Peripheral Neuropathy

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DESCRIPTION

Peripheral neuropathy is a neurodegenerative disorder characterized by damage to one or more peripheral nerves. The peripheral nervous system (PNS) transmits information from the central nervous system (CNS) to limbs and organs. Central neuropathy refers to damage sustained by the brain or the spinal cord, both of which are encompassed by the CNS. Though neuropathies may originate in the CNS, the majority originate in the PNS (Uzun et al., 2006). Damage to peripheral nerves causes distortion of information transmitted to and from the body and extremities (i.e., anything beyond the brain and spinal cord), resulting in symptoms ranging from temporary numbness to paralysis and organ or gland dysfunction.

There are several types of neuropathies, each distinguishable by the extent and location of the nerve damage (Andreoli, 2007). When disturbance in function occurs in one nerve, the neuropathy is classified as a mononeuropathy simplex. Mononeuropathy multiplex indicates involvement of several nonadjacent peripheral nerves. Polyneuropathies are more common, concurrently affecting the function of numerous peripheral nerves. The subclassifications of polyneuropathies include axonal neuropathies, principally affecting the axons, demyelinating neuropathies, involving the myelin sheath surrounding the axons, and neuronopathies, involving neurons of the PNS.

NEUROPATHOLOGY/PATHOPHYSIOLOGY

While there are numerous ways to classify and specify the disorder for diagnosing, the definitive symptoms appear to be caused by the cell body's response to the damage. This occurs most often in either the axon or the myelin sheath of the affected nerve fibers (Cros & Siao, 2001). Often, the neurophysiological features of axonal neuropathy are caused by traumatic injury and are symmetrical sensory and/or motor symptoms. In contrast, demyelinating neuropathies are often a result of nerve compression, creating interruptions in the transmission of nerve impulses (Cros & Siao, 2001).

Peripheral neuropathy is considered to be either an acquired disorder or a hereditary disorder. Idiopathic inflammatory neuropathies are a classification of acquired neuropathies. They are caused by inflammation from activities of the immune system and include acute idiopathic polyneuropathy (Guillain–Barré syndrome) and chronic inflammatory demyelinating polyneuropathy (see these entries within this book for further review).

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Metabolic and nutritional neuropathies can be caused by diabetes, uremia, liver disease, vitamin B12 deficiency, and other endocrinopathies including hypothyroidism and acromegaly. Vitamin B12 is an essential element of healthy nerve function, with a deficiency leading to widespread nerve tissue damage. Peripheral neuropathy is the most common complication of diabetes. Mild neuropathies accompanied by sensory abnormalities are found in up to 70% of diabetic patients while symptomatic neuropathies affect an additional 5–10% (Andreoli, 2007). The clinical form of neuropathy most diabetic patients present with is symmetric polyneuropathy associated with rapid physiologic dysfunction and hyperglycemia.

Infective and granulomatous neuropathies have a variety of causes including AIDS, diphtheria, leprosy, sarcoidosis, sepsis and multiorgan failure. Though diphtheria and leprosy are rare, Lyme disease is more common and can cause a rapidly developing polyneuropathy.

Mixed connective tissue disease, polyarteritis nodosa, rheumatoid arthritis, and systemic lupus erythematosus are known causes of vasculitic neuropathies. Certain connective tissue disorders, including polyarteritis nodosa and rheumatoid arthritis, may cause mononeuropathy multiplex or even polyneuropathy.

Neoplastic and paraproteinemic neuropathies are caused by compression and infiltration resulting from a tumor, paraneoplastic syndromes, paraproteinemias, and amyloidosis. Neurofibromatoses, genetic diseases involving tumors growing directly on nerve tissue, are often associated with polyneuropathies.

Drug-induced and toxic neuropathies are caused by alcohol, organic compounds such as hexacarbons and organophosphates, heavy metals such as arsenic, gold, lead, platinum, and thallium, and tryptophan. Thiamine deficiency is a common problem associated with alcoholism and can cause neuropathy of the extremities. Excessive alcohol consumption alone may directly cause damage to the peripheral nerves. Peripheral nerve damage is also a side effect of long-term use of anticancer drugs, anticonvulsants, and antibiotics.

Finally, hereditary neuropathies of an idiopathic nature may be caused by Friedreich's ataxia and familial amyloidosis, while hereditary neuropathies of a metabolic nature may be caused by porphyria, metachromatic leikodystrophy, Krabbe's disease, abetalipoproteinemia, Tangier's disease, Refsum's disease, and Fabry's disease. Hereditary neuropathies result from genetic code errors or new genetic mutations and may begin in infancy in severe cases or in early adulthood for milder forms (Dyck & Thomas, 2005).

NEUROPSYCHOLOGICAL/CLINICAL PRESENTATION

Proper operation of the axon and its myelin sheath is vital for the normal functioning of myelinated nerve fibers. When the axon begins to degenerate as a result of a variety of metabolic, toxic, and heritable causes, the myelin also begins to break down. Axonal degeneration of long nerve fibers is usually the underlying cause of polyneuropathies. When demyelination occurs in peripheral nerves as a result of demyelinating neuropathies, functional deficits identical to those resulting from axonal degeneration are produced. Demyelinating neuropathies are largely caused by inherited disorders of myelin, autoimmune attacks on myelin, and mechanical, toxic, and physical injuries to the nerve (Dyck & Thomas, 2005).

Clinical presentation of the neuropathies depends on the associated pathophysiologic mechanisms and the anatomical location of the nerve damage. Peripheral nerves have highly specialized functions, leading to a wide array of symptoms when the nerves are damaged. The most common symptom of damage to peripheral motor nerves is muscle weakness, while other symptoms may include cramps, muscle loss, bone degeneration, and fasciculations (Andreoli, 2007). Sensory nerve damage generally results in impaired and abnormal sensations and numbness. When involvement of peripheral nerve fibers of a certain size is selective, dissociated sensory loss may occur. In such cases, certain sensory modalities are impaired while others are preserved. Additional sensory and reflex changes associated with the motor neuron deficits suggest peripheral nerve damage.

When small fibers within nerves are affected by the neuropathy, pain is a prominent symptom. Pain is an established symptom of neuropathies related to alcohol use and diabetes and may also be a feature of entrapment neuropathies. Patients with diabetic polyneuropathies generally present with autonomic dysfunction, spontaneous sensations of neuropathic pain including dysesthesias, and reduced pain sensibility. Diabetic neuropathy occurs most often in patients with insulin-requiring long-standing diabetes, and a diagnosis can be confirmed with electrodiagnositic studies. In a study conducted by Turcot et al. (2009), participants with diabetic neuropathy also presented with greater postural instability than control participants. L-Periaxin is a protein involved in the stabilization of mature myelin in peripheral nerves. Lawlor et al. (2002) report the presence of anti-L-periaxin antibodies in patients with diabetes-associated peripheral neuropathy, resulting in morphologic abnormalities of the sensory nerves.

When the neural pathways that mediate reflexes are interrupted, tendon reflexes may be impaired or lost. Patients with polyneuropathies generally experience loss of ankle reflexes first, followed by additional tendon reflex loss.

Symptoms associated with Guillain–Barré syndrome and diabetic neuropathies or those caused by amyloidosis include coldness of extremities, bladder and bowel function disturbances, impotence, and postural hypotension. Along with being an additional symptom of amyloidosis, enlarged peripheral nerves may be an indication of hereditary motor and sensory neuropathies and neuropathies associated with leprosy, Refsum's disease, and acromegaly. Again, the interested reader is referred to these entries within this text.

DIAGNOSIS

Due to high symptom variability, diagnosis of neuropathy can be challenging. Symptoms of a sensory nature are often the first clue of peripheral nerve involvement. Acute development of symptoms usually relates to an inflammatory polyneuropathy, while gradual evolution of symptoms is an indication of hereditary or metabolic polyneuropathies. Mononeuropathies with acute symptom presentation generally result from traumatic causes. Symptoms caused by minor traumatic injuries and entrapment of nerves are more likely indications of mononeuropathies of gradual onset. Along with drug and alcohol history, occupational history should also be considered during patient evaluation as certain industrial substances may lead to peripheral neuropathy.

When a diagnosis of neuropathy is suspected, electromyography may be used to determine location of denervation. Nerve conduction velocity tests are used to determine whether nerve damage is the result of myelin sheath or axon degeneration. Demyelinating and axonal neuropathies may be differentiated using electrodiagnositc or histopathologic studies. Nerve biopsies may also be conducted to determine degree of nerve damage. When a diagnosis of pheripheral neuropathy is confirmed electrodiagnostically, additional laboratory studies including blood glucose levels, serum vitamin B12, and liver and thyroid function blood tests should be conducted. Magnetic resonance imaging can detect nerve damage caused by compression and can provide data on muscle size and quality. Abnormal antibodies associated with neuropathies can be identified during examination of cerebrospinal fluid, and simple tests to evaluation ability to register sensations may reveal the size of sensory nerve fibers affected.

Sabin (2001) notes, however, that if one can establish type of onset, family history, general medical state, and previous exposure to medications or toxins, one can easily diagnose the individual accurately. Burns et al. (2006) also reported a simple way to clarify the varying classifications is by asking “What?,” “Where?,” “When?,” and “What setting?,” and by following a type of decision tree based on the responses to those questions.

TREATMENT

Treatment approaches are driven by the accurate diagnosis, as they are either symptomatic or etiological in nature (Vanotti et al., 2007). Though treatments of inherited forms of neuropathy are limited, depending on classification, acquired neuropathies may be treated in several ways. Generally the underlying cause of the neuropathy is treated first to limit progression of the symptoms or even reverse the neuropathy itself before symptomatic treatment is initiated.

Adopting a healthy lifestyle, avoiding exposure to toxins, limiting alcohol consumption, correcting vitamin deficiencies, and eating a balanced diet are all helpful strategies to reduce the physical symptoms of many neuropathies. Regular exercise may help in reducing cramps associated with certain neuropathies and improve muscle strength, a vital component of avoiding muscle wasting associated with limb paralysis.

Plasmapheresis shortens recovery time associated with Guillain–Barré syndrome, while intravenous immunoglobulin in high doses is an equally effective treatment. Careful monitoring of respiratory function in patients with Guillain–Barré syndrome, diphtheritic neuropathy, and other idiopathic inflammatory neuropathies is essential to effective treatment.

Correction of blood glucose levels is important for prevention and reducing neuropathic symptoms associated with diabetic neuropathy. Anticonvulsants and tricyclic antidepressants have also been found to reduce the painful symptoms of diabetic neuropathy

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J. T. Hutton
Rachel Rock
O. J. Vidal
Antonio E. Puente