An acute handshake ulnar mononeuropathy

Case Presentation

A Saudi male aged 48 years presented to the accident and emergency with a history of right upper limb weakness and numbness for less than 2 weeks. His symptoms started suddenly after shaking hands with a senior military official. There was no history of trauma, fever, or any form of sickness prior to the incident. However, he is a known case of Graves’ disease on carbimazole. He felt palpitations and sweating of the hands 3 weeks earlier and took a few doses of carbimazole by himself. On examination, he was fully conscious/oriented with normal higher mental function and cranial nerves. The right upper limb had a claw hand deformity with weakness of the intrinsic small muscles of the hand including adductor pollicis, sparing the thenar eminence muscles. Power in the wrist, elbow, and shoulder along with their reflexes were normal. There was a reduced pinprick and touch sensation in the distribution of the C8 T1 dermatomes.

Investigations. A CT brain, MRI brain, and cervical spines, complete blood count, erythrocyte sedimentation rate, and CSF were unremarkable. Antinuclear antibodies were positive. However, antineutrophil cytoplasmic antibody (ANCA) was negative.

Neuro-physiological study. Sensory and motor conduction study was carried out using standard techniques. The abnormal findings of the right ulnar are summarized in Table 1.

<table>
<thead>
<tr>
<th>Nerve and site</th>
<th>Latency (msec)</th>
<th>Amplitude (mV)</th>
<th>Conduction velocity (m/sec)</th>
<th>F. Wave (msec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ulnar right wrist (m)</td>
<td>3.3</td>
<td>0.4</td>
<td></td>
<td>27.1</td>
</tr>
<tr>
<td>Below elbow (m)</td>
<td>6.4</td>
<td>0.7</td>
<td>56</td>
<td></td>
</tr>
<tr>
<td>Above elbow (m)</td>
<td>8.5</td>
<td>0.7</td>
<td>48</td>
<td></td>
</tr>
<tr>
<td>Axilla (m)</td>
<td>10.0</td>
<td>0.7</td>
<td>100</td>
<td></td>
</tr>
<tr>
<td>Supraclavicular fossa (m)</td>
<td>13.1</td>
<td>0.7</td>
<td>48</td>
<td></td>
</tr>
<tr>
<td>Right ulnar (s)</td>
<td>NR</td>
<td></td>
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</tbody>
</table>

m - motor, s - sensory, NR - no response

The sensory-motor conduction of the other nerves for the upper and lower limbs were within normal limits.

Needle examination. The right abductor digitii minimi (ADM), first dorsal interosseus (FDI) and flexor carpi ulnaris (FCU) were extensively studied using techniques for quantitative electromyography. Here, there was no spontaneous activity. No motor unit action potentials (MUAPs) could be recruited from the ADM. The other 2 muscles showed markedly reduced recruitment of MUAPs. However, the recruited units were normal in configuration.
Neurosciences Quiz

Questions

Based on his abnormal electro diagnostic test parameters (Table 1):

1. What was the type of lesion and why?
2. Why was the spontaneous activity absent (if it is axonal loss)?
3. Could it be a case of C8-T1 radiculopathy?
4. Could it be a case of non-localizing ulnar mono-neuropathy?
5. What could be the etiological cause of the unilateral ulnar nerve palsy?

The patient was given pulse steroids (methyl prednisolone), 1 gm intravenously, once daily for 5 days. His symptoms and power slightly improved. The diagnosis was drug induced ulnar mononeuritis.

Answers

1. Axonal loss. There was absence of sensory nerve action potentials (SNAP) (Figure 1) and low amplitude compound muscle action potentials (CMAPs) for the right ulnar nerve (Table 1) with relatively normal distal motor latency and conduction velocity. These findings indicate that a Wallerian degeneration with axonal loss had taken place.

2. Absence of spontaneous activity in the small muscles of the right hand and flexor carpi ulnaris (FCU) instead of axonal loss might be due to a time factor. It is well known that appearance of the denervation potentials depends on the length of the nerve between muscles being studied and the site of the lesion. For example, a lesion of L5-S1 roots may take 5-6 weeks for the distal leg and foot muscle to develop fibrillation potentials.1

3. A C8-T1 radiculopathy was unlikely, because of the absence of right ulnar SNAPs (Figure 1). Absence of SNAPs suggests that the lesion must be at or distal to the dorsal root ganglia of the spinal nerves. Moreover, presence of normal SNAP for the right medial antebrachial cutaneous nerve may also suggest sparing of the medial cord.
4. Non-localizing ulnar mononeuritis is the most suitable diagnosis, because of the absence of conduction block with uniform CMAPs amplitude decrease, while the right ulnar nerve was stimulated at different sites along its course such as at the wrist, around the elbow, axilla, and supraclavicular fossa (Table 1).

5. Propylthiouracil (carbimazole) treatment of Graves’ disease had been postulated to provoke an ANCA-associated vasculitis, resulting in non-localizing ulnar mononeuritis.

Discussion

Non-localizing neuropathy is a familiar pattern in the clinical EMG laboratory. Nerve conduction and EMG findings are involved in one nerve only. The involved nerve may have sensory motor axonal loss where the CMAPs and SNAPs are low in amplitudes without focal slowing and/or conduction block. The distal latencies and conduction velocities are slightly slowed. Needle examination showed neuropathic abnormalities.1

The patient taking carbimazole 3 weeks prior to the onset of his acute symptoms might have resulted in an immune-mediated vasculitis and ischemic neuritis of the right ulnar nerve.2,3

References