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Diabetic Neuropathy: A Brief Overview

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About the Study

Diabetes is one of the most prevalent causes of neuropathy, and it has a high rate of morbidity and mortality, resulting in more hospitalizations for diabetics than any other illness. A variety of neuropathy patterns can be recognised. These are divided into three categories: Symmetric, asymmetric, and focal. A distal symmetric sensory motor neuropathy affects at least half of people with long-term diabetes. Even in the pre-diabetic state, peripheral neuropathy can occur early in the course of diabetes. Up to 56 percent of individuals with idiopathic peripheral neuropathy have impaired glucose tolerance, which is three times the incidence of matched controls with normal glucose tolerance. The degree and duration of hyperglycaemia are important risk factors for the development of peripheral neuropathy.

Hypertension, high triglycerides, cigarettes, obesity, micro vascular illness, and cardiovascular disease are all risk factors. The onset is usually gradual, beginning in the toes and forefeet and advancing proximally over time. The majority of patients have both large and small nerve fibre involvement, while large fibre involvement frequently predominates. Both motor and sensory nerves are affected by large fibre involvement. Pain is limited, but when it does occur, it is described as a dull, cramping discomfort. Before symptoms appear, a physical examination may identify abnormalities. Loss of vibratory sensation and position sensation, as well as reduced Achilles reflexes, is some of the first symptoms. Even when monofilament testing is normal, using a tuning fork to detect vibratory loss can reveal neuropathy. Electro diagnostic investigations provide findings that are consistent with axon loss and demyelination.

Sensory abnormalities are the first to appear, with sensory investigations revealing the earliest changes on the skin. Because of their more distal placement, medial plantar nerve conduction investigations are more sensitive in patients younger than 60. Motor nerve conduction problems develop later in the disease's progression. A symmetrical pattern of neuropathic abnormalities will be visible on an EMG needle test, with a distal to proximal gradient. Although quantifiable strength testing often reveals decreasing strength, this often occurs before there is any clinical impairment. Small unmyelinated C fibre involvement causes considerable discomfort and hyperalgesia, and it can happen early in diabetes. There is a loss of temperature awareness as well as autonomic abnormalities such as loss of sweating, dry feet, and vasomotor alterations as the disease advances. Foot ulcers and infections are more likely a result of this. The discomfort may decrease as the neuropathy advances, but this is an indication of disease progression rather than regression. There is no difference between small fibre illness and large fibre disease when small fibre disease develops without large fibre involvement. When small fibre illness develops without large fibre involvement, physical examination findings can be limited despite considerable symptoms. Furthermore, electro diagnostic investigations for tiny fibre nerves are less sensitive and can be normal. Galvanic skin responses can be aberrant, but they're not always accurate. Skin biopsy is not done on a regular basis, however it can be used to determine the quantity of tiny fibres present. It is critical to emphasise the importance of blood sugar control in preventing diabetic PN. Glycaemic management has been reported to reduce the prevalence of PN by over 70% and autonomic dysfunction by more than 50%. Because tight glycaemic control has been found to reverse PN38, it's critical to educate newly diagnosed type 2 diabetes patients about the necessity of starting strong glycaemic control as soon as possible to prevent the risk of PN.

Proximal Neuropathy is a type of neuropathy. Diabetic amyotrophy, diabetes lumbosacral radiculoplexus neuropathy, and Bruns-Garland syndrome are all terminology used to describe diabetic proximal neuropathy. It usually manifests as acute or sub-acute discomfort in elderly people with type 2 diabetes that is unilateral or bilateral, and is frequently accompanied by weight loss. The pain tends to decrease over weeks to months, and is accompanied by significant atrophy of the thigh muscles, particularly the quadriceps and adductors with the gluteal and hamstring muscles maintaining relatively unaffected. Sensory abnormalities are most commonly observed in the femoral and saphenous distributions.

Diabetic individuals with pre-existing distal neuropathy are usually often diagnosed with proximal diabetic neuropathy. In a younger patient with early, moderate diabetes, a variation can present with acute and debilitating discomfort. On needle EMG examination, electro diagnostic investigations reveal a pattern of lumbosacral plexopathy, which is sometimes associated with multilevel radiculopathy. With reduced CMAP but relatively conserved distal latency, femoral nerve conductions suggest primarily axonal involvement. Even in patients who have no clinical complaints of distal neuropathic symptoms, distal nerve conduction investigations may reveal signs of contemporaneous distal neuropathy. An immune-mediated microvasculitis affecting the nerve roots and plexus is assumed to be the cause of damage. Diabetic amyotrophy is treated with strict glycemic control and rigorous neuropathic pain treatment.