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Cyclist's Palsy: A Case Report of Acute Ulnar Neuropathy at the Wrist

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Abstract

Ulnar neuropathy at the wrist is uncommon and can be caused by various extrinsic and intrinsic factors. Prolonged pressure on a bicycle handlebar can cause symptoms of distal ulnar palsy, known as cyclist's palsy. It can present with purely sensory, purely motor, or combined sensorimotor symptoms and is a challenging clinical diagnosis. Our patient presented with acute right-hand grip weakness and tingling in both hands after a bike ride. A prompt and detailed nerve conduction study (NCS) and needle electromyography (EMG) diagnosed right ulnar neuropathy in Guyon's canal, affecting only its deep motor branch, distal to the hypothenar branch. Stimulation in the palm showed a conduction block in these fibres, thus establishing a neuropraxic neuropathy. In addition, there was evidence of bilateral Carpal Tunnel Syndrome. The patient recovered completely with conservative management. This case illustrates the use of NCS/EMG to diagnose an uncommon neuropathy, resulting in optimal patient outcomes.

Keywords: Cyclist; Hand weakness; Ulnar neuropathy; Nerve conduction study; Conduction block.

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Introduction

Ulnar neuropathy at the wrist (UNW) is a challenging diagnosis because it is uncommon and can present with varied clinical features depending on the exact site and the fibres involved. Causes include trauma; external compression (e.g., ganglions, lipomas, anomalous anatomy); chronic repetitive injury (use of jackhammer, long-distance cycling), etc.[1] Handlebar (cyclist's) palsy is characterized by ulnar neuropathy at the Guyon's canal, usually developing after long-distance riding.[2] Its diagnosis requires a high index of clinical suspicion, along with focused imaging and electrodiagnostic testing. We describe here a case of acute cyclist's palsy in which nerve conduction studies (NCS) and needle electromyography (EMG) clinched the diagnosis.

Case Report

A 37-year-old woman developed sudden weakness in her right hand, with pain in the right wrist and paresthesia in the right hand more than the left hand 5–6 days prior,

Figure 1. Froment's Sign.

Figure 2. Benediction sign in right hand.

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Table 1. Sensory Nerve Action Potential (SNAP).

Nerves/ Sites	Lat ms	Amp (2-3) microvolt	Distance cm	SNCV m/s	Comments
R Median - Digit II (Orthodromic)					
Index Finger- Wrist	3.28	8.6	13.5	**41.1**	**Normal amplitude but slowed across the wrist**
L Median - Digit II (Orthodromic)					
Index Finger- Wrist	3.28	12.0	12.5	**38.1**	
R Ulnar - Digit V (Orthodromic)					
Little Finger - Wrist	1.56	16.1	9	57.6	
L Ulnar - Digit V (Orthodromic)					
Little Finger - Wrist	1.72	11.3	9	52.4	**Normal**
R Dorsal ulnar cutaneous					
Forearm - dorsum of hand	1.41	45.6	9	64.0	
R Radial - Superficial Branch					
Forearm - base of thumb	1.82	54.7	12	65.8	

Abbreviations: Lat - Latency, Amp - Amplitude, SNCV: Sensory nerve Conduction Velocity
The abnormal findings are highlighted in Bold

while attempting to ride a bike. She attributed the wrist pain to holding the bicycle handle too tightly for fear of falling.

On examination of the right upper limb: thumb adduction; fingers' adduction and abduction (except the little finger); and flexion of the fourth and fifth metacarpophalangeal joints were graded 3/5 on the MRC scale. Froment's sign (Picture 1), Benediction sign (Picture 2), and the card test were positive. Right thumb abduction, finger extension, and wrist movements were normal. All muscles of the left upper limb showed grade 5/5 power. Arterial pulsations were intact with no palpable swellings. The Tinel test at the wrist for the median nerve was positive bilaterally. Other systems were normal. Her past medical history was unremarkable. A working diagnosis of right ulnar neuropathy at the elbow or wrist with possible

Table 2. Compound Muscle Action Potential (CMAP).

Nerves/ Sites	Lat m/s	Amp1-2 mv	Amp 2-4 mv	Area %	Duration %	Distance cm	MNCV m/s	Comments
R Median - APB								
Wrist	**4.16**	13.2	8.4	100	100	7		**TL: Prolonged CMAP: normal amplitude**
Elbow	7.92	12.7	8.0	97	106	22	57.1	
L Median -APB								
Wrist	**4.32**	17.4	10.1	100	100	7		
R Ulnar - ADM								
Wrist	1.82	12.3	7.9	100	100	7		
Below ME	5.68	10.9	6.7	92.5	101	23	59.7	**CMAP: normal No segmental slowing across elbow**
At Me	6.15	10.9	6.8	95	102	26	60.1	
Above ME	7.45	10.6	6.6	99.6	100	11	62.1	
Erbs point	12.03	10.8	6.7	98.8	101			
L Ulnar - ADM								
Wrist	2.29	10.5	6.5	100	100	7		
R Ulnar - FDI								**TL at wrist: Prolonged PCB noted on wrist stimulation**
Palm	2.29	17.3	9.8	100	100			
Wrist	**4.43**	2.4	**1.5**	**20**	105			
L Ulnar – FDI								
Palm	2.19	16.7	9.7	100	100			
Wrist	3.44	15.3	8.9	92.4	107			

Abbreviations: Lat - Latency, Amp - Amplitude, MNCV: Motor nerve Conduction Velocity, TL: terminal Latency, ME: medial epicondyle, PCB: Partial Conduction Block
The abnormal findings are highlighted in Bold

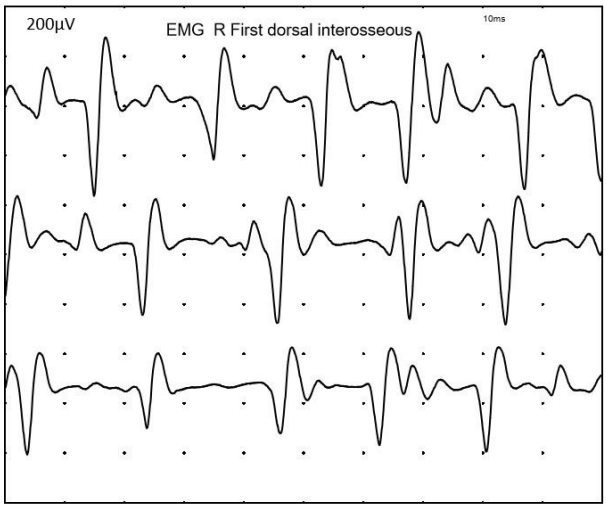

Figure 4. Needle EMG of right First dorsal interosseous muscle

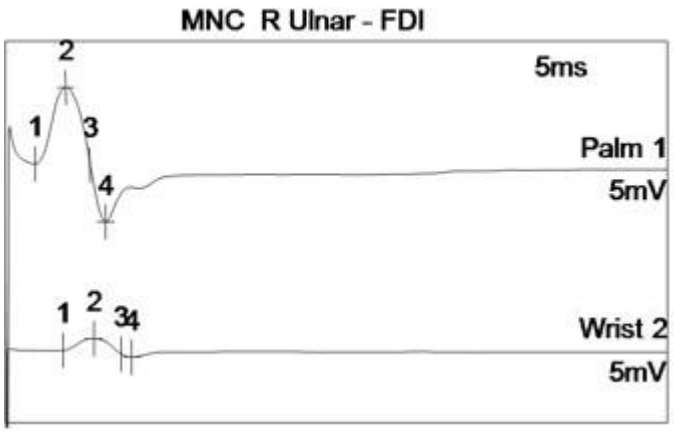

Figure 3. Compound Muscle Action Potential recording from Ulnar nerve - First dorsal interosseous muscle on palm and wrist stimulation.

Table 3. F waves.

Nerve	F min lat ms	COMMENTS
R Median - APB	27.66	
L Median - APB	26.30	
R Ulnar - ADM	25.31	Normal
L Ulnar - ADM	25.57	
L Ulnar - FDI	26.93	
R Ulnar - FDI	**29.11**	Prolonged

Abbreviations: Fmin Lat: minimum latency of F waves.
The abnormal findings are highlighted in Bold

bilateral median nerve involvement vs. C8 radiculopathy was made.

To localize the site of involvement, a detailed electrodiagnostic evaluation of both upper limbs was conducted. Routine NCS of the ulnar nerves — sensory nerve action potential (SNAP) to digit V and compound muscle action potential (CMAP) of abductor digiti minima (ADM) — were bilaterally normal. The dorsal ulnar cutaneous SNAPs were also bilaterally normal. Assessment of the sensory and motor fibers of both median nerves (SNAP of digit II and CMAP of abductor pollicis brevis) showed prolonged distal latencies at the wrist with normal amplitudes (Tables 1, 2). This focal slowing across the wrist region indicated bilateral median entrapment neuropathies (Carpal Tunnel Syndrome).

However, this finding of "slowing" did not explain her sudden onset of weakness in the right hand. Hence, further assessment for right ulnar neuropathy at the elbow — a common site for pressure palsy — was done. Segmental

studies of the ulnar nerve across the elbow were normal with no conduction blocks noted up to supraclavicular stimulation. The CMAP from the right first dorsal interosseous (FDI) was recorded; it showed severely attenuated amplitude (1.5 mV) and a prolonged distal latency at the wrist (4.43 ms) compared to the left hand (amplitude 8.8 mV, distal latency 3.59 ms). The F-waves from right FDI also showed prolonged minimal latency (29.11 ms) compared to the contralateral (left) FDI (26.93 ms). They also compared with F-waves recorded from the ipsilateral (right) ADM muscle (25.31 ms) (Table 3). Since the duration of weakness was only 5–6 days, to assess for conduction block, the FDI branch was stimulated in the palm, which showed a normal amplitude CMAP (9.8 mV). This drop in CMAP area of ~ 80% with no significant temporal dispersion suggested a severe partial conduction block in the motor fibres to the right FDI muscle distal to the wrist (Table 2, Figure 3). On EMG, no spontaneous activity was noted in any muscle. The dorsal interossei muscles of the right hand (first to fourth) showed normal morphology, fast-firing motor unit potentials, and a severely reduced interference pattern (Figure 4), correlating with the conduction block noted on NCS. All other right ulnar-innervated muscles (abductor digiti minimi, flexor carpi ulnaris, flexor digitorum profundus), the median-innervated muscle (abductor pollicis brevis), and radial-innervated muscles (extensor indicis proprius) showed a normal EMG pattern.

The final diagnosis was right ulnar neuropathy affecting only its deep motor branch. The site of involvement was in Guyon's canal (Zone IV), distal to the hypothenar branch, and the pathophysiology was consistent with focal demyelination (neuropraxia). In addition, there was evidence of bilateral Carpal Tunnel Syndrome affecting both the sensory and motor fibers of the median nerves. MRI of the right wrist was done to rule out structural pathology; it was normal.

A short course of steroids and multivitamins with physiotherapy was started. A figure 8 or ring splint for the fingers was provided for daytime use. A resting wrist-hand orthosis was advised during sleep. One month later, she had recovered full clinical power.

Discussion

Ulnar neuropathy at the wrist from cycling ("cyclist's palsy") typically arises due to prolonged pressure on the Guyon's canal combined with wrist posture that compresses or stretches the ulnar nerve. In previously published reports, presentation has often involved both motor and sensory branches, or mixed sensorimotor findings. For instance, Frontera WR[7] reported cyclist's palsy with both sensory and motor involvement; Noth et al.[8] similarly documented mixed lesions. However, in our case, the sensory fibres of the ulnar nerve were preserved, and only the deep motor branch was affected. This selective involvement is less commonly reported.

Our report adds several distinguishing features:

Localization by segmental stimulation in the palm-to-wrist route of the deep motor branch to FDI enabled detection of a conduction block distal to the hypothenar branch, which is rarely documented in detail in prior literature.

The relatively acute onset (5–6 days prior) contrasts with many reports involving chronic or repetitive cycling over long distances. In this patient, the duration was short, yet the degree of motor deficit was considerable, reinforcing the notion that even brief, intense compressive stress may cause serious motor involvement.

Coexisting bilateral Carpal Tunnel Syndrome in this patient emphasizes the need to examine for multiple entrapments; sometimes, symptoms may be misattributed or confounded if bilateral median nerve slowing is ignored.

Prognosis in this case was excellent with conservative management alone (splints, physiotherapy, short-course steroids), consistent with neuropraxia. The absence of spontaneous denervation potentials on EMG, rapid recovery, and isolated motor branch involvement support a favorable outcome.

In comparison, many prior cases either had mixed sensory-motor involvement, chronic onset, or required more aggressive interventions. For example, in studies by Maimaris & Zadeh (1990) and Sparapani et al. (2020), the clinical picture included sensory signs. The functional recovery timelines in such cases were also often longer.

Learning Points

Cyclist's palsy may occur even after relatively short-duration rides; awareness of posture, grip, and proper equipment is important.

Early and detailed electrodiagnostic testing—especially focusing on distal latencies, F-wave studies, and segmental stimulation—can help localize lesions, differentiate motor vs sensory involvement, and detect conduction blocks.

Conservative management may suffice in isolated deep motor branch lesions (neuropraxia) when diagnosed early, with a good prognosis.

Conclusion

This case of cyclist's palsy showcases the role of a detailed electrodiagnostic evaluation with focus on conduction blocks and prolonged latencies in documenting acute involvement of Ulnar nerve.

Acknowledgments

None.

Competing Interests

The authors declare no conflict of interest.

Ethical Consideration

The case report was prepared with the written informed consent of the patient and approved by the institutional Ethics Committee – Approval number DHR-EC/2024-09/146.

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