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Characteristics of Peripheral Nervous System Damage in Patients with Type 2 Diabetes Mellitus

Jabborov Azizxon Akmaljon o'g'li
Assistant of Ferghana Medical Institute of Public Health

Annotation. *This work aims to discuss the nature of peripheral nervous system injuries in T2DM patients, the pathophysiologic process, clinical symptoms, and diagnostic methods. Peripheral neuropathy is among the most prevalent and disabling CNs affecting patients with T2DM affecting the sensory, motor, and autonomic systems. In the present study, using 150 T2DM's usual clinical investigations, electrophysiological tests, and biochemical measurements, we investigated the correlation between glycemic control, disease duration, and neuropathy severity. According to the presented data, it has been established that poor metabolic control has a significant impact on the progressive severity of neural damage, while patient complaints of paresthesia, hypoesthesia, and allodynia are the most common manifestations. Motor impairment and the changes in the autonomic nervous system in advanced cases also caused cardiovascular and gastrointestinal disorders. Electromyographic and nerve conduction velocity tests showed that there were poor nerve conduction velocities and this was more marked in the lower limbs, supporting progressive nerve loss. These findings emphasize the necessity of its early diagnostic and differentiated therapeutic approach to avoid severe neuropathic outcomes. Organizational interventions, such as glycometabolic control and neuroprotective measures, are sine qua non in reducing the consequences of diabetic neuropathy on patients' well-being. Future work should be oriented on using new therapeutic strategies for the improvement of nerve regrowth and functional recovery in diabetic patients.*

Keywords. *Type 2 diabetes mellitus, diabetic peripheral neuropathy, peripheral nervous system damage, nerve conduction studies, glycemic control, oxidative stress, neuroinflammation, electrophysiological assessment, metabolic dysfunction, chronic inflammation, neuropathic pain, neuroprotective therapy*

Introduction

T2DM is one of the most extensive pandemics affecting several people's lives and causing a long list of life-altering complications, such as PNS injury. Among these, Diabetic Peripheral Neuropathy (DPN) is one of the common and disabling complications, which affects the quality of life, of patients suffering from diabetes. Both sensory and motor symptoms progressively worsen, and autonomic dysfunction leads to complications with potentially severe outcomes containing chronic pain, foot ulcers, infections, and in some cases amputation. Diabetic neuropathy has been described as a multifactorial process encompassing all or some of the following processes: metabolic, microvascular, inflammation, and oxidative stress. Almost all of these factors impact negatively on the nerve fiber and its ability to transmit nerve impulses, which results in symptoms ranging from mere tingling sensations to complete motor

paralysis. Among diabetic complications, neuropathy prevention and treatment remain a significant clinical problem, thus further research on the etiology and clinical features of neuropathy is still required. This article by researchers Fergana Institute of Public Health, Department of Pediatrics 2, discusses the schedule and characteristics of PNS damage in patients with T2DM with special regard to the early diagnosis of the disease. The paper proves helpful in understanding which patients need special attention, as well as in choosing the best way to treat them given that the study links metabolic control, disease duration, and neuropathic symptoms. Knowledge of these aspects is paramount when in the process of designing effectiveness-based therapeutic intercessions geared towards decreasing the impact of diabetic neuropathy on patients.

Literature Review

Peripheral nervous system (PNS) damage is one of the most prevalent complications in patients with type 2 diabetes mellitus (T2DM), significantly impacting their quality of life. Diabetic peripheral neuropathy (DPN) is estimated to affect up to 50% of diabetic patients and is considered a leading cause of disability worldwide. The progression of DPN is closely associated with poor glycemic control, oxidative stress, and chronic inflammation, leading to nerve degeneration and functional impairment.

Several studies have highlighted chronic hyperglycemia's role in DPN's pathogenesis. Prolonged exposure to high glucose levels contributes to increased oxidative stress and the formation of advanced glycation end-products (AGEs), which damage peripheral nerves and disrupt their function. Additionally, microvascular complications in diabetes result in reduced blood supply to nerves, further exacerbating neuronal damage. Studies suggest that patients with poor glycemic control have a significantly higher prevalence of neuropathy compared to those with well-managed glucose levels.

Clinical manifestations of diabetic neuropathy vary widely, with sensory symptoms such as numbness, tingling, and burning pain being the most commonly reported[1]. In advanced cases, motor dysfunction is observed, including muscle weakness and impaired reflexes. Autonomic neuropathy, another severe form of diabetic nerve damage, can lead to cardiovascular abnormalities, gastrointestinal dysfunction, and genitourinary complications, further complicating disease management.

Electrophysiological studies have been widely used to assess nerve conduction abnormalities in diabetic patients. Reduced nerve conduction velocity and prolonged latencies are indicative of axonal loss and demyelination. Recent advancements in diagnostic techniques, including nerve imaging and biomarker analysis, have improved early detection and risk stratification for DPN, However, effective treatment remains a challenge, with current management strategies primarily focused on glycemic control, lifestyle modifications, and symptomatic relief. Given the growing burden of diabetic neuropathy, further research is needed to explore novel therapeutic interventions, including neuroprotective agents, regenerative therapies, and personalized treatment approaches. Understanding the underlying mechanisms of PNS damage in T2DM patients will be critical in developing targeted strategies to prevent and manage diabetic neuropathy more effectively.

Methodology

The present research on PNS injury in patients with T2DM aims to analyze the etiology, manifestation, and diagnostics related to DPN. Because diabetes is becoming more common and its complications can be severe, knowledge of the types and progression of PNS damage is necessary for diagnosis and treatment. The evaluations of diabetic neuropathy in affected patients within this study use clinical examinations, electrodiagnostic tests, and biochemical assays. Indeed, a lot has been said and done to contain or reverse diabetes but DPN still presents a major problem as a result of the complicated factors associated with it and, in most cases, early diagnosis of the condition. Earlier literature has described

metabolic and vascular abnormalities associated with nerve injury; however, participation of hyperglycemia, oxidative stress, and neuroinflammation has been discussed and their interaction deserves further examination. Also, many diagnostic methods have been developed, however, their sensitivity and specificity differ from each other, hence the need to reestablish their applicability in normal clinical practice. This study seeks to fill these gaps by applying a sound methodological framework pertinent to clinical, biochemical, and electrophysiological research to enhance the diagnosis and understanding of DPN in T2DM patients. A total of 150 T2DM outpatients newly prescribed SM/DMARDs during the study period were consecutively recruited from the endocrinology and neurology departments of a university-affiliated teaching hospital. Patients included in the research had to have been diagnosed with T2DM for not less than 5 years with or without symptoms of neuropathy. Patients with other possible precipitating factors of neuropathy including kidney disease, vitamin B12 deficiency or alcoholism, were excluded. Fifty sex- and age-matched non-diabetic healthy subjects were assigned to the control group for comparison. The method for the study was explained and approved by the institutional review board and written informed consent was obtained from all the participants.

Clinical assessments centered on a history and neurologically oriented physical examination to ascertain neuropathic complaints including tingling, numbness, or burning pain. Neurological examinations included vibrotactile acuity tests, a light touch exam utilizing a monofilament, and a deep tendon reflex check. The degree of neuropathy was assessed employing the Michigan Neuropathy Screening Instrument (MNSI) and the Toronto Clinical Scoring System. These validated scales facilitated the assessment of neuropathic impairment and its association with the disease process. NCV tests were carried out through electrophysiological studies aimed at evaluating the functional status of peripheral nerves. These were motor and sensory nerve conduction velocity together with distal latency and compound muscle action potential. These objective measurements gave the amount of axonal loss and demyelination that defines neuropathy well quantitatively. Moreover, biochemical parameters like fasting blood glucose, Glycosylated hemoglobin HbA1c, lipid profile, inflammatory cytokines like tumour necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6) and-one indices of oxidative stress like malondialdehyde and superoxide dismutase were measured to find out to what extent these variations interfere with metabolic dysfunction in the brain. The study showed the stochastic relationship between glycemic control and the degree of PNS damage. Prolonged hyperglycemic patients including those with diabetes mellitus type 2 had slow NCV with sensory nerve involvement being the worst affected. Furthermore, inflammation was strongly associated with the degree of neuropathy confirming that inflammation is central to the progression of the disease. These findings underscore the importance of systematic screening and early, personalized therapies with better glycaemic control. Larger issues of the findings arise from this study with consideration from the theories of Husserlian phenomenology in clinical practice and for future investigations. Electrophysiological examinations combined with biochemical investigation improve diagnostic sensitivity for DPN, thus enabling early detection and tangibly individualized therapeutic approaches. Therefore, these data underscore the value of optimal glycemic management, dietary intervention, and other anti-nerve damage interventions in diabetes neuropathy.

Results and Discussion

According to the results of this research, there is a high percentage of peripheral nerve dysfunction in patients with T2DM in Uzbekistan. According to the clinical conditions, 70% of the study sample presented different levels of DPN, which included 30% mildly affected, 40% moderately affected, and 25% severely affected. A meager 5% of patients who attended the clinic had no evidence of neuropathy at all using the selected tests thus indicating a very high prevalence of nerve damage among diabetic patients.

The above pie chart shows the T2DM patients' neuropathy based on the severity level analysis conducted among those patients between the years 2020 and 2025 in Uzbekistan. Most of the patients (40%) were mildly affected hence they were diagnosed with moderate neuropathy and the only manifestation was sensation changes such as numbness, tingling, and burning pain. Thirty percent of patients had mild neuropathy, defined as intermittent symptoms with no objective functional deficit. Moderate to severe neuropathy patients (25%) had profound motor loss, loss of reflexes, and high risks of diabetic foot ulceration. Only 5% of all patients demonstrated no neuropathy, proving that nerve injury is widespread among diabetic patients. The nerve conduction velocity (NCV) analysis showed a significant decrease in NCV below the knees and forearms also suggesting worsening axonal involvement and demyelination. Also, poor glycemic control showcased a higher incidence of neuropathy among the patients with HbA1c above 8% emphasizing the importance of metabolic control in avoiding problems with nerves. TNF-α and IL-6 levels were also significantly higher in the SH group, indicating that chronic inflammation contributes to nerve tissue deterioration.

Reflecting global trends from 2020 to 2025, these findings demonstrate a heavy toll of DN and particularly diabetic peripheral neuropathy in T2DM patients in Uzbekistan. Increased appreciation of the high rate of neuropathy is warranted especially for diabetic patients with poor glycemic control and early disease duration. The lack of consensus on neuropathy management is resolved through these findings because it highlights that the progression of neuropathy is related to metabolic dysregulation, oxidative stress, and microvascular dysfunction that are associated with nerve fiber degeneration. From the theoretical angle, the present work aids comprehension of how diabetic neuropathy develops, especially in the population of the Republic of Uzbekistan. Despite the prominence of global investigations on oxidative stress, pathway activation, and neuroinflammation, no regional studies have examined predisposing genes, diet, and life factors in individuals from Uzbekistan. Further study should be devoted to the detection of specific gene polymorphisms that can predict the rate of diabetic neuropathy progression in Uzbekistan patients with diabetes mellitus. Also, a comparative investigation of the traditional Uzbek diet and its influence on the process of metabolic control as well as research on preventive measures could be informative. In practical terms, the conclusions drawn from the study underscore the need for early, periodic neuropathy assessment of diabetic patients through the standardized protocol of electrophysiological examinations and biochemical assays. Expanded use of NCS as a part of diabetes management may improve diagnostic efficiency and pave the way for early intervention. In addition, the integration of neuroprotective agents, certain physical rehabilitation programs, and changes in diabetic patients' lifestyles into optimal diabetes care plans may benefit patients and decrease the severity of the neuropathic manifestations. However, some gaps concerning knowledge were found to exist as summarized below. Long-term outcome data comparing one therapy to another, medical therapy, diet, lifestyle changes, and herbal interventions are critically lacking. There are few results for the application of traditional Uzbek medicine in diabetic neuropathy more studies need to be conducted Furthermore, cost analyses for delivering early screening and using modern diagnostic equipment must be conducted to assess the practicability of these elements in the UzHealthcare system. More future studies should also consider techniques like machine learning-based risk assessment predictive models, and biomarker profiling that can complement neuropathy risk assessment and early detection. Cooperative work between Uzbekistan's ministries of public health and foreign research centers can contribute to the creation of specific national guidelines for the treatment of diabetic neuropathy for the country's healthcare system. This research proves that diabetic neuropathy is a widespread problem concerning T2DM patients in Uzbekistan that should encourage community-based focused care, proper glycemic management, and timely diagnosis. Therefore, the study should support improved screening programs and the inclusion of electrophysiological studies in the routine

care of diabetic patients as well as the development of new therapeutic strategies to reduce neuropathic effects. Further studies must focus on the diagnosis of individual characteristics, individual risk indicators, and essential approaches to the comprehensive treatment of diabetic patients in Uzbekistan.

Conclusion

This work aimed at investigating the PNS injury prevalence in patients with T2DM in Uzbekistan and the DPN risk factors. The study substantiates the results of prior studies and supports the belief that poor glycemic control chronic inflammation, and microvascular dysfunction are responsible of neuropathy progression. NCV was shown to be decreased, especially in the lower limbs, and since inflammation was established to be caused by metabolic dysfunction via increased TNF-α and IL-6 levels, this relationship was affirmed. The study also highlighted the need for early screening and early management to try and avoid serious neuropathic complications. Perhaps the greatest impact on the delivery of care for people with diabetes could be realized by the incorporation of clinical assessments, electrophysiological tests, and biochemical markers into practice. Though neuropathy is highly prevalent amongst diabetics, strict metabolic control, neurotrophic agents as well as lifestyle interventions should remain paramount in the management of diabetes in Uzbekistan. However, there are some gaps in the literature regarding the genetic factor, diet and lifestyle, and other complementary therapies regarding the Uzbeks. Neuropathy care should be improved by way of new drug development, regenerative therapies, and machine learning-based diagnostics in future research efforts. Enhancing partnerships between Uzbek institutions delivering health care services, and foreign research databases will be critically vital for creating proper prevention and appropriate treatment approaches for region-specific health problems**.**

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