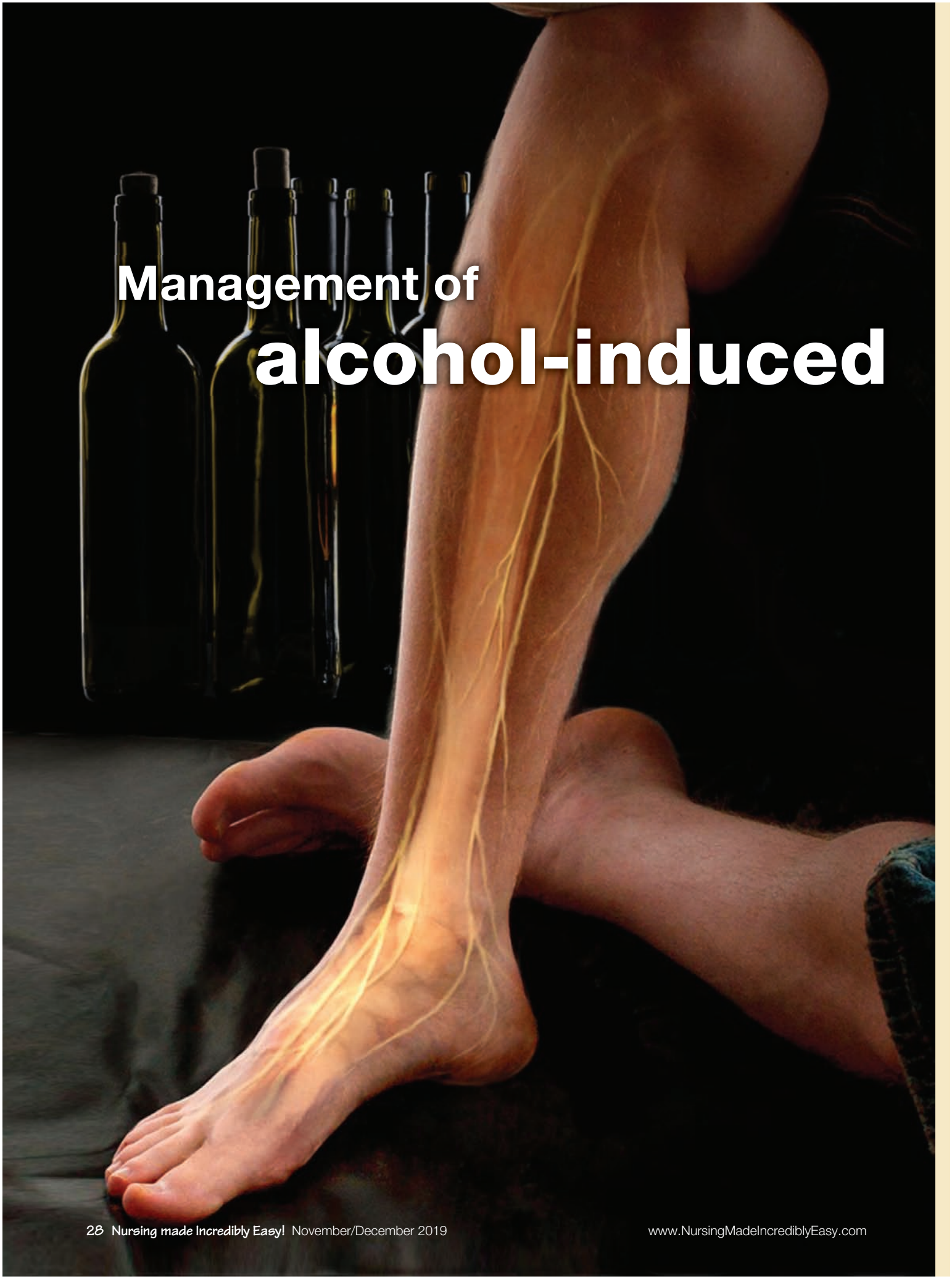

Management of alcohol-induced

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peripheral neuropathy

This condition can impact quality of life for your patients. Learn how to recognize the signs and symptoms so you can connect patients with the resources they need.

By Richard L. Pullen, Jr., EdD, MSN, RN, CMSRN, CNE-cl, and Gerardo A. Ruiz, BSN, RN, CCRN

Alcohol-induced peripheral neuropathy (PN) is a chronic and painful condition in which the neurotoxic effects of alcohol and nutritional deficiencies cause a pathologic response in nerve function. This article presents the pathophysiology, signs and symptoms, diagnostic approaches, treatment options, and nursing care of patients with alcohol-induced PN.

Impact and prevalence

PN is damage to one or more peripheral nerves, leading to sensory, motor, and autonomic dysfunction. The term polyneuropathy is used when multiple peripheral nerves are damaged. The prevalence of PN ranges from 2.4% to 8% per 100,000 individuals worldwide. The Foundation for Peripheral Neuropathy and the US Food and Drug Administration estimate that 20 million people in the US experience PN. Neuropathic syndromes may develop from diabetes mellitus, vasculitis secondary to immune-mediated diseases, genetic disorders, malignancies, infections, vitamin deficiencies, medications, toxins, trauma, compression, and chronic heavy alcohol consumption.

The prevalence of alcohol-induced PN varies depending on demographics and the diagnostic criteria used in individual research studies. The precise number of people with alcohol-induced PN isn't known. According to studies by the CDC, nearly 30% of adults in the US consume alcohol excessively. Excessive or heavy alcohol consumption is defined by the CDC when men have 15 or more drinks each week and women have 8 or more drinks each week. The National Institute on Alcohol Abuse and Alcoholism reports that 16 million people in the US have been diagnosed with alcohol use disorder (AUD). A person is diagnosed with AUD when he or she meets at least 2 of the 11 criteria in the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders,* Fifth Edition. AUD is generally synonymous with the term *alcoholism*. Multiple research studies indicate that 11% to 66% of people with AUD have alcohol-induced PN.

The clinical presentation of alcohol-induced PN is similar to other etiologies of PN. However, the pathophysiology of alcohol-induced PN is different than other forms of PN. Multiple variables contribute to this painful neuropathic syndrome, including the toxic effects of alcohol on neurons and nutritional deficiencies. Alcohol-induced PN can be incapacitating and debilitating, and the patient must also cope with the impact

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that chronic heavy alcohol consumption has on physical and mental health and personal, social, and professional relationships.

Systemic effects of heavy alcohol consumption

The morbidity and mortality of chronic heavy alcohol consumption encompass a wide range of systemic diseases with negative health outcomes that may be interrelated with a patient's neuropathic syndrome (see *Selected systemic effects of heavy alcohol consumption*). Clinical correlation of signs and symptoms and close patient surveillance are essential. The peripheral nervous system plays a major role in the development of many of the signs and symptoms of alcohol-induced PN.

Selected systemic effects of heavy alcohol consumption

Neurologic

- Wernicke encephalopathy results from thiamine deficiency, causing confusion, eye muscle weakness, and ataxia
- Korsakoff syndrome, a neuro-psychiatric manifestation of Wernicke encephalopathy, causes deficits in memory and cognitive skills
- Ventricular enlargement causes cognitive dysfunction
- Cerebellar degeneration from nutrition deficiencies and neurotoxicity of alcohol causes gait dysfunction
- Marchiafava-Bignami disease, a degeneration of nerve fibers in the corpus callosum from nutritional deficiencies and toxic effects of alcohol on nerves, causes personality changes, seizures, dementia, and coma
- PN from thiamine deficiency and toxic effects of alcohol on nerves causes numbness, burning pain, muscle cramps and weakness, decreased sensations, and loss of deep tendon reflexes
- Skeletal muscle myopathy causes muscle cramps, soreness, and weakness
- Alcohol withdrawal syndrome related to metabolic and nutritional insufficiencies and reduction or abstinence from alcohol causes anxiety; agitation; headache; sweats; gaps in memory; tremor; and tactile, auditory, and visual disturbances

Cardiovascular

- Cardiomyopathy characterized by cardiomegaly and ventricular hypertrophy related to toxic effects of alcohol on the cardiac muscle fibers causes a decrease in cardiac output and heart failure
- Hypertension results from an increase in sympathetic nervous system activity related to the toxic effects of alcohol
- Atrial and ventricular dysrhythmias occur because of the toxic effects of alcohol in the cardiac conduction system and may coexist with cardiomyopathy
- Reduced platelets and increased clotting factors due to heavy alcohol consumption predispose the patient to bleeding
- Changes in the structure and number of red and white blood cells predispose the patient to anemia and infection
- The interplay of coronary artery disease, hypertension, cardiac rhythm disturbances, cardiomyopathy, and heart failure may lead to stroke

Pulmonary

- Reduced amount of the antioxidant glutathione predisposes the patient to lung disease
- Increased susceptibility to infection such as pneumonia
- Obtunded, stuporous, or comatose state, causing aspiration

GI and hepatobiliary

- Alcoholic fatty liver disease may lead to cirrhosis of the liver
- Alcohol-induced hepatitis causes extensive liver inflammation, leading to fibrosis and cirrhosis
- Cirrhosis of the liver leads to portal hypertension and end-stage liver disease
- Pancreatitis from extensive inflammation of the acinar cells in the pancreas
- Peptic ulcers from the toxic effects of alcohol in the mucosal lining of the stomach and duodenum; a precursor of peptic ulcer disease may be chronic gastritis (Most peptic ulcers are caused by *Helicobacter pylori* infection.)
- Oral and esophageal cancer may occur secondary to the toxic effects of alcohol on oral and esophageal mucosa and alcohol breakdown to acetaldehyde, a known carcinogen

Musculoskeletal

- Decreased bone mass and density from poor absorption of vitamin D
- Low testosterone levels may occur secondary to heavy alcohol consumption, interfering with calcium absorption
- Decreased muscle mass from the toxic effects of alcohol on mitochondrial cells
- Gait instability, muscle weakness, and osteoporosis predispose the patient to falls and fractures

Endocrine

- Fluctuations in blood glucose levels
- Disrupted absorption of calcium, leading to osteoporosis
- Men: Decreased testosterone, sexual desire, and performance and secondary sexual characteristics, including body hair, shrinking of testes, gynecomastia, and shifting of body fat from the abdomen to the hips
- Women: Decreased testosterone, sexual desire, and performance and infertility, menstrual irregularities, and miscarriage

The peripheral nervous system is comprised of axons—clusters of nerve fibers within a neuron that transmit electrical impulses to and from the central nervous system. Dendrites carry the electrical impulses along the axon through the synapse, allowing neurons to communicate with each other. Axons are encased by myelin—the fatty connective tissue that accelerates the rate of electrical impulses through neurons. Neuron injury may occur when the axon is directly damaged, there are conduction disturbances through the nerve, or there's demyelination of nerve cells. The axon is the primary focus of pathology in alcohol-induced PN. The neurotoxic effects of alcohol or one of its metabolites and vitamin deficiencies cause axonal injury by decreasing nerve fiber density, leading to painful PN and motor and autonomic dysfunction.

The neurotoxic effects of alcohol cause damage to the axon through demyelination of sensory and motor fibers, primarily from the presence of acetaldehyde. Alcohol is predominantly metabolized by the enzyme alcohol dehydrogenase (ADH) in the stomach, intestines, and liver. ADH is further metabolized to the carcinogen acetaldehyde, which is highly toxic to bodily tissues, including the brain, neurons, heart, liver, pancreas, and gastrointestinal (GI) tract. An accumulation of acetaldehyde in the serum causes direct damage to the axon in alcohol-induced PN. Additionally, chronic heavy alcohol consumption is toxic to the metabotropic glutamate receptor 5, which activates neuronal protein kinase C and causes neuropathic pain.

Vitamin B1 (thiamine) is responsible for converting carbohydrates into energy and proper functioning of the brain, neurons, and muscles. It must be obtained from nutritional sources because it isn't normally produced in the body. A vitamin B1 deficiency from poor intake or absorption or hepatic dysfunction from chronic heavy alcohol consumption interferes with the synthesis of many enzymes, including pyruvate dehydrogenase (PDH), which is found in the mitochondria and plays an important role in carbohydrate metabolism. PDH also facilitates the production of the neurotransmitter acetylcholine for the synthesis of myelin, proper axon structure and function, and electrical conduction through the neuron. Chronic vitamin B1 deficiency is associated with bilateral sensorimotor polyneuropathy, beginning in the lower extremities. Neuropathy precedes the onset of systemic and cognitive changes associated with vitamin B1 deficiency.

A major treatment goal is for the patient to be involved in support groups or counseling that will help him or her reduce or abstain from alcohol use.

Patients may also have a deficiency in vitamin B12 (cobalamin), affecting the axon and causing muscle weakness, sensory disturbances, and anemia. Vitamin B9 (folic acid) levels tend to be decreased, reducing the density of small and large nerve fibers. Important in carbohydrate metabolism and neuron function, vitamin B3 (niacin) may also be decreased.

Sensory, motor, and autonomic symptoms

Generally symmetrical, peripheral nerve damage may be focal, multifocal, or diffuse. A variety of sensory, motor, and autonomic symptoms develop over months to years and worsen with time. These symptoms may not respond favorably to treatment, especially if the patient doesn't reduce or abstain from alcohol consumption. The capacity of peripheral nerves to regenerate is limited.

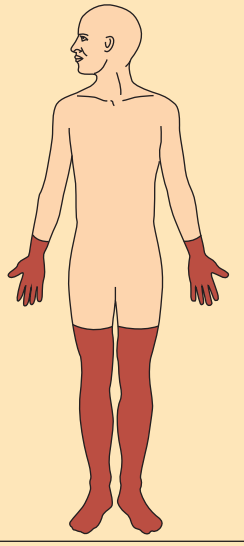
Patients experience dysesthesias, which are abnormal sensations that may be difficult for them to describe. Pain is usually present and characterized by burning, sharp, prickling, tingling, tickling, and pins-and-needles sensations, and may coexist with numbness. Sensory involvement evolves from distal to proximal, initially involving the toes, feet, and legs, followed by the fingers, hands, and arms. The distal-to-proximal evolution is known as a stocking-glove pattern (see *Picturing diffuse stocking-glove pattern in PN*).

Patients may have diminished sensation to vibration and proprioception to touch, heat, and cold. For example, a patient may have difficulty sensing or isolating a pinprick during an assessment of lower extremity sensory function. Patients may also have difficulty sensing the temperature of bath water that may be too warm or too cold, or the presence and extent of any tissue injury to the toes, feet, and legs. For example, a patient may not be aware of the beginning stages of an ingrown toenail. Patients may sit, stand, and walk in a way that maneuvers the feet to minimize or avoid painful dysesthesias.

Nerve degeneration progresses from sensory symptoms to include motor function problems of the lower and upper extremities. Gait may be impacted, creating safety issues with ambulation. Muscle aches, weakness, spasms, and cramps are common. Deep tendon reflexes may be diminished. Patients may stand and walk with a wide base of support to maintain balance. They may have peroneal nerve damage, causing foot drop with difficulty raising the feet sufficiently to walk safely without stumbling. A history of falls and fractures may be noted. Patients may have difficulty buttoning a shirt or blouse and with handwriting and holding objects.

Autonomic nerve damage may cause a fluctuation in heart rate and BP, leading to orthostatic hypotension. Patients are likely to experience heat intolerance, excessive sweating, difficulty while swallowing, nausea, diarrhea, and constipation. Urinary hesitance and incontinence are common. Sexual drive and performance are diminished in both men and women, including erectile dysfunction in men.

Picturing diffuse stocking-glove pattern in PN



Source: Louis ED, Mayer SA, and Rowland LP. *Merritt's Neurology.* 13th ed. Philadelpiha, PA: Wolters Kluwer; 2015.

Diagnostic testing

An analysis of lab data may correlate with the patient's neuropathic syndrome and systemic symptoms. Elevated levels of the liver enzymes gamma-glutamyl transpeptidase, aspartate aminotransferase, alanine aminotransferase, and alkaline phosphatase may indicate long-standing heavy alcohol consumption and its effects on hepatic function. Decreasing albumin, increasing bilirubin, and prolonged clotting factors may indicate hepatic decompensation. Red blood cells (RBCs) tend to be larger than normal (macrocytosis) and reduced in number from a deficiency in vitamin B9 or B12 or GI bleeding. Vitamin B1 and vitamin B3 levels are likely to be reduced. There may be an increase in erythrocyte macrocytic volume because alcohol interferes

with the development of normal RBCs. Glucose fluctuation, hyponatremia, hypokalemia, and hypomagnesemia are common features. An elevated carbohydrate-deficient transferrin level is a sensitive indicator of chronic heavy alcohol consumption. Transferrin is a serum protein that facilitates iron transport under the control of glycosyltransferase, which is a carbohydrate chain.

To evaluate the peripheral nervous system for primary neurologic pathology or secondary systemic disorders, electrodiagnostic testing consists of nerve conduction studies to measure nerve conduction and an electromyogram (EMG) to measure muscle conduction.

Nerve conduction studies are used in the diagnosis of PN related to diabetes mellitus, Guillain-Barré syndrome, myasthenia gravis, multiple sclerosis, amyotrophic lateral sclerosis, carpal tunnel syndrome, Charcot-Marie-Tooth disease, cervical and lumbar radiculopathy, and chronic heavy alcohol consumption. The median and ulnar nerves are evaluated for motor function and the median, ulnar, and sural nerves are evaluated for sensory function. The sural nerve plays an important role in the diagnosis of alcohol-induced PN because it's located in the calf and innervates sensory function in the lower legs where symptoms begin. Nerve conduction velocity may be normal or mildly diminished in the early stages of axonal degeneration, whereas demyelination causes significant slowing of conduction. The sensory nerve action potential shows decreased conduction amplitude in axonal injury. The H-reflex and F-wave are measures of peripheral nerve conduction, often delayed or absent in alcohol-induced PN. Abnormalities in the F-wave response are a sensitive and early indicator of alcohol-induced PN.

Needle EMG usually involves an evaluation of a proximal and distal muscle. A more comprehensive EMG analysis may be conducted when the patient has lumbosacral radiculopathy. The primary findings in alcohol-induced PN are a positive sharp wave, fibrillation potentials, and complex repetitive charges—electrical measures indicating severely degenerative muscle function.

A skin biopsy of the sural nerve may be conducted and will initially show small fiber neuropathy, which results from injury to nerve fibers that may be myelinated or unmyelinated. Injury to these small nerve fibers leads to somatic and autonomic symptoms. Allodynia—when pain is experienced by a stimulus that wouldn't normally cause pain—may be present in small fiber neuropathy. For example, a patient experiences pain or other dysesthesias when putting on socks. Patients with small fiber neuropathy commonly report burning pain and may tell you, "My feet burn." Patients may be hypersensitive to a stimulus (hyperesthesia). Patients may also experience numbness, restless legs syndrome, dry eyes and mouth, increased sweating, stomach problems, bladder control issues, skin discoloration, and cardiovascular symptoms.

Treatment options

A priority treatment approach is to increase the patient's caloric intake of nutritious foods. Chronic heavy alcohol consumption depletes hepatic proteins for energy and leads to a lack of B vitamins for carbohydrate metabolism and optimal functioning of the central and peripheral nervous systems. Patients may be severely malnourished and dehydrated with electrolyte imbalances. Nutrition and fluid requirements may be achieved through oral and I.V. replacement, especially when the patient is hospitalized and experiencing multisystem problems related to alcohol. Patients should follow a nutritious diet at home with lean meats, whole grains, vegetables, and fruits. Rich sources of vitamin B1 include organ meats, beef, pork, poultry, pasta, bread, rice, nuts, whole-grain cereals, and beans.

A priority treatment approach is to increase the patient's caloric intake of nutritious foods.

Benfotiamine, a synthetic derivative of vitamin B1, improves neuropathic pain and motor movement by increasing nerve conduction velocity. A nutritious diet; vitamin supplements, especially vitamins B1 and B12; and reduction of or abstinence from alcohol use is the only way to improve the patient's PN by allowing nerves to slowly regenerate.

Alcohol-induced PN can be managed with antiseizure medication, which has an anticholinergic effect on the central and peripheral nervous systems and blocks the uptake of serotonin and norepinephrine to decrease pain perception. Gabapentin, pregabalin, and carbamazepine are commonly used to alleviate burning and stabbing dysesthesias. Tricyclic antidepressants, including amitriptyline, desipramine, and nortriptyline, work similarly to the antiseizure medications. Duloxetine is a serotonin-norepinephrine reuptake inhibitor that may improve neuropathic pain. Desired outcomes of these medications include reduced pain and improved sleep. Taking these medications at bedtime may be indicated because of their sedative effects.

Capsaicin is a topical agent that modulates the inflammatory effects of the neurotransmitter neurokinin A to reduce neuropathic pain. Lidocaine skin patches may also be helpful. Nonsteroidal anti-inflammatory agents, acetylsalicylic acid,

Priority nursing interventions

Assessment

- Conduct a health history interview with a focus on your patient's neuropathic pain experience and alcohol consumption. Ask the following questions:
  - How intense is your pain?
  - How has pain affected your life?
  - Is your pain sharp, stabbing, burning, hot, cold, or numb sensations?
  - Do you ever feel as if needles or pins are sticking you?
  - Is your skin sensitive to touch?
  - What makes your pain better or worse?
  - How has alcohol consumption affected your health?
- Correlate any systemic findings during the head-to-toe assessment, lab data, and electrodiagnostic studies with your patient's neuropathic pain that may indicate chronic heavy alcohol consumption.
- Assess sensory function by gently touching the soles of your patient's feet, dorsum of the feet, toes, lower legs, fingers, hands, and arms. A piece of cotton wool may also be used. Gently touching your patient's skin with a needle may be helpful. Use a tuning fork to assess your patient's perception of vibration. Use a test tube filled with warm water and a second test tube filled with cold water and apply to the skin to assess thermal perception. Ask your patient if he or she is able to feel and isolate your touch and if the touch elicited any pain or discomfort. Perform a bilateral comparison.
- Assess motor function, including your patient's ability to move his or her arms and legs. Check the strength and equality of handgrips. Assess strength and equality of legs by asking your patient to push against your resistance. Assess your patient's ability to sit and move to a standing position while protecting him or her from falling. Ask your patient to walk and assess his or her gait and the ability to lift the feet off the ground.
- Evaluate your patient's responses to medications, physical therapy, occupational therapy, and the replenishing of B vitamins through nutritious foods. A physical therapist, occupational therapist, and dietitian should join the nurse and physician on the interprofessional team.

Patient teaching

Teach your patient to:

- consume nutritious foods in a well-balanced diet with vitamin supplements as prescribed and drink plenty of water
- stand slowly from a sitting position to avoid orthostatic hypotension
- report unrelieved or worsening pain and any systemic symptoms that correlate with the neuropathic pain; for example, bladder incontinence, hypertension, mental status changes, edema and shortness of breath that might indicate heart failure, and sexual dysfunction
- check water temperature before taking a bath and wear protective gloves when cooking or cleaning the house using chemicals to prevent burns
- have proper lighting in the home, use nightlights, and remove objects or loose rugs from the floor to prevent falls
- use a wheelchair, cane, walker, and proper fitting shoes and orthotic devices to ambulate to prevent falls
- check feet frequently for skin irritation or wounds and consult with the healthcare provider for foot care
- be active as tolerated and avoid smoking
- reduce or abstain from alcohol use
- seek counseling and be involved in a support group.

and acetaminophen may be helpful in mild PN as a complement to other medication. Opioids may be considered if used judiciously. Research indicates that the antioxidant alpha-lipoic acid may improve pain through nerve regeneration. Transcutaneous electrical nerve stimulation may increase conduction through neurons and improve neuropathic pain.

Patients with neuropathic pain may be less mobile, predisposing them to conditions such as pneumonia, deep vein thrombosis, skin breakdown, muscle atrophy and weakness, and depression. Physical therapy should be included in the treatment plan to improve flexibility, strength, and balance. Occupational therapy can help the patient with self-care activities and safely navigating the home environment. Orthotics using splints and braces should be explored to help with ambulation. Encourage patients to be active as tolerated to promote tissue oxygenation, mobility, and well-being. For example, walking, swimming, and yoga are beneficial activities.

A major treatment goal is for the patient to be involved in support groups or counseling that will help him or her reduce or abstain from alcohol use. Because support groups and counseling come in many forms, the patient, as an integral member of the team, will decide which group(s) and counseling formats are best for him or her.

Nursing care

Nursing care begins with establishing a rapport with the patient during the health history interview and head-to-toe assessment. The goals of nursing care for patients with alcohol-induced PN are to control pain, promote optimal nutritional status and mobility, and evaluate the patient's responses to treatment. Ensuring patient safety and interprofessional collaboration are major frameworks for achieving these goals. See *Priority nursing interventions* for more information.

A team approach

Regeneration of peripheral nerves takes time and requires the patient and interprofessional team to collaborate to improve the patient's symptoms. Abstinence from alcohol as a part of the treatment plan will help provide the best outcome to enhance the patient's quality of life. ■

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