Chapter

Working Hand Syndrome: A New Definition of Nonclassified Polyneuropathy Condition

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Abstract

The aim of this chapter was to define an unexplained nonclassified polyneuropathy condition as a new neurological disease. This new diagnosis of occupation-related polyneuropathy has been named as "working hand syndrome (WHS)." This study collected and compared clinical and electrophysiological analyses data from healthy controls, WHS patients, carpal tunnel syndrome (CTS) patients, and polyneuropathy patients. The WHS patients presented to the clinic with pain, numbness, tingling, and burning sensations in their hands that increased significantly during rest and nighttime. However, there was no weakness in the muscles, and the deep tendon reflexes were normal in this disease. The patients had all been working in physically demanding jobs requiring the use of their hands/arms for at least 1 year, but no vibrating tools were used by the patients. All of the cases were men. I suppose that overload caused by an action repeated chronically by the hand/arm may impair the sensory nerves in mentioned hand/arm. In patients with these complaints, for a definitive diagnosis, similar diseases must be excluded. Nonetheless, the specific electrophysiological finding that the sural nerves are normal on the lower sides, as well as the occurrence of sensory axonal polyneuropathy in the sensory nerves without a significant effect on velocity and latency in the work-ups of the upper extremity are enough to make a diagnosis.

Keywords: working hand syndrome (WHS)

1. Inclusion

Polyneuropathies (PNP) are disorders of the peripheral nervous system that indicates any disorder of the peripheral nervous system. Polyneuropathy is one of the most prevalent neurologic conditions. Polyneuropathy has an estimated prevalence of 5–8% in the general population. However, if there are one or more risk factors involved, this rate can increase to 12–17%. Various systemic diseases, exposure to toxicity, drugs, infections, and hereditary diseases are considered causes. Young patients are much more likely to have a polyneuropathy on a genetic basis, elderly patients are much more likely to have idiopathic polyneuropathy, and middle-age patients are more likely to have acquired polyneuropathy. It needs to be done that family history and other important details of the individual's history and examination. Family history should focus on illnesses associated with neuropathy, such as diabetes mellitus, hypothyroidism, renal failure, hepatic disorder,

human immunodeficiency virus infection, and dysproteinemic disorders (10% of peripheral neuropathies are associated with dysproteinemias) and in those receiving chemotherapy and cancer. In the developed world, the most common cause of peripheral neuropathy is diabetes mellitus. Patients with cancer may develop neuropathy depending to nutritional deficiency and chemotherapy side effects. But the etiology of 20–25% of these neuropathies remains uncertain [1–3].

The clinical manifestations of peripheral neuropathy vary widely that weakness, fatigue, hypesthesia, ataxia, autonomic symptoms, and positive symptoms include cramps, twitching, and myokymia. Sensorimotor peripheral neuropathies are the most common form of neuropathy. Usually, there is a progression from distal to proximal. Diminished deep tendon reflexes, distal muscle weakness, and atrophy are common in advanced cases. Most neuropathies are chronic and progressive. Peripheral neuropathy may be symmetrically generalized, multifocal, or focal. Most neuropathies are symmetric and length-dependent. Chronic symmetrical polyneuropathy is the most common type of polyneuropathy and usually evolves over months. Sensory or motor symptoms in a more diffuse, involving both proximal and distal limbs in length-independent pattern. In these cases reflexes are globally reduced or absent. The earliest symptoms of polyneuropathy are usually sensory abnormalities. Sensory symptoms start in the feet, which are supplied by the longest axons. Pathologic mechanisms in peripheral neuropathy are distal axonopathy, myelinopathy, and neuronopathy. The symptoms ascend insidiously up the leg. The upper limb involvement may never occur. Development of symptoms in the hands and feet at the same time is atypical for a length-dependent neuropathy and may indicate coexisting disorder [2, 3].

One of the most common causes of neuropathic pain in the hands is physical compression of the nerves, known as compression neuropathy. Carpal tunnel syndrome (CTS) and cubital tunnel syndrome are examples. Direct injury to a nerve, interruption of its blood supply, or inflammation may also cause neuropathic pain.

Anamnesis, neurological checkup, and electrophysiological work-up are recommended for diagnosis [1–3].

2. Working hand syndrome

Working hand syndrome patients have neuropathic pain in their hands, and axonal neuropathy is detected only in the sensorial neurons of the upper extremity. The common trait for these patients is the fact that they used their hands/arms during heavy labor. I think that a significant number of patients as this should not to be underestimated in the general population. Common traits among the patients include man sex, use of the arms and hands in heavy labor, neuropathic pain in their

hands, and axonal polyneuropathy in the sensory median and ulnar nerves. The average age of the patients is 45.7 ± 20.4 years in working hand syndrome (WHS).

None of the WHS cases have systemic disease, and all of the cases are men. The use of the upper extremity while working a physically demanding job (construction worker, farmer, forester, crushing, tire repairer) requiring the use of the hands/arms for at least 1 year; presentation with pain, numbness, tingling and burning sensations (neuropathic) in the hands and fingers that increases significantly during rest and nighttime in the WHS [1].

3. Etiopathogenesis

Pathology in the sensory nerves can cause neuropathic pain. Sensory polyneuropathy is one of the most common causes of neuropathic pain. It is believed that WHS is likely a sensory neuropathy with such a mechanism as axonal polyneuropathy, because the ulnar nerve is more affected than the median nerve in the upper extremities in polyneuropathies. I posit that an overload caused by an action repeated chronically by the hand/arm may impair the sensory nerves in the said hand/arm. Not only the peripheral nervous system but also the local vessels may be affected. This process may result in vasoconstriction of the local vessels. This situation leads to hypoxia and a lack of nutrition in the sensory nerves. However, there is not a clear relation between WHS and its pathology. However, in my opinion, genetics, ergonomics, emotional stress, and biodynamic status play an important role in WHS, because this disease does not occur in everyone who is doing the same job [1].

4. Diagnosis

WHS is a polyneuropathy and occupational disease. Patients with WHS present with pain, numbness, tingling, and burning sensations in their hands that increases significantly during rest and nighttime. They also use their arms/hands for jobs that require heavy labor. The neurological examinations of patients with WHS are normal. Only the sensory nerves in the upper extremities are affected.

	Carpal tunnel syndrome	**Hand-arm vibration syndrome**	**Chronic ıdiopathic axonal polyneuropathy**	**Working hand syndrome**
Sural nerve	Normal	Normal	Decreased SNAP	Normal
The sensory median nerve distal latency/velocity	Delayed/Decreases	Delayed/Decreases	Normal/Normal	Normal/Normal
SNAP of the sensory median nerve	May be reduced	May be reduced	Reduced	Reduced
The sensory ulnar nerve distal latency/velocity	Normal/Normal	Delayed/Decreases	Normal/Normal	Normal/Normal
SNAP of the sensory ulnar nerve	Normal	May be reduced	Reduced	Reduced
CMAP of lower extremity motor nerves	Normal	Normal	Reduced	Normal

	Carpal tunnel syndrome	**Hand-arm vibration syndrome**	**Chronic ıdiopathic axonal polyneuropathy**	**Working hand syndrome**
The motor median nerve distal latency/velocity	May be delayed/ decreases	May be delayed/ decreases	Normal/Normal	Normal/Normal
CMAP of the motor median nerve	May be reduced	May be reduced	May be reduced	Normal
The motor ulnar nerve distal latency/velocity	Normal/Normal	Delayed/Decreases	Normal/Normal	Normal/Normal
CMAP of the motor ulnar nerve	Normal	May be reduced	May be reduced	Normal

SNAP, sensory nerve action potential; CMAP, compound muscle action potential.

Table 1.
Electrophysiological findings of working hand syndrome and similar diseases.

	Carpal tunnel syndrome	**Hand-arm vibration syndrome**	**Chronic ıdiopathic axonal polyneuropathy**	**Working hand syndrome**
Age	Intermediate and advanced ages	Young or middle ages	Intermediate and advanced ages	Young, middle or advanced ages
Gender	Female are generally affected	No significant gender differences	No significant gender differences	All male
Complaint	Neuropathic pain is often in the hands	Neuropathic pain is often in the hands	Especially neuropathic pain in the feet	Neuropathic pain is the hands
Risk factors	For example, rheumatism, pregnancy, diabetes, etc.	Continuous use of vibrating hand-held machinery	For example, diabetes, various cardiovascular risk factors, the metabolic syndrome, etc.	Patients used their hands/arms in heavy labor (no use of vibrating hand-held machinery)
Raynaud's phenomenon	No	Yes	No	No
Affected tissues	Only the median nerve	Median and ulnar nerves (motor and sensory nerves), blood vessels, nerves, muscles, and joints	Especially sural nerve and other sensory and motor nerves	Only the median and ulnar sensory nerves
Deep tendon reflexes	Unaffected	Usually unaffected	Usually decreases	Unaffected
Muscle weakness and atrophy	Advanced cases	Advanced cases	Advanced cases	No

Table 2.
Differential diagnosis of working hand syndrome.

For a definitive diagnosis:

1. All have been working in physically demanding jobs requiring the use of the hands/arms.

2. Patients exhibit neuropathic pain in their hands.

3. The exclusion of similar diseases (**Tables 1** and **2**).

4. Specific electrophysiological findings that the sural nerves are normal, as well as the occurrence of sensory axonal polyneuropathy in the sensory nerves without being greatly affected by speed and latency in the work-ups of the upper extremity, are enough to make a diagnosis [1].

5. Nerve conduction studies

The electromyographer plays an important role in the evaluation of patients with polyneuropathy. The results of nerve conduction studies and electromyography are useful in analyzing the underlying pathophysiology. The recording and measurement of the terminal latency, amplitude, duration of the evoked potential, and the conduction velocity. Nerve conduction studies are also valuable in differentiating whether a demyelinating process is acquired or inherited. Nerve conduction studies can identify the predominant pathophysiology (axonal loss or segmental demyelination) and establish whether sensory or motor findings predominate. In addition, the studies provide quantitating the severity and the distribution of the neuropathy. Electrophysiological work-ups show axonal damage (axonal neuropathy), demyelination (demyelinating neuropathy), and both (mix neuropathy). In the electrophysiological work-ups that involve distal latency, the amplitude, shape, and velocity of the motor and sensory nerves are checked. Axonal degeneration causes a decrease in amplitude, while demyelinating polyneuropathy causes delays in distal latencies and decreases in velocity. Acute axonal damage in the motor nerves can cause spontaneous activities in muscle fibers when checked with electromyography, where dilution in voluntary activity and chronic neurogenic motor unit potentials (MUP) are seen [1, 4].

The electrophysiological work-ups in the WHS are completed with standardized supramaximal percutaneous stimulation techniques. In the upper sides, a sensorial check-up is completed of the median and ulnar nerves. The sural nerves are used for a lower extremity sensory evaluation. For the median motor nerve evaluation, a 6–7 cm proximal of the abductor pollicis brevis muscle is supramaximally stimulated; the ulnar motor nerve is recorded from the abductor minimi muscle; the median sensorial nerve is recorded from the second finger; and the ulnar sensorial nerve is recorded from the fifth finger. For the sural nerves, the active electrode was placed between the lateral malleolus and the heel, and the reference electrode was placed 30 mm distally at the lateral edge of the foot. Supramaximal stimuli are applied at 13 cm proximal to the active electrode, just lateral to the midline of the calf. Amplitudes below 16 uV for the sensorial nerves in the upper sides and amplitudes below 10 uV for the sensorial nerves in the lower sensory sides (sural nerves) are considered the limits of sensory axonal neuropathy to assess its sensitivity and specificity. The use of an infrared lamp ensured that the temperature of the extremities during measurement has been done at 34°C or higher. In the electrophysiological findings of the WHS according to the normal, the distal latency and velocity of the median and ulnar sensorial nerves are similar in both hands. However, both the median sensory and ulnar sensory nerve amplitudes are decreased ($P < 0.05$). The motor nerve conduction work-ups of the upper and lower sides are similar in all differential diagnosis. The sural nerve results are similar on the lower sides in the normal, CTS, and WHS. The sural nerve results are significantly affected in the polyneuropathy ($P < 0.05$).

6. Clinical results

The deep tendon reflex polyneuropathy patients have a significantly decreased reflex when compared with all differential diagnosis ($P < 0.05$, Duncan). Regarding

the presence of atrophy when all cases are compared with the WHS, there is no significant difference. In terms of hand complaints, polyneuropathy has a higher complaint score (1.3 ± 1.33; $P < 0.05$) when compared with the healthy normal group. However, the WHS (3.00 ± 0.00) and CTS (3.00 ± 0.00) groups exhibit an increase in hand complaints when compared with both the healthy and polyneuropathy.

7. Other comorbid diseases in the WHS

In terms of diabetes mellitus, hypertension, cardiovascular diseases, hyperlipidemia, cigarette smoking, and the presence of atrophy, when all cases are compared with the WHS, there is no significant difference according to Fisher exact test.

8. Differential diagnosis in the WHS

The use of a vibrating tool by the patients and the presence of a nervous system disease, such as polyneuropathy, CTS, or hand-arm vibration syndrome (HAVS). The diagnosis of distal axonal sensory polyneuropathy is extracted from nerve conduction work-up reports based on the presence of bilateral, symmetric, and distal lower and upper extremity neuropathic pain. The motor nerves are unaffected, and there is no muscle weakness in this condition. Only the hands experience neuropathic pain in the WHS, while there is neuropathic pain in both the feet and hands in the polyneuropathy. Sensory nerve conduction work-ups of the median, ulnar, and sural nerves are widely used in the electrodiagnosis of sensory polyneuropathy. The long nerves are most commonly affected by polyneuropathy. Thus, the sural sensory nerve action potential (SNAP) amplitude is likely the most useful parameter for differentiating normal subjects from those with distal sensory polyneuropathy. Even the sural SNAP is most sensitive in the diagnosis of early distal sensory polyneuropathy. The sural nerve results are significantly affected in the polyneuropathy, while the WHS have normal sural nerve conduction work-ups.

Several diseases affect the nerves of the hand, the most common being CTS, which is caused by median nerves in the carpal tunnels becoming stuck. It is characterized by neuropathic complaints in the first four fingers and the palm of the hand. Its symptoms manifest usually during rest hours or nighttime, and the cases identified in the WHS are similar in that regard. This means the entirety of their hand and the fingers have neuropathic pain. Women are more commonly affected by CTS, and rheumatism, pregnancy, and diabetes are among the known risk factors for CTS. All of the WHS cases are men, and they have no known CTS risk factors. Characteristic electrophysiological findings of CTS include a progressively delayed sensory peak latency, and amplitude becomes smaller in the median nerve. In medium cases, similar findings appear in the motor nerves. In advanced cases, SNAP and compound muscle action potential (CMAP) values decrease, which means that in CTS, a delayed distal latency and decrease in velocity are pronounced in the median nerve. The ulnar nerve conduction work-ups in CTS are normal. In the WHS, according to the normal, distal latency and velocity are close to normal, but both the median sensory and ulnar sensory nerve amplitudes are decreased. Motor values are completely normal.

Guyon canal and cubital tunnel entrapment neuropathies can cause neuropathic pain, as well [8], but neuropathic pain is seen only in the ulnar nerve tract. In nerve conduction studies, distal latency and velocity are affected in the ulnar nerve. In all of the cases herein, neuropathic pain is identified in every region of the hand. Not only the ulnar nerve but also the median nerve is affected.

The mechanical energy created by vibrating tools, which enters the body through the fingers or palms, is called hand-arm vibration. These tools are generally used in the production, stone working, mining, construction, agriculture, and forestry sectors. HAVS is a clinical condition that occurs after exposure to hand-arm vibration. Symptoms of HAVS include numbness, pain, and reduced dexterity, strength, and sensation in the hands. In HAVS, the peripheral and central nervous systems are affected, which can lead to vascular, bone and joint, and tendon and muscle diseases. There is a direct correlation between the disease and the magnitude and duration of hand-arm vibration and cold temperatures. In the cases here in, no vibrating tools are used by the patients, but they engage in taxing labor using their hands (using such tools as a sledgehammer, hammer, saw, and carry stones). It is argued that the usage of beta-blockers and cigarettes and a decrease in blood circulation due to exposure to the cold lead to an increase in HAVS symptoms. According to the anamnesis of the patients in the WHS, their symptoms do not change in cold temperatures or after smoking. CTS is often observed in people with HAVS who engage in breaking stones, plating, and forestry. This means that HAVS itself can cause CTS. Electrophysiological studies aimed at defining the nature of a vibration injury have provided conflicting results. Usually, electrophysiological findings related to HAVS are similar to those related to CTS, and the effect on velocity is pronounced. These conditions can be seen together often.

The ulnar nerve is rarely affected in HAVS, but both the ulnar and median nerves are affected in the WHS. Especially, the ulnar nerve is affected. In HAVS, slowed sensory nerve conduction velocities are often observed in the hands. In the WHS, especially, the amplitude is low without being greatly affected by speed and latency. In vibration-associated neuropathies, conceivable target structures could be peripheral sensory receptors, large or thin myelinated nerve fibers, and small-caliber, nonmyelinated C fibers. Pathological studies by cutaneous biopsy have demonstrated demyelinating neuropathy in the digital nerves of individuals with HAVS [5–8].

9. Treatment options

In treatment of polyneuropathy, the primary goal in the evaluation of neuropathy is to identify the etiology and if possible treat the underlying cause. Medical causes such as diabetes mellitus, renal insufficiency, hypothyroidism, vitamin B-12 deficiency, and Guillain-Barré syndrome need specific treatments. But, there is no specific treatment for many chronic neuropathies such as chronic idiopathic axonal polyneuropathy or the hereditary neuropathies. One of the most limiting symptoms is neuropathic pain. The neuropathic pain can be effectively treated with an algorithmic approach. In the WHS, there is no specific treatment yet. However, I gave 75 mg pregabalin.

10. Conclusion and future directions

The WHS is a new disorder. It is also an occupational disease. I think that a significant number of patients as this should not to be underestimated in the general population. We only considered it previously as a sensory polyneuropathy in upper limbs. For this reason, we need to examine it more in detail from the etiopathogenesis to its treatment. This disorder is suggested to serve as a resource for patients, healthcare professionals, and members of the neurology community at large.

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