Medical Research Archives

ESMED
European Society of Medicine

OPEN ACCESS

Published: May 31, 2023

Citation: Soriano E, 2023. Challenges in the Evaluation of the Patient Presenting with Isolated Neurological Hand Dysfunction: A Case Series, Medical Research Archives, [online] 11(5). https://doi.org/10.18103/mra.v11i5.3797

DOI
https://doi.org/10.18103/mra.v11i5.3797

ISSN: 2375-1924

CASE REPORT

Challenges in the Evaluation of the Patient Presenting with Isolated Neurological Hand Dysfunction: A Case Series

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Purpose of article: This article is targeted primarily for primary care physicians with the goal of reviewing taking proper history and directed physical examination for patients presenting with neurological hand dysfunction. It is beyond the scope of this article to cover every etiology of neurological hand dysfunction but reviews the common causes and presents four cases seen in a rural Physical Medicine and Rehabilitation (PM&R) clinic.

ABSTRACT
Isolated neurological hand dysfunction including symptoms of weakness, numbness, clumsiness and dystonia are not uncommon complaints presenting to physicians in primary care as well as to neurologists, physiatrists, orthopedists and neurosurgeons. Etiologies of neurological hand dysfunction range from lesions of the central and peripheral nervous system pathways as well as the neuromuscular junction and muscle. In this article we present a series of four patients referred to a rural Physical Medicine & Rehabilitation presenting with similar symptoms of neurological hand dysfunction that ultimately had very different diagnoses. The goal of this article is to review the appropriate history, physical and examination findings and pertinent neuroanatomy needed to direct the clinician towards the workup needed to accurately diagnose and treat neurological hand dysfunction.

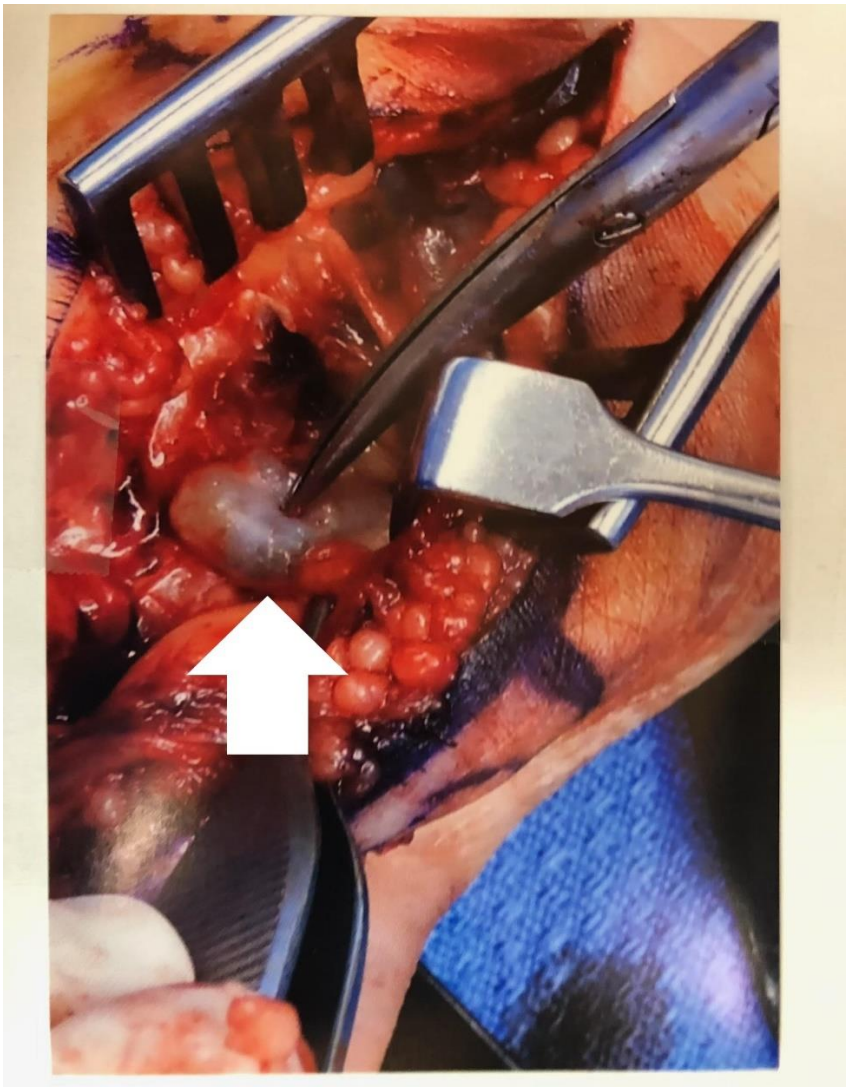
Introduction:

For even an experienced neurologist or physiatrist, the diagnostic workup of a patient presenting with hand weakness, numbness or dysfunction may be challenging. In the primary care setting, patients will frequently present with isolated neurological hand symptoms. Patients presenting with neurological hand dysfunction (NHD) may be a result of various central and/or peripheral nerve insults.[1] Central nervous system (CNS) etiologies include small isolated cortical infarctions, cortical basilar degeneration, multiple sclerosis, myasthenia gravis and cervical myelopathy. [1,2,3] Peripheral nervous system (PNS) etiologies include amyotrophic lateral sclerosis (ALS), multifocal motor neuropathy, cervical radiculopathy, thoracic outlet syndrome, brachial plexopathy, distal myopathy, peripheral entrapment neuropathy as well as other less common etiologies [1, 4, 5, 6, 7, **8**, **9**]. A comprehensive review of all the CNS disorders, myopathies and diseases of the neuromuscular junction causing NHD are beyond the scope of this article so we will instead focus on the more common etiologies of NHD resulting from insults involving the spinal cord, the anterior horn cell and down to the neuromuscular junction. We present in this article four cases of patients presenting with hand weakness, paresthesias and/or clumsiness in a rural PM&R clinic.

Case 1: A 65-year-old right-handed male was referred to the office for consultation and electrodiagnostic testing. Patient reported a 4-month history of what he described as stiffness and pain in the left hand and wrist. He complained of swelling in the wrist and fingers as well as difficulty making a fist or snapping his fingers. Symptoms had been present for approximately 3 months but were progressively worsening. He denied any other neurological symptoms. No swallowing difficulties or speech difficulties. Inspection revealed no muscular atrophy nor evidence of fasciculations. Motor examination revealed weakness with left wrist extension graded as 4/5 as well as 2/5 strength in the median and ulnar innervated muscles of the left hand. Patient was noted to have hyperreflexia in the left upper extremity with positive Hoffmann sign otherwise reflexes normal. Sensory examination was normal to pinprick. Proprioception and vibratory sensation were intact. There was no ankle clonus noted. Babinski sign was downgoing. Patient did have difficulty with tandem gait. Electrodiagnostic testing of the left upper extremity was performed on the day of his initial consultation. Nerve conduction studies (NCS) revealed no evidence of entrapment neuropathy, plexopathy or peripheral polyneuropathy. Needle examination revealed profound abnormalities with significant denervation potentials in both the median and ulnar intrinsic hand muscles as well as in the cervical paraspinal muscles. Initial differential diagnosis was central nervous system pathology including brain and/or spinal cord lesions as well as cervical radiculopathy and motor neuron disease. Cervical and brain MRI were ordered. No significant abnormalities were noted. Additional electrodiagnostic testing on the right upper extremity and paraspinal muscles was performed on the follow-up visit. Abnormalities were noted in the thoracic paraspinal muscles as well as in the intrinsic muscles of the right hand. We did not sample cranial nerve innervated muscles as patient presented with no bulbar signs or symptoms. Patient was referred to a tertiary Academic Center where diagnosis of amyotrophic lateral sclerosis was confirmed.

Case 2: A 53-year-old right-handed male was referred to the clinic for pain management. He presented with a two-week history of pain localized in the left scapula with radiation into the left medial arm and forearm with associated weakness in the left hand. He endorsed numbness and paresthesias in the left arm. Denied any bowel or bladder changes or loss of balance. Inspection revealed no muscle atrophy or fasciculations. Neurological examination revealed 2/5 strength in the intrinsic hand muscles and 4/5 strength with testing of the flexor carpi ulnaris. Patient was unable to snap his fingers. He had difficulty opposing thumb to his 5th digit. 2+ reflexes in the upper extremities bilaterally. Hoffmann sign was positive bilaterally. Spurling's Maneuver was positive on the left. Tandem gait was normal. Differential diagnosis included cervical myelopathy, cervical radiculopathy, brachial plexopathy and entrapment neuropathy. Electrodiagnostic testing was scheduled and MRI of the cervical spine ordered. Electrodiagnostic testing demonstrated mild-to-moderate median nerve entrapment at the left wrist as well as left C8-T1 radiculopathy. MRI of the cervical spine demonstrated a large disc herniation producing moderate mass effect on the spinal cord but also severe neural foraminal stenosis at C8-T1. Patient was referred for neurologic consultation and subsequently underwent cervical decompression with full neurological recovery. He was also referred to orthopedic surgery for management of residual carpal tunnel symptoms.

Case 3: A 63-year-old right-handed male was referred to the clinic for consultation and electrodiagnostic testing. The patient presented with bilateral hand numbness, right side greater than left. Past medical history was significant for a fall where he sprained his right wrist just prior to developing these symptoms. Patient was evaluated in the emergency department after the fall and x-rays were negative for fracture. Patient presented with chief complaint of numbness in both hands right side greater than left. He also complained of swelling of the right wrist with gradually increasing difficulty lifting pots in his job as a chef. Neurological examination revealed definite atrophy of the 1st dorsal interosseous muscle as well as the thenar and hypothenar muscles of the right hand. Mild atrophy noted in the left hand although not as severe as the right. Motor testing revealed 5/5 strength with the exception of 3/5 strength in the intrinsic muscles of the right hand and 4/5 strength in the intrinsic muscle of the left hand. Sensory examination revealed decreased pinprick in the 5th digit of the right hand. Reflexes were active and symmetrical at the biceps, triceps and brachioradialis. Hoffmann sign was absent. Tandem gait normal. No ankle clonus noted. Tinel's sign was positive at the wrist and elbow on the right. Electrodiagnostic testing was performed and revealed severe median nerve entrapment at the right wrist as well as severe right ulnar neuropathy at the elbow but also at Guyon's canal. There was also evidence of severe left median nerve entrapment at the wrist and mild left ulnar entrapment at the elbow. No evidence of cervical radiculopathy, myopathy or plexopathy. Patient was referred to Orthopedic surgery for consideration of carpal tunnel release on the right along with right ulnar nerve transposition and exploration of Guyon's canal. Upon surgery, patient was noted to have a large synovial cyst confirmed by pathology in Guyon's canal (Fig 1). He subsequently had carpal tunnel release on the left side as well and made a full recovery.



Case 4: A Seventy-eight-year-old male was referred to our office with chief complaint of bilateral wrist pain and bilateral hand numbness as well as chronic lower back pain and left leg pain resulting from prior lumbar decompression and fusion. He admitted to increasing difficulty with fine motor skills. Past medical history significant for carpal tunnel release with no improvement in his symptoms. Neurological examination revealed 5/5 strength with hyperreflexia and Hoffmann sign present in the bilateral upper extremities. Sensory examination was intact to pinprick, vibration and proprioception. No ankle clonus noted. Gait was primarily antalgic due to back and leg pain and tandem gait was difficult to assess. Electrodiagnostic testing was ordered and demonstrated no evidence of significant entrapment neuropathy or cervical radiculopathy. MRI of the cervical spine demonstrated severe central canal stenosis at C4-5 with myelomalacia and cord signal change. Patient was referred for neurosurgical consultation and underwent cervical decompression for cervical myelopathy.

Discussion:

All four of these patients presented with similar symptoms of hand dysfunction including various combinations of weakness, numbness, loss of strength and loss of fine motor skills. Inquiring if the patient is dropping objects, awakening with numbness and/or paresthesias as well as exacerbating activities will assist in directing the physician's examination and ordering appropriate diagnostic tests. For the primary care provider seeing patients with these symptoms, it is important to keep in mind the neurological pathways, specifically differentiating upper and motor lesions, and various pathologies that can lead to what appear to be very similar clinical symptoms (Fig 2). A thorough history and physical examination are critical in helping to determine the etiology and the appropriate diagnostic workup. Patients should always be asked about any loss of fine motor skills, falls or loss of balance. It is important to differentiate central nervous system pathology from peripheral nervous system pathology and be certain to conduct appropriate, simple examination tests including assessing for Hoffman's sign, ankle clonus and tandem gait (Fig 3). These three simple tests can, when positive, point the physician towards a central nervous system lesion or lesion of the alpha motor neuron. [3] Clinicians should inspect the hand and upper extremity including looking for evidence of atrophy, swelling, discoloration or dystonia. Observe the hand and arm for any evidence of course muscular fasciculations. A careful neurological examination including assessing deep tendon reflexes, motor testing pinprick sensation is critical. Pinprick sensation should be performed checking both dermatomal and peripheral nerve pattern. Keep in mind that upper motor neuron lesions typically present with hyperreflexia and increased muscle tone, while lower motor neuron lesions present with loss of muscle tone and hyporeflexia.[10] Significant motor deficits in the absence of any sensory deficits should raise suspicion for possible motor neuron disease. Most commonly this would include amyotrophic lateral sclerosis which typically presents with uselessness of hand, awkwardness with fine motor activities, muscle cramping and possible wasting of muscles, although multifocal motor neuropathies may also present with these symptoms. [11, 12, 13, 14] In the case of motor neuron disease, electrodiagnostic typically show normal sensory nerve conduction studies but evidence of abnormalities on needle examination of the muscles sampled. These patients should be referred to a neuromuscular disease specialist for comprehensive work up.

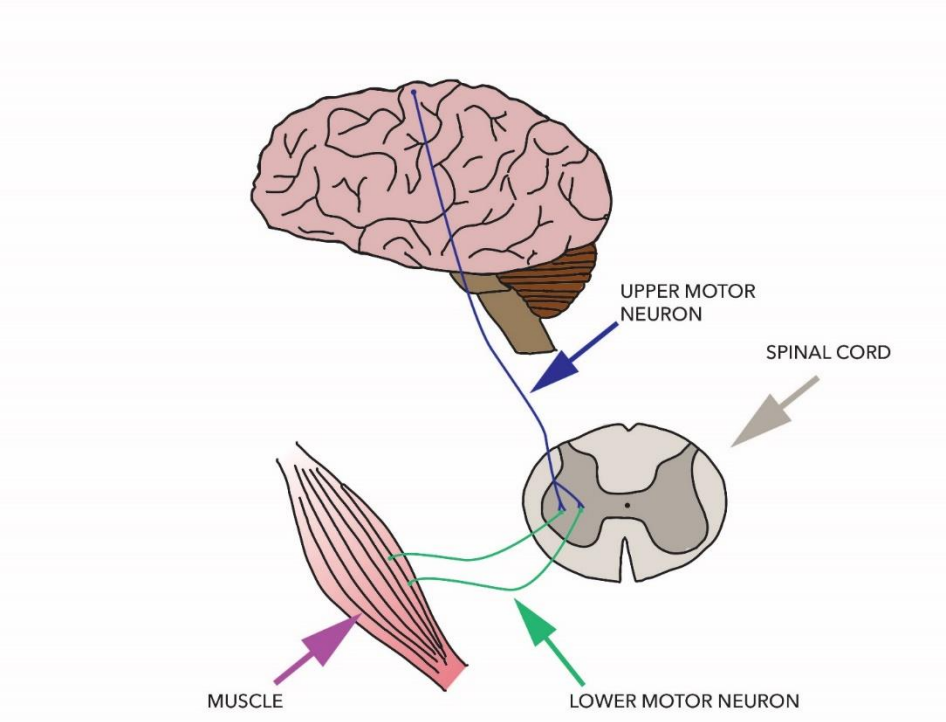


Fig 2

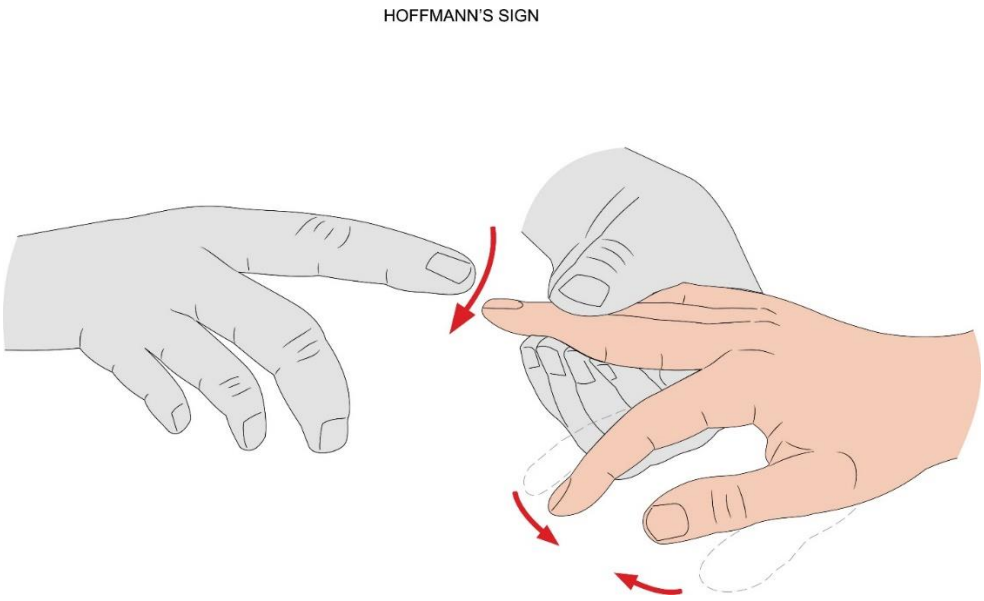


Fig 3

With upper motor neuron findings clinicians should first rule out cervical myelopathy which is certainly much more common than motor neuron disease or other central nervous system pathology. Cervical MRI is indicated to rule out spinal cord compression. If MRI demonstrates spinal cord compression in the presence of neurologic deficits neurosurgery or orthopedic spine consultation is recommended. Patient should be educated that decompression will decrease risk of progression of symptoms. Surgical decompression for the treatment of cervical spondylitic myelopathy was associated with improvement in functional, disability-related, and quality-of-life outcomes. [15]It should be noted that researchers have shown that both cervical cord compression as well as spinal nerve root compression predispose the peripheral nerves to compressive neuropathies, otherwise

known as "double crush syndrome". [16] Electrodiagnostic testing can assist in this diagnosis.

In the absence of examination findings suggesting upper motor neuron disease, common diagnoses would include cervical radiculopathy and/or peripheral entrapment neuropathy as well as possible brachial plexopathy, although less common. [4, 5, 6, 17, 18] Nerve root lesions at C8-T1 may manifest as weakness and loss of hand dexterity. [4] One may also find weakness in proximal C8-T1 innervated muscles including flexor carpi ulnaris and extensor digitorum proprius. In addition, sensory deficits should follow a C8 and/or T1 dermatomal pattern. Lower brachial plexus lesions can present with weakness and loss of dexterity in the hand as well. A description of all the various plexopathies is beyond the scope of this paper, but it should be borne in mind that Pancoast tumor may present as a lower trunk plexopathy. [19] This should be considered in a patient with findings suspicious for lower trunk plexopathy, particularly those with a history of smoking. Neurogenic thoracic outlet syndrome similarly can appear as a lower trunk plexopathy affecting the hand although again is less commonly seen in clinical practice. [20]

Conclusion:

Isolated NHD is not unusual to see in clinical practice. The most common etiologies of NHD are focal compressive mononeuropathy at the wrist, i.e., carpal tunnel syndrome, followed by compressive mononeuropathy at the elbow, i.e., cubital tunnel syndrome. [21] However, as we have demonstrated in these four cases, patients may present with similar complaints of weakness, numbness, paresthesias or clumsiness of the hand with vastly different etiologies including motor neuron disease, cervical myelopathy, cervical radiculopathy and double crush syndrome. It is clinically important to initially differentiate CNS versus PNS insult by asking appropriate questions during the taking of the history of present illness and performing a thorough neurological examination including checking deep tendon reflexes, pinprick sensory examination, motor examination and evaluating for pathological reflexes and assessing tandem gait. These simple tests will direct the clinician towards the appropriate imaging and ordering of electrodiagnostic testing and formulating a diagnosis and treatment plan.

Acknowledgments:

Dr. Todd Feathers, orthopedic surgeon, Oakland, Maryland, Figure 1.

Samantha Rose Monaghan, graphic designer and illustrator, Figures 2 and 3.

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