
Hypotheses explaining CMUA include an antibody-mediated channelopathy,1 ephaptic transmission at sites of demyelination,2 and nerve hyperexcitability in newly formed unmyelinated collaterals. CMUA in CIDP has been reported to respond to both immunosuppression2 and sodium channel antagonists.1

**AUTHOR CONTRIBUTIONS**

Dr. Chhibber: drafting/revising the manuscript, acquisition of data. Dr. Greenberg: drafting/revising the manuscript, study concept or design, analysis or interpretation of data, acquisition of data.

**REFERENCES**


**Table**

Nerve conduction studies in patient showing features of CIDP

<table>
<thead>
<tr>
<th>Motor nerve</th>
<th>Stimulation site</th>
<th>Distal motor latency, ms</th>
<th>Amplitude, mV</th>
<th>Conduction velocity, m/s</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left median</td>
<td>Wrist</td>
<td>7.1 (&lt;4.5)</td>
<td>8.7 (&gt;4.0)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Forearm</td>
<td></td>
<td>4.2b</td>
<td>28.6 (&gt;50)</td>
</tr>
<tr>
<td>Right median</td>
<td>Wrist</td>
<td>6.7 (&lt;4.5)</td>
<td>6.8 (&gt;4.0)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Forearm</td>
<td></td>
<td>3.7</td>
<td>22.6 (&gt;50)</td>
</tr>
<tr>
<td>Right ulnar</td>
<td>Wrist</td>
<td>6.3 (&lt;3.5)</td>
<td>3.6 (&gt;5.5)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Below elbow</td>
<td></td>
<td>1.4b</td>
<td>17.6 (&gt;50)</td>
</tr>
<tr>
<td>Right peroneal</td>
<td>Ankle</td>
<td>No response</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Right tibial</td>
<td>Ankle</td>
<td>No response</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

a Motor nerve conduction studies demonstrated prolonged distal motor latency, conduction velocity slowing, and conduction block in multiple nerves in keeping with CIDP. Normal values are listed in parentheses. Sensory nerve conduction studies demonstrated absent right median, ulnar, radial, and sural responses.

b Conduction block was defined as a drop in amplitude of >50% between sites of stimulation.

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**Disclosure** Dr. Chhibber reports no disclosures. Dr. Greenberg receives publishing royalties for *EMG Pearls* (Hanley & Belfus, 2004); has served as a consultant for MedImmune, LLC and receives research support from MedImmune, LLC, the NIH, and the Muscular Dystrophy Association; and has served as a consultant in medico-legal cases regarding zinc-induced copper-deficiency myelopathy.
Teaching Video NeuroImages: Widespread clinical myokymia in chronic inflammatory demyelinating polyradiculoneuropathy
Sameer Chhibber and Steven A. Greenberg
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