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ORIGINAL ARTICLE

CLINICAL-NEUROLOGICAL AND INSTRUMENTAL ASSESSMENT OF THE INFLUENCE OF CARBOHYDRATE METABOLISM ON THE COURSE OF DIABETIC NEUROPATHY

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Abstract

Diabetic neuropathy is one of the most common and disabling chronic complications of diabetes mellitus, significantly affecting patients' quality of life and functional capacity. Disturbances in carbohydrate metabolism play a crucial role in the development and progression of peripheral nerve damage through complex metabolic, vascular, and inflammatory mechanisms. The present study aimed to evaluate the influence of carbohydrate metabolism on the clinical course of diabetic neuropathy using neurological examination and instrumental diagnostic methods. A total of 150 patients with diabetes mellitus and varying degrees of neuropathic manifestations were examined. Clinical assessment included neurological evaluation of sensory, motor, and autonomic functions, while instrumental investigations involved electroneuromyography, quantitative sensory testing, and laboratory analysis of glycemic parameters. The results demonstrated a significant association between poor glycemic control and increased severity of neuropathic symptoms, nerve conduction abnormalities, and functional impairment. Patients with prolonged hyperglycemia exhibited more pronounced neurological deficits and structural nerve dysfunction. The findings emphasize the importance of strict metabolic control in preventing progression of diabetic neuropathy and improving neurological outcomes. Comprehensive clinical and instrumental evaluation provides valuable information for early diagnosis, monitoring, and individualized management of diabetic neuropathy.

Keywords: diabetic neuropathy, carbohydrate metabolism, diabetes mellitus, hyperglycemia, nerve conduction, neurological assessment, electroneuromyography, peripheral neuropathy, glycemic control, diabetic complications.

1. Introduction

Diabetes mellitus is a chronic metabolic disorder characterized by persistent disturbances in carbohydrate metabolism resulting from defects in insulin secretion, insulin action, or both. The global prevalence of diabetes continues to rise, creating a substantial burden on healthcare systems and increasing the frequency of long-term complications affecting multiple organ systems. Among these complications, diabetic neuropathy represents one of the most prevalent and clinically significant manifestations due to its progressive nature and profound impact on physical functioning and quality

of life.

Diabetic neuropathy encompasses a heterogeneous group of disorders affecting peripheral and autonomic nervous system structures. The condition develops gradually and is often associated with prolonged exposure to hyperglycemia, metabolic imbalance, oxidative stress, and microvascular dysfunction. Clinical manifestations vary considerably and may include sensory loss, pain, paresthesia, muscle weakness, impaired reflexes, and autonomic disturbances affecting cardiovascular, gastrointestinal, and genitourinary functions.

Carbohydrate metabolism plays a central role in the pathogenesis of diabetic neuropathy. Persistent elevation of blood glucose levels initiates a series of biochemical alterations that contribute to neuronal injury. Excess intracellular glucose activates multiple pathogenic pathways, including increased polyol pathway activity, accumulation of advanced glycation end products, oxidative stress generation, and activation of inflammatory mediators. These mechanisms collectively impair nerve function and promote structural degeneration of peripheral nerve fibers.

Microvascular abnormalities also contribute significantly to neuropathic progression. Chronic hyperglycemia damages endothelial cells and compromises blood supply to peripheral nerves. Reduced oxygen delivery and impaired nutrient transport lead to ischemic injury and further exacerbate neural dysfunction. Consequently, both metabolic and vascular factors interact to accelerate nerve damage in individuals with poorly controlled diabetes.

Clinical manifestations of diabetic neuropathy frequently correlate with the duration and severity of metabolic disturbances. Patients with inadequate glycemic control often demonstrate earlier onset of neurological symptoms and more rapid disease progression. Furthermore, fluctuations in glucose levels may contribute to instability of neurological function and increased risk of complications such as foot ulceration and lower extremity amputation.

Neurological examination remains a cornerstone of diabetic neuropathy diagnosis. Assessment of sensory perception, muscle strength, tendon reflexes, coordination, and autonomic function provides valuable information regarding disease severity and distribution. However, clinical examination alone may not detect early subclinical nerve involvement, emphasizing the importance of complementary instrumental investigations.

Electroneuromyography is considered one of the most informative diagnostic tools for evaluating peripheral nerve function in diabetic neuropathy. Measurement of nerve conduction velocity, amplitude, and latency allows objective assessment of neural integrity and facilitates detection of both axonal and demyelinating abnormalities. Additional diagnostic methods such as quantitative sensory testing and autonomic function assessment contribute to comprehensive characterization of neuropathic involvement.

Early identification of neuropathic changes is essential because timely intervention may slow disease progression and reduce the risk of severe complications. Intensive glycemic management has been shown to improve metabolic stability and may help preserve nerve function, particularly during the early stages of disease development. Therefore, understanding the relationship between carbohydrate metabolism and neurological impairment remains a priority in diabetic care.

Despite significant advances in diabetes management, diabetic neuropathy continues to present considerable diagnostic and therapeutic challenges. Variability in clinical presentation and disease progression necessitates comprehensive assessment strategies that integrate metabolic, neurological, and instrumental findings. Such approaches may improve early detection, facilitate individualized treatment planning, and enhance long-term patient outcomes.

The aim of this study was to evaluate the influence of carbohydrate metabolism on the clinical course of diabetic neuropathy through detailed neurological examination and instrumental assessment, and to determine the relationship between glycemic control and severity of peripheral nerve dysfunction.

2. Materials and Methods

This clinical observational study was conducted between 2024 and 2025 at specialized endocrinology and neurology departments. The investigation included 150 patients diagnosed with type 1 or type 2 diabetes mellitus who presented with varying manifestations of diabetic neuropathy. Participants ranged in age from 35 to 75 years and had a documented history of diabetes for at least three years. Patients were divided into three groups according to the degree of carbohydrate metabolism compensation. Group A consisted of patients with satisfactory glycemic control, Group B included individuals with moderately controlled diabetes, and Group C comprised patients with persistently

elevated glycemic indicators and poor metabolic control.
A comprehensive clinical-neurological examination was performed for all participants. Neurological assessment included evaluation of sensory perception, pain sensitivity, vibration sensation, temperature discrimination, tendon reflexes, muscle strength, coordination, and gait characteristics. Symptoms such as numbness, burning sensations, paresthesia, muscle cramps, nocturnal pain, and lower limb weakness were systematically documented.
Anthropometric measurements, blood pressure assessment, duration of diabetes, body mass index, and associated comorbidities were recorded. Laboratory evaluation focused on carbohydrate metabolism indicators including fasting plasma glucose, postprandial glucose, glycated hemoglobin (HbA1c), and insulin levels when clinically indicated.
Instrumental investigations included electroneuromyography (ENMG), which was used to evaluate peripheral nerve conduction velocity, amplitude of motor and sensory responses, distal latency, and electrophysiological evidence of axonal or demyelinating damage. Quantitative sensory testing was additionally employed to assess subtle sensory deficits that may not be evident during routine clinical examination.
Autonomic nervous system involvement was evaluated through cardiovascular reflex testing, heart rate variability assessment, and analysis of orthostatic responses. Patients with severe neurological disorders unrelated to diabetes, active infectious diseases, malignancies, alcohol-related neuropathy, or exposure to neurotoxic agents were excluded from the study.

3. Results

The study demonstrated a strong relationship between carbohydrate metabolism disturbances and the severity of diabetic neuropathy. Patients with poor glycemic control exhibited significantly more pronounced neurological impairment compared with individuals maintaining satisfactory metabolic regulation.
Clinical examination revealed that sensory disturbances were the most common manifestations of neuropathy. Numbness, tingling sensations, burning pain, and reduced vibration sensitivity were reported predominantly by patients with elevated HbA1c levels. These symptoms were especially prominent in the distal regions of the lower extremities, reflecting the characteristic pattern of diabetic polyneuropathy.
Motor dysfunction was observed more frequently among patients with prolonged diabetes duration and persistent hyperglycemia. Reduced muscle strength, impaired coordination, diminished ankle reflexes, and gait instability were identified with increasing frequency as metabolic control deteriorated. Several participants in the poorly controlled group demonstrated signs of advanced peripheral nerve involvement affecting daily functional activities.
Electrophysiological assessment confirmed significant abnormalities in nerve conduction parameters. Patients with inadequate carbohydrate metabolism compensation exhibited reduced motor and sensory nerve conduction velocities, prolonged distal latencies, and decreased response amplitudes. These findings indicated progressive deterioration of peripheral nerve structure and function associated with chronic metabolic imbalance.
Quantitative sensory testing revealed early impairment of thermal and vibration perception even among certain patients with relatively mild clinical symptoms. This observation suggests that instrumental diagnostic methods may detect neuropathic changes before substantial clinical disability develops.
Autonomic nervous system involvement was identified in a considerable proportion of participants. Orthostatic hypotension, reduced heart rate variability, excessive sweating abnormalities, and gastrointestinal autonomic disturbances occurred more frequently in individuals with long-standing hyperglycemia. The prevalence and severity of autonomic dysfunction increased proportionally with worsening glycemic indicators.
Correlation analysis demonstrated a significant association between HbA1c values and neuropathy severity scores. Patients exhibiting persistently elevated glycated hemoglobin concentrations showed greater neurological impairment, more extensive electrophysiological abnormalities, and increased prevalence of neuropathic pain syndromes.
The duration of diabetes also influenced disease progression. Individuals living with diabetes for more than ten years experienced substantially greater neurological deficits compared with patients diagnosed more recently, particularly when metabolic control remained inadequate over prolonged

periods.

4. Discussion

The findings of the present study confirm the central role of carbohydrate metabolism disturbances in the development and progression of diabetic neuropathy. Chronic hyperglycemia appears to initiate a cascade of metabolic and vascular abnormalities that collectively contribute to peripheral nerve injury. One of the principal mechanisms responsible for neuropathic damage involves prolonged exposure of neural tissues to elevated glucose concentrations. Excess intracellular glucose promotes activation of alternative metabolic pathways, accumulation of toxic metabolites, oxidative stress generation, and inflammatory responses. These processes impair neuronal function and accelerate structural degeneration of nerve fibers.

The significant association between HbA1c levels and neuropathy severity observed in this study highlights the importance of long-term glycemic control. Glycated hemoglobin serves as a valuable indicator of chronic metabolic status and provides insight into cumulative glucose exposure. Patients with persistently elevated HbA1c values demonstrated more severe clinical symptoms and greater electrophysiological impairment, supporting the concept that sustained hyperglycemia is a major determinant of neuropathic progression.

Electroneuromyographic findings provided objective evidence of peripheral nerve dysfunction and complemented clinical observations. Reduced nerve conduction velocity and diminished response amplitudes reflected both axonal injury and myelin damage. These abnormalities became increasingly pronounced as metabolic control worsened, emphasizing the value of instrumental assessment in monitoring disease evolution.

The detection of subclinical sensory impairment through quantitative sensory testing underscores the importance of early diagnostic strategies. Many patients may develop measurable nerve dysfunction before significant clinical symptoms become apparent. Early identification creates opportunities for timely intervention and potentially slows disease progression.

Autonomic neuropathy represents a particularly important aspect of diabetic nerve involvement. The observed cardiovascular and gastrointestinal manifestations indicate that diabetic neuropathy extends beyond somatic sensory and motor fibers. Recognition of autonomic dysfunction is essential because it may contribute significantly to morbidity and adversely affect patient survival.

The influence of diabetes duration on neuropathy severity further supports the cumulative nature of metabolic injury. Long-term exposure to hyperglycemia increases the likelihood of progressive neural damage, especially when glycemic control remains inadequate. Consequently, early and sustained metabolic management should be considered a primary preventive strategy.

These findings reinforce the necessity of multidisciplinary diabetic care integrating endocrinological management, neurological monitoring, lifestyle modification, and patient education. Comprehensive approaches addressing both metabolic and neurological aspects of disease may improve clinical outcomes and reduce disability associated with diabetic neuropathy.

Although the study generated valuable information, certain limitations should be acknowledged. The investigation was conducted within a defined patient population and observation period. Larger multicenter studies with extended follow-up durations may provide additional insight into long-term neuropathic progression and therapeutic responses.

5. Conclusion

The present study demonstrated a significant relationship between carbohydrate metabolism disturbances and the clinical course of diabetic neuropathy. Poor glycemic control was strongly associated with increased severity of neurological symptoms, impaired nerve conduction, autonomic dysfunction, and progressive peripheral nerve damage.

Clinical-neurological examination combined with instrumental diagnostic techniques provided comprehensive evaluation of neuropathic involvement and facilitated identification of both symptomatic and subclinical disease manifestations. Electroneuromyography and quantitative sensory testing proved particularly valuable for detecting early nerve dysfunction and monitoring disease progression.

Persistent hyperglycemia and prolonged diabetes duration emerged as major factors contributing to neurological deterioration. Patients with elevated glycated hemoglobin levels exhibited significantly

greater clinical and electrophysiological abnormalities than individuals with adequate metabolic control.

The findings emphasize the critical importance of strict carbohydrate metabolism regulation in preventing neuropathic progression and preserving neurological function. Early diagnosis, regular neurological assessment, and individualized metabolic management strategies may substantially improve outcomes for patients with diabetes mellitus.

Comprehensive integration of endocrinological and neurological approaches remains essential for reducing the burden of diabetic neuropathy and enhancing quality of life among affected individuals.

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