Lumbrical Sparing in Severe Carpal Tunnel Syndrome

Lata Kumaraswamy, DO, Vikram Narula, MD, and Michael Andary, MD, MS

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American Association of Neuromuscular and Electrodiagnostic Medicine

2621 Superior Dr NW Rochester, MN 55901

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CME Information
Product: CS25 - Lumbrical Sparing in Severe Carpal Tunnel Syndrome

Course Description
Intended Audience
This course is intended for Neurologists, Physiatrists, and others who practice neuromuscular, musculoskeletal, and electrodiagnostic medicine with the intent to improve the quality of medical care to patients with muscle and nerve disorders.

Learning Objectives
Upon conclusion of this program, participants should be able to:
1. formulate differential diagnosis for bilateral hand numbness.
2. describe nerve conduction study, electromyographic, and clinical findings consistent with carpal tunnel syndrome.
3. explain the significance of sparing of the lumbricals in carpal tunnel syndrome.

Release Date: 10/27/2008
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Duration/Completion Time: 1 hour

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Lumbrical Sparing in Severe Carpal Tunnel Syndrome

October 2008

CME Available from October 2008 through October 2011

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Presenting Symptom: Numbness in Both Hands

Case prepared by: Lata Kumaraswamy, DO, Vikram Narula, MD, Michael Andary, MD, MS

Affiliations: Michigan State University

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Appropriate Audience: Residents, neuromuscular fellows, and practicing physicians.

Learning Objectives: After completing this educational activity, participants will be able to:
1) Formulate differential diagnosis for bilateral hand numbness
2) Describe nerve conduction study, electromyographic, and clinical findings consistent with carpal tunnel syndrome
3) Explain the significance of sparing of the lumbricals in carpal tunnel syndrome

Level of Difficulty: Intermediate
Lumbrical Sparing in Severe Carpal Tunnel Syndrome

History

An 85-year-old female presents with a 5-6 year history of bilateral numbness and tingling in her hands. She reports her symptoms are worse at night, but seems to improve when she wears hand splints.

Commentary I

At this time, the differential diagnosis includes:

1. Carpal tunnel syndrome (CTS) is one of the most common entrapment neuropathies encountered. This is a likely diagnosis considering the paresthesias of the hands, nocturnal paresthesias and alleviation of discomfort with wrist splint at night. Patients usually describe having numbness and tingling in the distribution of the thumb, index finger, long finger, and lateral ring finger.

2. A C6-C8 cervical radiculopathy can be considered as part of the differential because of the paresthesias in the hands. However, with a cervical radiculopathy patients usually report neck pain with radiation of symptoms down the arm.

3. Cervical myelopathy from cervical stenosis is a strong consideration for bilateral numb hands. Myelopathy (spinal cord compression) with or without coexistent radiculopathy are both considerations. The presence of upper motor neuron signs, such as extensor plantar responses, spastic hypertonia, and gait abnormality (gait ataxia) should be looked for specifically. Cervical spondylotic myelopathy (CSM) is the most common form of myelopathy in older adults.

4. A peripheral polyneuropathy is a possibility since the patient describes having bilateral hand paresthesias. Typically there is a stocking glove distribution description of symptoms starting distally. That is, individuals usually have numbness and tingling in the feet that progresses up the legs. At this point, the paresthesias typically start to involve the fingers and hands. The key description that points us to the possibility of peripheral polyneuropathy is the symmetry of her symptoms.

5. A brachial plexopathy should be considered and patients will have symptoms including pain in the shoulder or upper arm, muscle weakness, atrophy and sensory loss. Etiologies may be due to trauma, metastatic cancer, lymphoma, thoracic outlet syndrome or idiopathic brachial neuritis (Parsonage-Turner syndrome).

History, Continued

Upon further history, the patient denies having neck pain or hand weakness, but she does note difficulty manipulating small objects. Her past surgical history is significant for a remote history of right carpal tunnel release and breast cancer with subsequent lumpectomy.

Commentary II

With the history of difficulty manipulating objects and absence of neck pain, one should still have carpal tunnel syndrome higher in the differential. Patients exhibit symptoms when the wrist is either in a flexed or extended position, and typically involve activities such as, holding a cup, reading the newspaper or
driving. In most individuals, sensory fibers are affected first. As the disease progresses, motor fibers become involved and weakness of the thumb abduction and opposition may arise along with thenar eminence atrophy.[14] As a result, patients often report difficulty with opening jars or turning doorknobs. The patient's clinical symptoms are not classic for a cervical radiculopathy because the symptoms are primarily in the hands. Patients with cervical radiculopathy usually have some symptoms proximal to the wrist or in the neck, however this cannot be ruled out until we have data from the physical examination and electrodiagnostic testing. Peripheral neuropathy could still be in the differential as her symptoms are symmetrical. Additionally, with a history of breast cancer, brachial plexopathy continues to be in the differential. Neurogenic thoracic outlet syndrome is unlikely secondary to lack of sensory changes in the fourth and fifth digits, medial hand and medial forearm and more thenar muscle atrophy compared to the hypothenar muscles. In neurogenic thoracic outlet syndrome that involves atrophy of the thenar (C8-T1) muscles there should also be sensory changes in the dermatomes of C8-T1. The sensory complaints are more diffuse in this patient.

Physical Examination

Spurling's Test: negative bilaterally producing no paresthesias in the upper extremities.

Tinel's Test: this provocative maneuver was performed by tapping the median nerve at the palmar surface of the wrist and the ulnar nerve at the elbow for about 10 seconds. This was negative bilaterally at the wrist and elbow producing no paresthesias.

Phalen's Test: Maintain flexed wrist position for 60 seconds. We didn’t perform the reverse Phalen's test.

Jobe’s Test, Neer’s Test and the Hawkin’s Kennedy Test: demonstrated no shoulder pain or signs of impingement.

Reflexes: Hoffman’s sign was negative bilaterally. Plantar responses were flexor bilaterally, i.e., Babinski sign was absent.

Muscle stretch reflexes: graded 2+ in the biceps, triceps and brachioradialis in both upper extremities using the National Institute Neurological Disorders and Stroke (NINDS) scale.

Muscle testing:

Manual muscle testing revealed:

<table>
<thead>
<tr>
<th></th>
<th>Deltoid</th>
<th>Supraspinatus</th>
<th>Biceps</th>
<th>Triceps</th>
<th>Wrist Extensor</th>
<th>First Dorsal Interossei</th>
<th>Abductor Pollicis Brevis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right</td>
<td>4/5</td>
<td>4/5</td>
<td>5/5</td>
<td>5/5</td>
<td>5/5</td>
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<td>5/5</td>
<td>5/5</td>
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</table>

Commentary III

The history and physical examination findings include: bilateral hand numbness, nocturnal paresthesias, symptoms worse at night and relief of discomfort with a wrist splint, difficulty manipulating objects, normal muscle stretch reflexes, right shoulder weakness and bilateral abductor pollicis brevis muscle weakness. The Tinel’s sign was negative on our exam. Durkan reported that Tinel’s sign at the carpal tunnel has a
sensitivity of 56% in those patient’s with electrodiagnostically confirmed carpal tunnel syndrome.[5] The diagnosis of carpal tunnel syndrome is likely with the clinical presentation. We must still consider a cervical radiculopathy or brachial plexopathy as part of our differential because of right shoulder weakness and abductor pollicis brevis muscle weakness and paresthesias bilaterally. Normal muscle stretch reflexes could still occur in the setting of either C5 or C8/T1 radiculopathy. The two most common causes of secondary neoplastic brachial plexopathy are primary or metastatic disease to the upper lobe of the lung (Pancoast tumor) and breast cancer.[3]

A peripheral neuropathy is still a possibility because of the symmetric nature of the patient’s presentation. A cervical myelopathy is unlikely as demonstrated by the lack of upper motor neuron signs. Electrodiagnostic testing will serve as an extension of our history and physical examination.

Electrophysiologic Data

<table>
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<tr>
<th>SENSORY NERVE CONDUCTION STUDIES</th>
<th>NERVE</th>
<th>SIDE</th>
<th>STIM SITE</th>
<th>RECORD</th>
<th>cm</th>
<th>AMPL microvolts</th>
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</tr>
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<th>Stim Site</th>
<th>Record</th>
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<th>Amplitude millivolts</th>
<th>Lat msec</th>
<th>CV M/sec</th>
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<td>Thenar</td>
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<td>Thenar</td>
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<td>2.9</td>
<td></td>
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Note: The ulnar motor to the thenar recording is a volume conducted compound muscle action potential from the ulnar muscles under the thenar eminence. It depends on the muscles innervated by the ulnar nerve (flexor pollicis brevis, adductor pollicis, intrinsics, or even opponens and abductor pollicis brevis if there is Riche-Cannieu or Martin-Gruber anastomosis).
**NEEDLE ELECTROMYOGRAPHY**

Insertional activity: N, sust, unsust
FIB: 0, 1+, 2+, 3+, 4+
OTHER: 0 or fascic, myotonia, myokymia
EFFort: N, decr
RECRuitment: N, inc or dec 1+, 2+, 3+, 4+
AMPplitude: N, inc or dec 1+, 2+, 3+, 4+
DURation: N, inc or dec 1+, 2+, 3+, 4+
POLyphasia: N, inc or dec 1+, 2+, 3+, 4+

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<th>FIB</th>
<th>OTH</th>
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<th>AMP</th>
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<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
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</tbody>
</table>

Only the ABP and FDI were recruited on our needle exam.

The ABP had decreased insertional activity and no motor units. We did not find fibrillations.

**Diagnostic Impression**

1. Severe left carpal tunnel syndrome - At first glance, this appears to be a complete lesion (i.e. no measurable median nerve function distal to the carpal tunnel). Thus all the axons have severe axonotmesis or complete neurapraxia.

2. Right median neuropathy at the wrist - This may represent residual slowing from the old carpal tunnel syndrome due to latency delay across the carpal tunnel which is still present following the carpal tunnel release in the past. The persistent conduction slowing may be the result of a lack of normalization of myelin thickness with an increased number of internodes.[4] The patient's history, physical examination,
and electrodiagnostic findings were consistent with residual slowing from the old carpal tunnel syndrome similar to that reported by Naidu.\[10\]

Documenting some median nerve axon sparing in the left carpal tunnel might give hope for improvement from surgery. Testing the lumbrical function in the median nerve has potential to document this. Although it is not certain that some spared axons improves prognosis, there is an indication and some opinions that an incomplete lesion may have a better prognosis. In 2007, Brannegan reported the second lumbrical fibers were protected from Wallerian degeneration in severe carpal tunnel syndrome with conduction slowing demonstrated by delayed distal latencies to the second lumbrical (mean, 9.1), seen in cases he analyzed.\[2\] It is not clearly understood why the second lumbrical fibers are affected early in mild carpal tunnel syndrome showing demyelinating changes, slow conduction velocity and resistance to axon injury in advanced compression.\[2\] Nobuta looked at 46 patients with severe carpal tunnel syndrome following carpal tunnel release. Compound muscle action potential distal latencies and amplitudes from the abductor pollicis brevis and second lumbrical were recorded before and after surgery.\[11\] He reported the second lumbrical was an important prognostic indicator for severe carpal tunnel syndrome and if that the distal latency was 10ms or greater, myelination of fibers would not be sufficient for surgery.\[11\]

Naidu looked at 50 patients who underwent carpal tunnel release. Pre-operative and 6 month post-operative nerve conduction studies were performed revealing distal motor latency and sensory amplitude improvement following surgery. However, sensory nerve conduction velocity remained slow despite improvement after surgery.\[10\]

Thus, lumbrical sparing may suggest improvement of carpal tunnel syndrome following surgical repair.

**ELECTROPHYSIOLOGIC DATA, CONTINUED**

<table>
<thead>
<tr>
<th>NERVE</th>
<th>SIDE</th>
<th>STIM SITE</th>
<th>RECORD</th>
<th>cm</th>
<th>AMPLITUDE</th>
<th>LAT</th>
<th>CV</th>
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</thead>
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</table>

**DIAGNOSTIC IMPRESSION CONTINUED**

1. There are electrodiagnostic abnormalities most consistent with a severe left carpal tunnel syndrome with axon loss. Motor units to the abductor pollicis brevis could not be found, but a few slow axons to the lumbricals were present suggesting an incomplete lesion.

2. The right median neuropathy is probably improving, but we cannot say for sure without comparison to the old EMG.

**Commentary V**

There are two issues that make this particularly interesting for carpal tunnel syndrome. These are:

1. The severity of the left CTS with absent median motor to the thenar muscles and absent SNAPs. The lumbrical sparing is helpful to clearly document some slowing in the carpal tunnel.
2. The residual slowing in the right carpal tunnel despite successful carpal tunnel release can be a diagnostic challenge.
The carpal tunnel is bound dorsally by the carpal bones and volarly by the transverse carpal ligament (flexor retinaculum). It contains the median nerve, four tendons of the flexor digitorum profundus, four tendons of the flexor digitorum superficialis and one tendon of the flexor pollicis longus.

Etiologies for carpal tunnel syndrome include, diabetes mellitus, hypothyroidism, amyloidosis, pregnancy, rheumatoid arthritis and cryptogenic with mechanisms including edema, fibrosis, and vascular sclerosis which results in compression of the median nerve causing ischemia, demyelination and in severe cases, Wallerian degeneration and axonal loss.[14] Risk factors for development of carpal tunnel syndrome include occupations that involve repetitive hand motions and vibration, female gender, increased body mass index, increasing age, and narrow wrist diameter.

Carpal tunnel syndrome usually presents with specific symptoms of pain and paresthesias in the median nerve distribution. In severe cases, weakness of the median innervated hand muscles is evident. Additionally, patients may complain of wrist pain, clumsiness and dropping small items. Although pain is usually prevalent in the hand or wrist, it may present in the shoulder, elbow and forearm. Symptoms are precipitated by overuse of the hand with repetitive hand, wrist, and grip movements. Patients often shake their hand to relieve nocturnal paresthesias (flick sign).

Clinical evaluation includes obtaining a good history and physical examination of the patient. Provocative maneuvers (Tinel’s sign and Phalen’s test) can elicit paresthesias in the distribution of the median nerve of the hand and should be performed with manual muscle testing, muscle stretch reflexes and Spurling’s test.

Electrodiagnostic testing can help differentiate potential etiologies of pain and paresthesias in a person’s hands. Typically, electrodiagnostic testing in carpal tunnel syndrome demonstrates slowing of the distal latency in median sensory and motor fibers across the wrist. In severe cases, Wallerian degeneration of the median sensory and median motor fibers to the abductor pollicis brevis may result in an absent response with stimulation. As a result, one is unable to localize the lesion to the wrist.[2] Further testing with median motor to the lumbricals may improve localization in severe carpal tunnel syndrome.[2] There have been several reports by Brannegan, Kaul, Loggigan, and Perroto showing the preservation of median motor fibers to the lumbricals in the face of absent motor conduction to the abductor pollicis brevis. This has possible clinical implications because some preservation of motor fibers to the lumbricals suggests an incomplete lesion of the median nerve. An incomplete lesion refers to either neuropraxia or axonotmesis. Neuropraxia results in conduction block across the affected segment, which is reversible. Axonotmesis involves disruption of the axon, with preservation of the perineurium, endoneurium, and epineurium. With axonotmesis, the prognosis is better than neurotmesis.[1] Interestingly, Sunderland’s classification does not address or classify the most common pathology seen in CTS; demyelination with slowing, and no neuroapraxia. This suggests that the traditional classification system could use modification to address demyelination without neuroapraxia. An incomplete lesion may indicate a better prognosis for surgery by proving at least one or more median nerve axons are still functioning.

The etiology for the right hand dysesthesias is difficult to clearly delineate. The mild median slowing across the carpal tunnel is most consistent with residual slowing seen after successful carpal tunnel release.[10] However, it is impossible to completely rule out recurrent carpal tunnel syndrome.

Bibliography