	67%
Froment's sign	59	63%
Wartenberg's sign	46	49%
Claw-hand deformity	42	45%
Hypoesthesia in digits IV-V	58	62%
Paresthesia	49	52%
Neuropathic pain	37	39%

**Association between lesion level and deficit****Table 2.****Neurological manifestations by level of injury**

<b>Level of injury</b>	<b>Dominant manifestations</b>	<b>Mean MRC (interosseous muscles)</b>
Above-elbow segment	Severe motor deficit, clawing	2.1 ± 0.7
Cubital tunnel	Sensory symptoms, moderate weakness	3.0 ± 0.8
Guyon's canal	Localized hand weakness, minimal sensory loss	3.4 ± 0.6

**Electrophysiological data**

- Axonal degeneration — **46%**
- Demyelination — **34%**
- Mixed pattern — **20%**

Conduction block was present in **28%** of compressive lesions.

**Table 3.****EMG findings and functional correlation**

<b>EMG pattern</b>	<b>Sensory loss</b>	<b>Hand dysfunction severity</b>	<b>Pain intensity</b>
Axonal	+++	High	Moderate
Demyelinating	+	Moderate	Low
Mixed	++	High	High

### Correlation analysis

- Motor deficit ↔ axonal degeneration:  $r = -0.62$ ;  $p < 0.001$
- Neuropathic pain ↔ compressive injury:  $r = 0.44$ ;  $p = 0.03$
- Hand function impairment ↔ level of lesion:  $r = -0.51$ ;  $p < 0.01$

### Discussion

This study confirms that UNI manifests primarily as intrinsic hand muscle weakness, loss of dexterity, sensory impairment, and characteristic deformities. These findings are consistent with recent publications (Novak, 2021; Lundborg, 2020; Spinner, 2021).

Traumatic injuries produced the most severe motor deficits, as previously described by Kline (2018) and Midha (2020). Compression neuropathies were characterized by neuropathic pain and sensory deficits, consistent with Dellon (2020) and Kim (2018).

High-resolution ultrasound demonstrated high sensitivity for diagnosing cubital tunnel syndrome, supporting data from Martinoli (2018) and Visser (2020).

Electrophysiology remains the gold standard for lesion localization and prognostic evaluation. Axonal degeneration is strongly associated with poor recovery, matching conclusions from Zochodne (2021) and Brown (2020).

### Conclusion

1. Ulnar nerve injury leads to significant motor and sensory deficits, particularly affecting intrinsic hand muscles.
2. The level of injury determines the severity and pattern of clinical manifestations.
3. EMG and ultrasound provide essential diagnostic information for accurate localization and prognosis.
4. Compressive neuropathies frequently produce neuropathic pain.
5. Early diagnosis and structured rehabilitation significantly improve functional outcomes.

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