Study about Natural Supplements used Peripheral Neuropathy

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Introduction

Peripheral neuropathy occurs when damage to the peripheral nervous system, which are the nerves located outside the spinal cord and brain. These nerves are responsible for carrying signals to the rest of the body, including the contraction of the body's muscles, urination, the blood vessels, the bones, the heart, digestion, sexual function, and the immune system. Symptoms can range from mild to severe and can develop over a long period of time. Signs of peripheral neuropathy include sharp or jabbing pain, gradual numbness, lack of coordination, extreme sensitivity to touch, muscle weakness, and in severe cases, paralysis. This neuropathy is brought on by several different conditions such as diabetes, tumors, infections, autoimmune diseases, inherited disorders, bone marrow disorders, certain cancers, and other diseases. People with a family history of neuropathy, alcohol misuse, diabetes, infections, kidney, liver, or thyroid disorders, and vitamin deficiencies are more likely to develop this neuropathy. Several studies have shown that obesity also elevates the risk of an individual developing this disease (Callaghan et al., 2018). This neuropathy is prevalent in old individuals, thus significantly reducing the quality of life (Callaghan et al., 2018).

Diagnosis

The diagnosis of this neuropathy necessitates a thorough medical examination to determine the different etiologies (Lehmann et al., 2020). Treating peripheral neuropathy is not always straightforward. Patients are referred for evaluation of peripheral neuropathy and have cervical myelopathy or bilateral lumbosacral radiculopathy (Siao & Kaku, 2019). Sometimes patients with multiple sclerosis come to be referred for evaluation of peripheral neuropathy since they present with numbness in the feet and gait imbalance (Siao & Kaku, 2019). One of the outstanding presentations of this neuropathy is symmetrical numbness of the feet with or without numbness of the hand (Siao & Kaku, 2019).

Clinical patterns

The first step in diagnosis is recognizing specific patterns that a patient with this neuropathy presents. These patterns are determined mainly by the signs and symptoms a patient with this disease portrays. Lehmann et al. (2020) proposed five clinical practices that could help doctors diagnose this neuropathy. The first pattern involves a slow progressive, distal symmetric, and primarily sensory neuropathy (Lehmann et al., 2020). This presentation is seen mainly in diabetes, chemotherapy, and alcoholic neuropathy patients. This patient needs limited evaluation when determining the diagnosis. The second pattern is slow and long-standing neuropathy, characterized by foot and muscle wasting (Lehmann et al., 2020). Patients with this neuropathy are limited, and diagnosis used to be focused...
on genetic testing (Lehmann et al., 2020). The third pattern is the onset of neuropathy with or without proximal involvement. Patients with this clinical presentation suggest that the neuropathy was acquired from an immune-mediated condition (Lehmann et al., 2020). Physicians here need to focus more on antibody testing. The fourth pattern involves rapid disease progression, neurological pain, multifocal symptoms, and autonomic dysfunction (Lehmann et al., 2020). This possible neuropathy with this presentation is amyloidosis, vasculitis, or paraneoplastic (Lehmann et al., 2020). Diagnosis of these three neuropathies required a patient to provide a detailed clinical history. The final pattern is sensory ataxic, which is related chiefly to sensory neuronopathy (Lehmann et al., 2020). Patients with this neuropathy present with a sense of vibration and proprioception (Lehmann et al., 2020).

Some patients with this neuropathy also portray some muscle strength. The diagnosis of this neuropathy involves the evaluation of mitochondrial and autoimmune disorders.

**Clinical History**

The diagnosis of this disease begins with a detailed history that includes family, occupation, and a list of medical uses (Siao & Kaku, 2019). Family history includes ethnic background, parent and sibling details such as the presence of possible inherited neuropathies, and consanguinity (Siao & Kaku, 2019). Social history consists of the residential area where the patient was currently residing, sexual history, alcohol and drug use, sexual orientation, and disease history (Siao & Kaku, 2019). The medical examination starts with a detailed history of symptoms, occupational and family history, and physical and neurological exam. Different patterns presented by this condition help build differential diagnoses based on how they present themselves. This presentation helps identify the etiology and helps the doctors know which treatment plan they will use to treat this neuropathy. After a detailed history has been recorded, the next vital step in evaluation is conducting a quantitative test.

**Tandem Gait Testing**

Tandem gait is one of the physicians' examinations (Margolesky & Singer, 2017). This test is widely used to screen patients with vestibular and neurological disorders (Cohen et al., 2017). Ordinary healthy people without neurological disorders; however, this performance decreases with time (Cohen et al., 2017). Individuals with neurological diseases cannot perform this test while their eyes are closed (Cohen et al., 2017). However, the test lacks a standard guide that could help researchers determine the sensitivity of abnormal tandem gait evaluation for peripheral neuropathies and neurodegenerative diseases (Margolesky & Singer, 2017).

**Nerve Biopsy**

Doctors perform another neurological test to determine if the patient has peripheral neuropathy: a nerve biopsy, M.R.I., and spinal tap. Nerve biopsy is one of the most recommended diagnoses for this neuropathy (Luigetti et al., 2019). Many neuropathies in the past produced peculiar lessons that were easily identified in the biopsy (Luigetti et al., 2019). Nerve biopsy helps diagnose dysimmune and vasculitic neuropathies (Luigetti et al., 2019). However, it is not effective in detecting inherited neuropathies. Genetic testing is highly recommended in diagnosing patients with peripheral neuropathies that result from inheritance (Lehmann et al., 2020). Current genetic testing includes NGS, which detects D.N.A. variants associated with peripheral neuropathy (Bacquet et al., 2022).

**Neurological Examination**

Evaluating the degree of nerve damage is essential in determining a particular clinical pattern (Lehmann et al., 2020). The doctor stimulates a nerve to assess if it will produce action potential (Rudin, 2019). The doctor will record the time from when a nerve stimulation occurred to when the action will be made and the magnitude produced (Rudin, 2019). These measures predict the extent to which this neuropathy has damaged the nerve. The two common neurological examinations are E.M.G. and N.C.S. An electromyogram (E.M.G.) is considered the standard for diagnosing this neuropathy (Jia et al., 2019). E.M.G. and N.C.S. confirm that the patient suffers from peripheral neuropathy, not other mimics like distal myopathy (Lehmann et al., 2020). They are also used to assess nerve damage’s primary mechanism, such as demyelinating or axonal (Lehmann et al., 2020). These two tests are also used to determine which nerves are unaffected.
Peripheral Nerve Imaging

Ultrasound can be used to diagnose peripheral neuropathies. Ultrasound is used for patients suspected that they have an immune-mediated neuropathy. This neuropathy is primarily characterized by increased nerve cross-section, which can be imaged by an ultrasound (Lehmann et al., 2020). With nerve ultrasound, M.R.I. can view nerve segments that electromyography cannot access (Telleman et al., 2021). Nerve imaging helps to determine which nerves will be a target for biopsy examination (Lehmann et al., 2020).

Blood Tests

These laboratory tests are usually done to check if the patient has insufficient vitamins, toxic elements, or evidence of immune reaction. Blood tests are specific to the type of peripheral neuropathy; hence the doctor will first evaluate the clinical presentation of the neuropathy before ordering a blood test. Standard blood tests include folate and Vit. B12 levels, Hepatitis B and C, Lyme disease, HIV/AIDS, and antibody reaction tests (Lehmann et al., 2020).

Laboratory tests also include renal function, HbA1c, glucose level, liver function, and thyroid tests (Lehmann et al., 2020). Research has shown that deficiency of vitamin B12 can cause this neuropathy (Mallet et al., 2020). The shortage of this vitamin has also been linked with diabetes, one of the risk factors that elevate a person’s risk of acquiring peripheral neuropathy (Mallet et al., 2020).

Case Study I

*Patient:* Male.

*Age:* 61-year-old.

*History:* A 61-year-old male, currently a union boilermaker and a steelworker since the late 70s, with pre-diabetes, hypertensive, peripheral neuropathic nerve pain, degenerative disc disease, and disc herniation. Experienced long-term exposure to environmental toxins including arsenic, lead, tungsten, antimony, thallium, cadmium, cesium, nickel, niobium, rubidium, titanium, and other heavy metals (working in a boilermaker, refinery, and steel mill). His symptoms included brain fog, sciatic pain, lumbar spine pain, and inability to sleep due to pain, and sitting and standing for long periods would cause pain and lower his quality of life. His medications consisted of Lotensin, Hydrochlorothiazide, Fenofibrate, and Vitamin D.

*Treatment/Method:* He started on ten drops of Proprietary blend 1 B.I.D.

**Legend**

*Proprietary blend 1:* silica, vitamin C, and trace minerals.

*Proprietary blend 2:* N-acetyl L-tyrosine, anhydrous caffeine, L-theanine, velvet bean seed, pine bark, curcumin, and vitamin D.

*Proprietary blend 3:* black seed oil, resveratrol, turmeric, raspberry ketone, apple cider vinegar, aloe Vera, and d-ribose

*Results:* After administering Proprietary blend 1, he noticed significant improvement in mental clarity, 90% improvement in neuropathic foot pain, and experienced improved quality of sleep.

Discussion

**Patient Diagnosis**

The patient suffers from a toxic neuropathy caused by exposure to harmful chemical substances like lead, arsenic, tungsten, antimony, thallium, cadmium, cesium, nickel niobium, rubidium, titanium, and other heavy metals.
Working in the boilermaker, refinery, and steel mill leads to chronic exposure to these toxic chemicals. Chronic and acute exposure results in nervous system disorders, immune system disorders, cancer, skin lesions, birth malformations, vascular damage, and kidney and G.I. dysfunction (Eskut & Koskderelioglu, 2021). Heavy metals result in neurotoxicity using oxidative stress, dysregulation of cell signaling, cell membrane disruption, and impairment of neurotransmission (Eskut & Koskderelioglu, 2021).

**Heavy mental and Neurotoxicity**

Lead toxicity is one of the significant causes of polyneuropathy characterized by wrist drops. The arsenic neurotoxic mechanism has not been proved, but researchers suggest it occurs through glutathione conjugation and oxidative methylation (Eskut & Koskderelioglu, 2021). Arsenic causes polyneuropathy, which does not cause multi organ involvement (Valappil & Mammen, 2019). Acute thallium poisoning results in seizures, mental disturbance, polyneuropathy, and memory impairment (Wang et al., 2021).

**Prevention and Treatment for Peripheral Neuropathy**

Different strategies have been proposed to prevent and treat peripheral neuropathies. There is no specific treatment for peripheral neuropathy as other underlying conditions in the body cause it. To manage it, treating the underlying conditions and their symptoms is the most used treatment plan to improve its symptoms. These underlying conditions are assumed to be the key triggers of peripheral neuropathy; hence keeping them under check improves the conditions and speeds recovery.

**Nutritional Treatment of Peripheral Neuropathy**

Nutrition regimens have shown evidence that they can treat peripheral neuropathy. There are proprietary nutritional blends used to treat and enhance symptoms of this condition: Silica, vitamin C, trace minerals, black seed oil, turmeric, L-tyrosine, anhydrous caffeine, and other nutrients. The patient was administered a proprietary blend composed of silica, vitamin c, and trace elements in this case study. Vitamin C Edobor’s (2021) research on Wistar rats with diabetic peripheral neuropathy showed that administration of vitamin C reduces pain in them. Vitamin is a dietary supplement and an essential micronutrient vital for biological processes. Vitamin C has antioxidant activities that help overcome oxidative stress caused by reactive oxygen species (ROS) (Edobor, 2021). Most heavy metals damage the cell through oxidative stress. However, the administration of vitamin C helps cells counter oxidation stress through antioxidation. This treatment helps in managing pain in a patient with neuropathies. Pain is one of the significant symptoms of peripheral neuropathy that decreases the quality of life (Abdelrahman & Hackshaw, 2021). Evidence has shown that pharmacological treatment is ineffective in managing patients that result from peripheral neuropathy (Abdelrahman & Hackshaw, 2021). Huff et al.’s (2020) study shows that vitamin C is vital for myelinating Shawn cells in the (PNS) peripheral nervous system. This myelination results in the PNS nerves’ saltatory conduction of electrical activities (Huff et al., 2020). Shawn cells also play a vital role in nerve regeneration after an injury. Huff et al. (2020) study used the mouse to illustrate how maternal vitamin C results in hypomyelination in the early development of infant mice. Vitamin C regulates periaxin and M.B.P. (Huff et al., 2020).

**Zinc Supplements**

Zinc is an essential micronutrient that has been used to treat a variety of sickle cell, neuropathic pain, muscle degeneration, and alcoholic liver disease (Abdelrahman & Hackshaw, 2021). Zinc deficiencies have been linked with immune suppression, apoptosis, and D.N.A. damage. The deficiency of trace elements like zinc also elevates the risk of an individual acquiring diabetes and diabetic conditions like diabetic neuropathy (Liu et al., 2014). The mechanism in which zinc is used to treat neuropathic pain since it plays a vital role in regulating oxidative stress. Zinc stimulates metallothionein synthesis by acting as a gene and expression protein (Liu et al., 2014). Mouse research has shown that zinc increases the mRNA transcript of proteins translated to produce metallothionein. Metallothionein is a powerful antioxidant that reduces oxidative stress that heavy metals have caused. When administered zinc supplements, rats with induced diabetic neuropathy showed reduced pain (Abdelrahman & Hackshaw, 2021). Metallothionein is also an anti-inflammatory agent which reduces neuropathy pain by reducing the pressure on the nerve endings (Abdelrahman & Hackshaw, 2021).
**Vitamin B**

This vitamin is one of the essential vitamins the body requires for biological processes. Vitamin B6, B1, B12, and B9 play a vital role in biological processes like immunity, R.N.A., D.N.A. synthesis, and metabolism (Abdelrahman & Hackshaw, 2021). Patients that have deficiencies in vitamin B12 have been shown to exhibit signs of lower and upper neuron dysfunction (Staff & Windebank, 2014). These patients also exhibit cognitive dysfunction (Staff & Windebank, 2014). Vitamin B6 has been shown to increase nerve conductance in rats with diabetic neuropathy (Abdelrahman & Hackshaw, 2021). Research has shown that vitamin B12 reduces neuropathy pain (Abdelrahman & Hackshaw, 2021).

**Vitamin D**

No research has provided a precise mechanism in which vitamin D reduces pain in neuropathies. Neuropathy pain has been treated with vitamin D in patients with type II diabetes (Abdelrahman & Hackshaw, 2021). One study proposes that vitamin D reduces pain in neuropathies by regulating cytokines (Abdelrahman & Hackshaw, 2021). Cytokines are a potent inflammatory mediator which results in acute or chronic inflammation. Inflammation increases the pain in neuropathies since it increases pressure on nerve endings. Vitamin D regulating cytokines will result in a reduction of inflammation.

**Aluminosilicate**

This regimen is used to treat or prevent neuropathies that result from toxic agents. Aluminosilicate is an adjuvant for zeolites which is a vital element due to its detoxification properties. Zeolite properties help the body detoxify harmful components like heavy metals consumed from the environment. Detoxification reduces pain in the peripheral nerve (Justia Patent, 2012). Silica reduces the pain in patients with neuropathies by removing heavy metals from cells. This removal results in a reduction of oxidative stress hence relieving the pain.

**Conclusion**

Peripheral neuropathies occur when there is damage to the nerves of the PNS. Damage to these nerves reduces dysfunction in motor and sensory functions. Individuals suffering from peripheral neuropathy tend to experience mild to severe symptoms, which continue to be severe as time goes by. Some of the symptoms of this condition include lack of coordination, paralysis, gradual numbness, and sharp pain. Several etiologies have been identified to be the cause of this neuropathy. These etiologies include diabetes, toxins, infections, autoimmune disease, and specific cancer. Individuals with these conditions tend to portray an elevated risk of acquiring peripheral neuropathy. Diagnosis of this neuropathy is not straightforward, and the doctor needs to follow standard protocols to determine if the neuropathy affects the patient. The doctor must conduct a detailed clinical history and perform quantitative tests to confirm and resolve the severity of the condition. Current treatment strategies focus on treating the etiology after the doctor. Antidepressants, anesthetics, and narcotics are some of the recommended pharmacological therapies for treating neuropathies. The medicines are also used to relieve the symptoms of peripheral neuropathy. Research has been increasing to find other more effective methods than pharmacological therapies. Nutritional supplements, including vitamins, herbal products, and trace elements, effectively manage pain in these neuropathies (Abdelrahman & Hackshaw, 2021). Extensive research on mouse models has illustrated their efficacy and mechanism in pain management. There is a significant need to translate these researches into a clinical setting to determine their efficiency in human beings and how they will help patients manage pain due to peripheral neuropathy.

**References**