Electrophysiological Features of Ulnar Tunnel Syndrome Caused by Ganglion – A Descriptive Study

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Abstract

Objective: Ulnar tunnel syndrome (UTS) is an uncommon ulnar neuropathy. Clinical and electrophysiological diagnosis of UTS is difficult. The purposes of this study were to assess the diagnostic value of the nerve conduction measurements for UTS caused by ganglion, and to investigate the electrophysiological features of UTS.

Methods: The subjects were five patients with UTS. Before surgery, all patients had motor weakness and intrinsic muscle atrophy with positive Froment’s sign, and three patients had numbness and hypesthesia in the ulnar nerve distribution. In all patients, a magnetic resonance imaging (MRI) of the wrist demonstrated soft tissue mass at the ulnar tunnel. The compound muscle action potential (CMAP) from the abductor digiti minimi (ADM) muscle and the first dorsal interosseous (FDI) muscle, and sensory nerve action potential (SNAP) from the little finger were recorded and analyzed. All patients underwent surgery of ulnar tunnel release and excision of the ganglion. Static 2 points discrimination tests (2PD) on the little finger, pinch strength were assessed before and after surgery.

Results: ADM-CMAP and FDI-CMAP were recorded in all five patients and all of them showed abnormality in ADM- and FDI-CMAP. Delayed latency (mean: 5.4 msec) and/or low amplitude (mean: 1.4 mV) were seen for ADM-CMAP and for FDI-CMAP (mean: 7.1 msec, 2.6 mV). SNAP was recorded in four patients and it all showed normal latency and amplitude. After surgery, all patients obtained complete recovery of motor function and sensation. Mean 2PD improved from 7.8 mm to 5.0 mm, and mean pinch strength increased from 1.8 kg to 4.8 kg postoperatively.

Conclusion: Both ADM-CMAP and FDI-CMAP were important for definite electrophysiological diagnosis of ulnar tunnel syndrome caused by ganglion. Residual delayed latency and low amplitude were seen after surgery regardless of complete recovery of intrinsic muscle.

Keywords: Ulnar tunnel syndrome; Ganglion; Electrophysiology; Abductor digiti minimi; First dorsal interosseous

Introduction

Ulnar tunnel syndrome (UTS) is an uncommon ulnar entrapment neuropathy. Guyon [1] described the anatomy of the area in 1861, and Hunt [2] reported three patients of neuritis of the deep palmar branch of the ulnar nerve. Seddon [3] described ulnar nerve palsy due to carpal ganglion, and Richmond [4] reported four cases of ulnar nerve compression caused by a carpal ganglion. In 1965, Dupont et al. [5] described the term ulnar tunnel syndrome and reported four cases. UTS can be caused by a variety of factors, and many previous reports described that one of the most common causes of UTS was a ganglion [3,10,13,15,16]. Magnetic resonance imaging (MRI) provides valuable information for UTS caused by ganglion [15,17]. However, the exact clinical diagnosis is not always easy, and electrophysiological diagnosis is essential to localize the lesion [11-13,15-21]. A few electrophysiological studies for UTS caused by ganglion have been reported [11-17], and they described the delayed conduction at the wrist. Furthermore, there are few electrophysiological reports for UTS caused by ganglion examined before and after surgery [11,15]. The purposes of this study were to assess the diagnostic value of the nerve conduction measurements for UTS caused by ganglion, and to investigate the electrophysiological features of UTS by the data examined before and after surgery.

Patients and Methods

Between December 2010 and July 2016, five upper limbs from five patients with UTS caused by ganglion were reviewed after a mean follow-up of 8 months (range 4-12). Details of five cases are shown in Table 1. The ages of the patients at surgery ranged from 45 to 66 years, with a mean age of 55 years. There were four females and one male.
The affected side was right in three and left in two, the dominant extremity was involved in three. Mean duration of symptoms was 2.4 months (range 1–5). Written informed consent was obtained from each patient. UTS caused by ganglion was diagnosed based on clinical symptoms, MRI findings and electrophysiological evaluation. All patients had motor weakness and intrinsic muscle atrophy with positive Froment’s sign, and three patients had numbness and hypesthesia in the ulnar nerve distribution. Intrinsc muscles include the abductor digiti minimi (ADM) muscle and the first dorsal interosseous (FDI) muscle. Tinel’s sign at the ulnar tunnel was not seen in any of these patients. Static 2 points discrimination test (2PD) on the little finger ranged from 5 to 12 mm, with a mean of 7.8 mm (SD: standard deviation 3.1). Pulp pinch strength revealed from 0.5 to 3.2 kg, with a mean strength of 1.8 kg (SD: 1.0). In all patients, MRI of the extremity was involved in three. Mean duration of symptoms was 2.4 months (Mo: Months).

Electrophysiological evaluation included motor and sensory nerve conduction measurements were performed before and after surgery. The compound muscle action potential (CMAP) from ADM and FDI, and sensory nerve action potential (SNAP) from the little finger were recorded and analyzed. Observation was made using a Nicolet Viking electromyography system (Nicolet instruments, Madison, WI, USA). Palmar skin temperatures were not allowed to fall below 32. ADM- and FDI-CMAP were recorded with surface electrodes by supramaximal stimulation of the ulnar nerve at the wrist with no conduction delay at the cubital tunnel. All patients underwent surgery of ulnar tunnel release and excision of a ganglion. At surgery, we identified the site of compression by a ganglion within the ulnar tunnel in each case, and classified them into three zone [10]. Zone 1 is the area proximal to the bifurcation of the ulnar nerve, Zone 2 compress the motor branch of the nerve after it has bifurcated, and Zone 3 carries the superficial or sensory branch of the ulnar nerve. Static 2 points discrimination (2PD) test on the little finger, pinch strength and Froment’s sign were assessed after surgery. Complications including hematoma, infection and nerve injury were also examined. The data were analyzed with the Student’s t-test and the Mann-Whitney U test. A p-value less than 0.05 was considered statistically significant.

### Results

Before surgery, ADM-CMAP and FDI-CMAP were recorded in all five patients and they all showed abnormality in ADM- and FDI-CMAP. Delayed latency (mean: 5.4 ms, SD: 3.8) and or low amplitude (mean: 1.4mV, SD: 1.4) were seen for ADM-CMAP and for FDI-CMAP (mean: 7.1 ms, SD: 3.2, mean: 2.6 mV, SD: 4.6). Specifically, case 2 with a large ganglion showed largely delayed latency (11.8 ms) and very low amplitude of ADM-CMAP (0.2 mV) and FDI-CMAP (12.6 ms, 0.1 mV). SNAP were recorded in four patients and they all showed normal latency and amplitude (Table 2).

At surgery, after releasing the ulnar tunnel by division of the volar carpal ligament, compression in Zone 1 was seen in four patients, in which a ganglion was compressing the motor branch (to ADM, FDI) and the sensory branch of the ulnar nerve proximal to the fibrous arch, and Zone 2 was in one, in which a ganglion was mainly compressing the motor branch (to FDI) at the fibrous arch (Table 1). There was no case in Zone 3. A ganglion rose from the triquetromate joint in all cases of ulnar tunnel syndrome caused by ganglion (F: Female; M: Male; R: Right; L: Left; m: Month(s); 2PD: 2 Points Discrimination Test on Little Finger; Pinch: Pulp Pinch Strength; Zone: Site of Compression; Size: in coronal (length/width/) and axial (thickness) MRI (mm); Mo: Months).

### Table 1: Details of five cases of ulnar tunnel syndrome caused by ganglion (F: Female; M: Male; R: Right; L: Left; m: Month(s); 2PD: 2 Points Discrimination Test on Little Finger; Pinch: Pulp Pinch Strength; Zone: Site of Compression; Size: in coronal (length/width/) and axial (thickness) MRI (mm); Mo: Months)

<table>
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<tr>
<th>Case</th>
<th>Age</th>
<th>Gender</th>
<th>side</th>
<th>Duration of symptoms</th>
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<th>Pinch kg</th>
<th>Zone (size) of ganglion</th>
<th>FollowUp</th>
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<td>3.2</td>
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<td>F</td>
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<td>3</td>
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<td>L</td>
<td>3 m</td>
<td>7</td>
<td>0.5</td>
<td>1 (7/7/8)</td>
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<tr>
<td>4</td>
<td>66</td>
<td>F</td>
<td>L</td>
<td>2 m</td>
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<td>2 (8/5/5)</td>
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<tr>
<td>5</td>
<td>56</td>
<td>M</td>
<td>R</td>
<td>1 m</td>
<td>5</td>
<td>2.1</td>
<td>1 (8/8/11)</td>
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Parameter measurements before / after surgery

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<tr>
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After surgery, all patients obtained complete recovery of motor function and sensation. Mean 2PD improved from 7.8 mm to 5.0 mm (SD: 0, no significance), and mean pinch strength increased from 1.8 kg to 4.8 kg (SD: 1.5, p<0.02), with negative Froment’s sign. Postoperative ADM- and FDI-CMAP showed the shortening of latency (mean: ADM- 3.3 ms, SD: 0.6, FDI- 4.6 ms, SD: 0.6, no significance) and the increase of amplitude (mean, ADM- 4.0 mV, SD: 2.3, FDI- 4.7 mV, SD: 4.9, no significance). However, they did not recover to the normal range, except ADM-latency in one, ADM-amplitude in two, FDI-latency in one, and FDI-amplitude in three (Table 2).

Illustrative case

A 56-year old right hand dominant male presented with one month history of onset and intrinsic weakness of his right hand (case 5). Right hand showed intrinsic muscle atrophy and weakness with positive Froment’s sign, and clawing of the little finger, without sensory loss. 2PD was 5 mm, and pinch strength 2.1 kg. Axial T1 weighted MRI showed cystic mass lesion at the ulnar tunnel (Figure 1).

ADM-CMAP revealed normal latency (2.7 ms) and low amplitude (0.6 mV) (Figure 2A), and FDI-CMAP showed delayed latency (6.4 ms) and low amplitude (1.5 mV) (Figure 3A), nevertheless SNAP indicated normal latency and amplitude. Intra-operatively a 8/8/11 mm ganglion was mainly compressing the motor branch of the ulnar nerve in Zone 1 (Figure 4). The ganglion was excised and histology confirmed diagnosis. Six months after surgery, complete recovery of motor function was obtained with pinch strength 7.4 kg, ADM- and FDI-CMAP showed the shortening of latency and the increase of amplitude (Figure 2B and Figure 3B).

Table 2: Details of electrophysiological data of five cases (ADM: Abductor Digiti Minimi; CMAP: Compound Muscle Action Potential; FDI: First Dorsal Interosseous; Lat.: Latency (msec); Amp.: Amplitude (mV,μV); SNAP: Sensory Nerve Action Potential; N.D.: Not Done; _: Normal range).

<table>
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<tr>
<th>Case</th>
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<th>side</th>
<th>Lat.</th>
<th>Amp.</th>
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<td>L</td>
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<tr>
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<td>1.5/12.2</td>
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</table>

Discussion

Ulnar tunnel syndrome (UTS) is distinctly uncommon ulnar entrapment neuropathy. UTS can be caused by a variety of intrinsic and extrinsic factors [7,11,14,17], and the literature reported that a ganglion was one of the most common causes of UTS [3-10,13,15,16]. MRI provides valuable information for UTS caused by ganglion [15,17]. However, the exact clinical diagnosis and the location of lesion can be difficult, and electrophysiological diagnosis may help to confirm the diagnosis and to localize the level of neuropathy [11-13, 15-21].
Electrodiagnostic data should include conduction velocity across the elbow as well as the wrist to rule out cubital tunnel syndrome [17], therefore, we performed the nerve conduction measurements including ADM-CMAP, FDI-CMAP and SNAP to rule out the cubital tunnel syndrome and to confirm the diagnosis of ulnar tunnel syndrome.

Anatomically, ulnar tunnel is classified into three zone [10]. Zone 1 is the area proximal to the bifurcation of the ulnar nerve. Zone 2 affects the motor branch (except the branch to ADM) of the ulnar nerve after it has bifurcated, and Zone 3 compress the superficial or sensory branch of the ulnar nerve. Depending on the site of compression the entrapment may be motor, sensory or mixed clinically [16]. In our series, Zone 1 was in four cases and Zone 2 was in one, however, case 5 with Zone 1 showed no sensory loss.

According to the anatomical Zones, ADM-CMAP, FDI-CMAP and SNAP indicate following data theoretically. In Zone 1 compression, SNAP amplitude are diminished and ADM- , FDI-CMAP are normal [17]. In our series of five cases, four cases of Zone 1 showed delayed ADM-latency in three, low ADM-amplitude in four, delayed FDI-latency in four, low FDI-amplitude in three, and SNAP was normal in three. Case 4 with Zone 2 lesion indicated delayed latency and low amplitude in ADM- and FDI-CMAP, and normal SNAP (Table 2). Therefore, both ADM- and FDI-CMAP were important for definite diagnosis of UTS and SNAP was not so valuable to confirm the diagnosis. On the other hand, there have been several reports concerning the short segment incremental study (SSIS, inching method) of FDI-CMAP [18-21], and described that SSIS was valuable for diagnosis of precise localization of UTS. However, SSIS was somewhat time-consuming and technically difficult, particularly palmar sites of stimulation [20], therefore we performed traditional nerve conduction measurements of FDI-CMAP and ADM-CMAP.

A few electrophysiological studies for UTS caused by ganglion have been reported [11-16], and they described the delayed conduction at the wrist. Furthermore, there are few electrophysiological reports for UTS caused by ganglion examined before and after surgery [11,15]. Ebeling et al. [11] reported one case with a ganglion among nine cases of UTS, and described that ADM-latency shortened from 4.4 ms to 2.9 ms, and FDI- latency shortened from 20.5 ms to 4.4 ms postoperatively. Erkin et al. [15] reported a case with a ganglion, and stated that FDI-latency shortened from 3.5 ms to 3.2 ms (normal value<3.2), and FDI-amplitude increased from 2.1 mV to 5.4 mV (normal value=6.0) postoperatively. In our series, ADM- and FDI-CMAP did not recover to the normal range postoperatively, except ADM-latency in one, ADM-amplitude in two, FDI-latency in one, and FDI-amplitude in three (Table 2). Regardless of complete recovery of intrinsic muscle after surgery, residual delayed latency and low amplitude were seen. In these cases, to explain neurophysiologically, myelinization and axonal regeneration of fibers in ADM and FDI branch would not be sufficient after a mean follow-up of 8 months.

There were several limitations with this study. First, number of subjects was small (five patients). Second, we could not clear the relationship between the size of ganglion and the electrophysiological data or the recovery time for intrinsic muscles. Third, mean follow-up period of 8 months was not so long, therefore, long-term follow-up would be needed to detect the recovery of ADM-CMAP and FDI-CMAP.

Conclusion

Both ADM-CMAP and FDI-CMAP were important for definite electrophysiological diagnosis of ulnar tunnel syndrome caused by ganglion. Residual delayed latency and low amplitude were seen after surgery.

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References


