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The Relationship between Serum Uric Acid Levels and Diabetic Peripheral Neuropathy in Type 2 Diabetes Mellitus

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Abstract: ***This cross-sectional study explored the relationship between serum uric acid (SUA) levels and diabetic peripheral neuropathy (DPN) in patients with type 2 diabetes mellitus (T2DM). The study included 70 patients with clinically confirmed DPN from a tertiary care hospital in South India. The mean age of participants was 60.4 years, with males comprising 78.6% of the population. The mean duration of diabetes was 12.81 years, and the average body mass index (BMI) was 23.90 kg/m². The Participants had mean fasting and postprandial blood sugar levels of 164.58 mg/dL and 267.43 mg/dL, respectively, while the mean HbA1c was 8.88%, reflecting the moderate glycemic control. The mean SUA level was 5.15 mg/dL, with 10% of patients exhibiting hyperuricemia. Nerve conduction studies assessed motor and sensory nerve function. Motor nerve amplitudes ranged from 3.1 to 16.1 mV, with conduction velocities between 31.6 and 51.5 m/s. Sensory nerve amplitudes were between 1.0 and 7.1 µV, and conduction velocities ranged from 11.3 to 42.0 m/s. Statistical analysis using Spearman correlation revealed no significant relationships between SUA levels and nerve conduction parameters, including motor and sensory amplitudes or velocities. Correlation coefficients ranged from -0.171 to 0.181, with all p-values exceeding 0.05. The study concludes that SUA levels do not significantly correlate with nerve conduction parameters in DPN patients, suggesting limited utility of SUA as a biomarker for nerve damage. These findings highlight the multifactorial nature of DPN, where glycemic control, oxidative stress, and vascular health play key roles. Future studies with larger populations are recommended to further investigate SUA's role in DPN and its potential therapeutic implications.***

Keywords: Diabetic neuropathy, serum uric acid, type 2 diabetes, nerve conduction, hyperuricemia

1. Introduction

Type 2 diabetes mellitus (T2DM) is associated with a range of long-term complications, including cardiovascular disease, renal failure, and nephropathy. These complications contribute significantly to the global burden of disease, as the prevalence of diabetes-related health issues has increased substantially, becoming a major public health concern. T2DM accounts for approximately 90–95% of diabetes cases, with older adults being particularly affected. Among the complications of diabetes, diabetic peripheral neuropathy (DPN) stands out as one of the most common and debilitating. DPN affects up to 50% of individuals with diabetes and is characterized by nerve damage that leads to symptoms such as pain, numbness, and loss of sensation, especially in the lower limbs. The manifestations of DPN vary depending on the involvement of peripheral sensory, motor, or autonomic nerves, resulting in a complex presentation of symptoms. Peripheral neuropathy, the most prevalent form of diabetic neuropathy, is experienced by about half of all diabetes patients at some point and is often considered one of the earliest microvascular complications of the disease. Early diagnosis and effective management of DPN are essential to prevent further complications and improve the quality of life for those affected [1,2].

Despite its significance, the global prevalence of DPN and its risk factors, particularly in low- and middle-income countries, remain insufficiently explored. Research indicates that multiple factors contribute to the development of DPN, including insulin resistance, dyslipidemia, hyperglycemia, hyperuricemia, and metabolic syndrome. A 2020 study identified serum uric acid (SUA) levels of 7.3 mg/dL or higher as being associated with an increased risk of peripheral neuropathy. High SUA levels in patients with T2DM have been correlated with a greater frequency of both macrovascular and microvascular complications. Elevated SUA is also linked to oxidative stress caused by hyperuricemia, which can lead to insulin resistance, the progression of diabetes, and cardiovascular diseases. This highlights the importance of addressing metabolic factors to reduce the risk of diabetic complications [3,4,5].

Nerve conduction velocity (NCV) testing is a widely accepted method for evaluating diabetic peripheral neuropathy. By measuring the speed of electrical impulses along peripheral nerves, NCV testing provides crucial information about nerve function. Exploring the relationship between uric acid levels and nerve conduction parameters could enhance understanding of the underlying mechanisms of DPN and offer valuable insights into its management. However, such research is scarce in certain regions, including India, emphasizing the need for more localized studies to address this gap [6].

Diabetes mellitus (DM) is one of the earliest known diseases in human history, with references dating back about 3,000 years in ancient Egyptian medical texts. It is now the most prevalent endocrine disorder, affecting over 100 million individuals worldwide, which accounts for approximately 6% of the global population. DM arises due to insufficient insulin production by the pancreas, leading to disturbances in blood glucose levels [7,8].

The condition is broadly classified into two primary types: type 1 diabetes mellitus (T1DM) and type 2 diabetes mellitus (T2DM). T1DM is characterized by a complete or near-complete lack of insulin, while T2DM involves insulin resistance coupled with inadequate insulin secretion.

Gestational diabetes is a form of diabetes that occurs during pregnancy. Additionally, there are rare types of diabetes caused by genetic defects, medications, infections, endocrinopathies, or pancreatic damage, collectively grouped under "Other Specific Types [9]."

T2DM, a chronic metabolic disorder, has shown a steadily increasing prevalence worldwide. This trend is projected to continue due to factors such as population aging and urbanization. In 2017, more than 462 million people globally were diagnosed with T2DM, representing 6.28% of the world's population. The prevalence varied across age groups, affecting 4.4% of those aged 15–49, 15% of those aged 50–69, and 22% of individuals over 70 years of age. Diabetes is the ninth leading cause of mortality worldwide, contributing to over one million deaths annually. The rising incidence of diabetes is most notable in developed regions such as Western Europe, though low- and middle-income countries are also seeing increasing cases. The highest prevalence occurs around 55 years of age, with a nearly equal gender distribution. By 2030, it is estimated that the global prevalence of T2DM will reach 7,079 cases per 100,000 individuals. These projections underscore the urgent need for effective public health and clinical strategies to address the growing diabetes epidemic and its associated complications [9-13].

This study hypothesizes that serum uric acid (SUA) levels play a role in the development of diabetic peripheral neuropathy (DPN) in individuals with type 2 diabetes mellitus (T2DM). The primary aim is to investigate the relationship between SUA levels and DPN in T2DM patients. The objectives include assessing SUA levels in T2DM patients with DPN and correlating these levels with nerve conduction study parameters, including motor conduction velocity, action potential amplitude, sensory conduction velocity, and sensory nerve action potential amplitude.

2. Material and Methods

This observational cross sectional study was conducted at the Department of General Medicine, Justice K S Hegde Charitable hospital, Constituent unit of Nitte from 01-10-2022 to 30-04-2024. Ethical approval has been obtained from the Ethical Approval Committee of Justice K S Hegde Charitable hospital, Constituent unit of Nitte.

Study Population

The study population includes 70 type 2 diabetes mellitus patients with diabetic peripheral neuropathy aged above 18 years, selected through convenience sampling. Sample size calculation was based on a correlation coefficient of 0.396 for sensory nerve conduction velocity and serum uric acid, with 95% power and 5% alpha error, using nMaster software. Exclusion criteria include inability to consent, medications altering serum uric acid, hepatic or renal dysfunction, malabsorption, malnutrition, and alcohol abuse.

Data Analysis

Data was entered into MS Excel and analyzed using SPSS version 26.0. Qualitative data was presented as frequency and percentage, while quantitative data was summarized using mean (SD) and median (IQR). Spearman correlation was applied to assess the relationship between serum uric acid levels and nerve conduction parameters, as continuous variables were not normally distributed. A p-value below 0.05 was considered statistically significant.

3. Results

The study population consisted predominantly of males (78.6%) and females (21.4%), with 41.4% of the patients having hypertension. The mean age was 60.4 years, and the average duration of diabetes was 12.81 years. The mean BMI was 23.90, while the mean FBS, PPBS, and HbA1c were 164.58 mg/dl, 267.43 mg/dl, and 8.88%, respectively. The mean serum uric acid level among the patients was 5.15 mg/dl.

Table 1: Descriptive statistics of the age and clinical parameters of patients

	Mean	Median	Std. Deviation	IQR
Age	60.40	61.00	11.10	53,68
Duration of DM (Years)	12.81	14.00	5.79	8,16.25
BMI (Kg/m²)	23.90	23.85	3.24	21.25,25.95
FBS (mg/dl)	164.57	156.00	56.03	123.75,193.25
PPBS (mg/dl)	267.43	260.00	86.09	204.25,334.25
HBA1C (%)	8.88	8.50	2.45	7.1,10.425
Uric Acid (mg/dl)	5.15	4.80	1.50	4.1,6.4

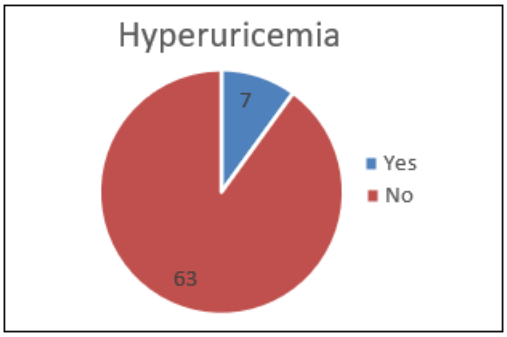

Figure 1: Distribution of hyperuricemia among patients

Among the study population, 10% of the patients were found to have hyperuricemia, while the remaining 90% did not exhibit elevated uric acid levels.

The majority of patients (52.9%) had uric acid levels in the range of 3-5 mg/dL, followed by 32.9% with levels between 5-7 mg/dL. A smaller proportion of patients had uric acid levels below 3 mg/dL (4.3%), while 10% exhibited levels exceeding 7 mg/dL, indicative of hyperuricemia.

Table 2: Descriptive statistics of the age and clinical parameters of patients with and without hyperuricemia

	Hyperuricemia Yes				Hyperuricemia No			
	Mean	Median	Std. Deviation	IQR	Mean	Median	Std. Deviation	IQR
AGE	63.29	59.00	10.81	54,75	60.08	61.00	11.17	53,68
DURATION (YEARS)	15.57	15.00	2.07	14,16	12.51	12.00	6.00	7,17
BMI (Kg/m²)	26.14	25.70	3.28	22.5,30.1	23.65	23.50	3.17	20.9,25.6
FBS (mg/dl)	176.14	156.00	84.47	124,206	163.29	156.00	52.79	123,190
PPBS (mg/dl)	277.86	289.00	122.60	199,318	266.27	256.00	82.35	208,344
HBA1C (%)	9.40	8.60	4.32	6.6,11	8.82	8.40	2.21	7.2,10.1

Patients with hyperuricemia had a higher mean age (63.29 years) compared to those without hyperuricemia (60.08 years). The mean duration of diabetes was also longer in the hyperuricemia group (15.57 years) than in those without hyperuricemia (12.51 years). Similarly, the mean BMI was elevated in patients with hyperuricemia (26.14 kg/m²) compared to those without (23.65 kg/m²). Fasting blood sugar (FBS) levels were slightly higher in the hyperuricemia group (176.14 mg/dL) compared to the non-hyperuricemia group (163.29 mg/dL), and postprandial blood sugar (PPBS) levels followed a similar trend, with means of 277.86 mg/dL and 266.27 mg/dL, respectively. Mean HbA1c levels were also higher in the hyperuricemia group (9.4%) compared to those without hyperuricemia (8.82%), reflecting poorer glycemic control among patients with elevated uric acid levels.

Table 3: Descriptive statistics of the amplitude and velocity of various motor and sensory nerves

	Mean	Median	SD	IQR
MOTOR RIGHT MEDIAN AMPLITUDE (mv)	14.9	15.7	5.5	10.575,18.525
MOTOR RIGHT MEDIAN CONDUCTION VELOCITY (m/s)	45.5	44.6	5.0	43,48.5
MOTOR LEFT MEDIAN AMPLITUDE (mv)	16.1	17.1	5.6	12.025,21.125
MOTOR LEFT MEDIAN CONDUCTION VELOCITY (m/s)	46.9	46.6	4.9	43.7,51.175
MOTOR RIGHT ULNAR AMPLITUDE (mv)	12.5	12.4	4.1	10.1,14.525
MOTOR RIGHT ULNAR CONDUCTION VELOCITY (m/s)	51.5	47.4	35.0	44.2,51.5
MOTOR LEFT ULNAR AMPLITUDE (mv)	13.3	13.4	5.6	9.725,16.9
MOTOR LEFT ULNAR CONDUCTION VELOCITY (m/s)	47.0	47.4	6.8	44.05,51.7
MOTOR RIGHT PERONEAL AMPLITUDE (mv)	3.1	2.7	3.3	0.75,4.05
MOTOR RIGHT PERONEAL CONDUCTION VELOCITY (m/s)	31.6	35.8	12.9	31.275,38.625
MOTOR LEFT PERONEAL AMPLITUDE (mv)	3.6	2.6	3.9	0.9,5.3
MOTOR LEFT PERONEAL CONDUCTION VELOCITY (m/s)	33.5	36.2	10.4	32.2,38.6
MOTOR RIGHT TIBIAL AMPLITUDE (mv)	4.0	4.2	3.4	0.875,5.6
MOTOR RIGHT TIBIAL CONDUCTION VELOCITY (m/s)	31.6	34.4	12.9	30.6,38.6
MOTOR LEFT TIBIAL AMPLITUDE (mv)	4.7	4.6	4.2	1,6.8
MOTOR LEFT TIBIAL CONDUCTION VELOCITY (m/s)	32.0	33.7	12.1	30.05,38.625
SENSORY RIGHT MEDIAN AMPLITUDE (µv)	6.7	6.3	5.0	2.875,10.425
SENSORY RIGHT MEDIAN CONDUCTION VELOCITY (m/s)	40.3	46.4	16.8	38.175,50.1
SENSORY LEFT MEDIAN AMPLITUDE (µv)	7.1	7.0	5.4	3.45,9.2
SENSORY LEFT MEDIAN CONDUCTION VELOCITY (m/s)	41.1	44.0	14.2	38.8,48.6
SENSORY RIGHT ULNAR AMPLITUDE (µv)	5.7	4.8	3.9	2.7,8.4
SENSORY RIGHT ULNAR CONDUCTION VELOCITY (m/s)	42.0	46.4	16.0	42,51.2
SENSORY LEFT ULNAR AMPLITUDE (µv)	5.1	4.5	4.1	2.6,7
SENSORY LEFT ULNAR CONDUCTION VELOCITY (m/s)	37.9	43.0	18.2	38.675,48.2
SENSORY RIGHT SURAL AMPLITUDE (µv)	3.1	0.0	4.1	0,5.225
SENSORY RIGHT SURAL CONDUCTION VELOCITY (m/s)	20.2	0.0	22.6	0,42.825
SENSORY LEFT SURAL AMPLITUDE (µv)	2.6	0.0	3.7	0,4.8
SENSORY LEFT SURAL CONDUCTION VELOCITY (m/s)	21.5	0.0	23.8	0,47.325
SENSORY RIGHT SUPERFICIAL AMPLITUDE (µv)	1.1	0.0	2.1	0,1.925
SENSORY RIGHT SUPERFICIAL CONDUCTION VELOCITY (m/s)	12.8	0.0	21.1	0,41.475
SENSORY LEFT SUPERFICIAL AMPLITUDE (µv)	1.0	0.0	1.8	0,2.25
SENSORY LEFT SUPERFICIAL CONDUCTION VELOCITY (m/s)	11.3	0.0	19.9	0,12.35

In patients with diabetes and peripheral neuropathy, motor and sensory nerve conduction studies revealed varied results across different nerves. Motor nerve amplitudes ranged from 3.1 to 16.1 mV, with conduction velocities between 31.6 and 51.5 m/s. Sensory nerve amplitudes were between 1.0 and 7.1 µV, while conduction velocities ranged from 11.3 to 42.0 m/s. Despite these variations, no significant correlation was observed between nerve conduction parameters and serum uric acid levels, indicating that uric acid may not be a reliable marker for nerve damage in this context.

Table 4: Correlation between uric acid and the nerve conduction parameters

	Rho (correlation coefficient	P value
MOTOR RIGHT MEDIAN AMPLITUDE (mV)	0.129	0.288
MOTOR RIGHT MEDIAN CONDUCTION VELOCITY (m/s)	-0.081	0.506
MOTOR LEFT MEDIAN AMPLITUDE (mV)	0.161	0.183
MOTOR LEFT MEDIAN CONDUCTION VELOCITY (m/s)	0.037	0.763
MOTOR RIGHT ULNAR AMPLITUDE (mV)	0.139	0.253
MOTOR RIGHT ULNAR CONDUCTION VELOCITY (m/s)	-0.058	0.631
MOTOR LEFT ULNAR AMPLITUDE (mV)	0.181	0.133
MOTOR LEFT ULNAR CONDUCTION VELOCITY (m/s)	0.036	0.768
MOTOR RIGHT PERONEAL AMPLITUDE (mV)	0.065	0.594
MOTOR RIGHT PERONEAL CONDUCTION VELOCITY (m/s)	-0.017	0.888
MOTOR LEFT PERONEAL AMPLITUDE (mV)	0.092	0.447
MOTOR LEFT PERONEAL CONDUCTION VELOCITY (m/s)	-0.171	0.157
MOTOR RIGHT TIBIAL AMPLITUDE (mV)	0.007	0.955
MOTOR RIGHT TIBIAL CONDUCTION VELOCITY (m/s)	0.012	0.924
MOTOR LEFT TIBIAL AMPLITUDE (mV)	-0.003	0.983
MOTOR LEFT TIBIAL CONDUCTION VELOCITY (m/s)	-0.006	0.962
SENSORY RIGHT MEDIAN AMPLITUDE (µV)	-0.042	0.731
SENSORY RIGHT MEDIAN CONDUCTION VELOCITY (m/s)	0.056	0.648
SENSORY LEFT MEDIAN AMPLITUDE (µV)	0.071	0.556
SENSORY LEFT MEDIAN CONDUCTION VELOCITY (m/s)	0.096	0.428
SENSORY RIGHT ULNAR AMPLITUDE (µV)	-0.065	0.594
SENSORY RIGHT ULNAR CONDUCTION VELOCITY (m/s)	0.034	0.780
SENSORY LEFT ULNAR AMPLITUDE (µV)	-0.082	0.497
SENSORY LEFT ULNAR CONDUCTION VELOCITY (m/s)	-0.005	0.969
SENSORY RIGHT SURAL AMPLITUDE (µV)	-0.013	0.916
SENSORY RIGHT SURAL CONDUCTION VELOCITY (m/s)	-0.109	0.370
SENSORY LEFT SURAL AMPLITUDE (µV)	-0.087	0.476
SENSORY LEFT SURAL CONDUCTION VELOCITY (m/s)	-0.014	0.906
SENSORY RIGHT SUPERFICIAL AMPLITUDE (µV)	0.128	0.290
SENSORY RIGHT SUPERFICIAL CONDUCTION VELOCITY (m/s)	0.100	0.412
SENSORY LEFT SUPERFICIAL AMPLITUDE (µV)	0.109	0.370
SENSORY LEFT SUPERFICIAL CONDUCTION VELOCITY (m/s)	0.084	0.487

Among diabetic patients with peripheral neuropathy, there was no significant correlation between serum uric acid levels and various motor and sensory nerve conduction parameters, including amplitudes and velocities across multiple nerves. Correlation coefficients for all measured parameters ranged from -0.171 to 0.181, with p-values consistently above 0.05, indicating a lack of statistically meaningful associations. This suggests that uric acid levels may not directly influence nerve conduction characteristics in this population.

4. Discussion

Diabetic peripheral neuropathy (DPN) is a common and debilitating microvascular complication of type 2 diabetes mellitus (T2DM), affecting a significant proportion of individuals with long-standing diabetes. It manifests primarily as distal symmetrical sensory polyneuropathy and can lead to reduced sensation, motor dysfunction, and severe disability, particularly in the lower limbs. Approximately 50% of individuals with T2DM who have had the disease for more than ten years develop DPN, making it one of the most prevalent complications. This condition can significantly impair quality of life and is associated with an increased risk of falls, ulcers, and amputations. The underlying pathophysiology of DPN involves a combination of factors, including hyperglycemia, oxidative stress, inflammation, and impaired nerve blood supply. Among these, serum uric acid (SUA) levels have garnered attention due to their potential role in oxidative stress, a key contributor to nerve damage in DPN [14,15].

This study was conducted to investigate the relationship between SUA levels and DPN in T2DM patients attending a tertiary care hospital in South India. The objective was to assess the serum uric acid levels in these patients and determine if there was any correlation between SUA and nerve conduction study (NCS) parameters, which include sensory and motor nerve conduction velocities (CV) and amplitudes. A total of 70 patients with T2DM and diagnosed DPN were included in the study. The patients' clinical parameters, including the duration of diabetes, blood pressure, body mass index (BMI), HbA1c levels, and serum uric acid levels, were measured, and NCS was performed to evaluate sensory and motor nerve function. The study aimed to assess the role of SUA in DPN and examine whether elevated levels of SUA were correlated with alterations in nerve conduction parameters, which are commonly used to assess the severity of neuropathy [16,17].

The study population had a mean age of 60.4 years, aligning with similar findings in studies conducted globally, where the mean ages typically ranged from 59 to 60 years. The gender distribution showed that 78.6% of participants were male, a higher proportion compared to other studies, which often reported male prevalence closer to 60%. This higher prevalence among men might reflect variations in lifestyle, genetic predisposition, or disease progression. The mean duration of diabetes in this cohort was 12.81 years, comparable to findings in similar research, which reported durations around 11 to 12 years. The mean BMI of 23.90 was consistent with previous findings indicating BMI values

in the range of 23 to 24, suggesting that participants generally had normal BMI levels, minimizing the potential influence of obesity-related factors on the development of diabetic peripheral neuropathy [18,19].

The mean HbA1c level in the study was 8.88%, indicating moderate glycemic control. Poor glycemic control is a well-established risk factor for the development and progression of diabetic peripheral neuropathy (DPN), as prolonged hyperglycemia exacerbates oxidative stress and accelerates nerve damage. This highlights the critical importance of effective blood glucose management in preventing DPN. The mean serum uric acid (SUA) level in the study group was 5.15 mg/dL, consistent with findings in other populations. Variations in SUA levels observed in different studies may be attributed to differences in study populations or diagnostic criteria [20].

The role of serum uric acid (SUA) in diabetic peripheral neuropathy (DPN) remains a topic of debate. Elevated SUA levels have been implicated in increased oxidative stress, which contributes to neuronal injury and the progression of DPN. While uric acid functions as an antioxidant, offering protection against oxidative stress, its excessive levels can act as pro-inflammatory agents, potentially causing vascular dysfunction—a critical factor in DPN. In this study, the mean SUA levels in DPN patients were marginally higher than in the general population. However, the correlation between SUA levels and nerve conduction study parameters was weak or non-significant, suggesting limited utility of SUA as a reliable biomarker for DPN in this context. This finding aligns with observations from other research, which also reported no significant relationship between SUA levels and nerve conduction velocities in patients with type 2 diabetes mellitus and DPN. These results underline the complex and dualistic role of SUA, emphasizing the need for further investigation to clarify its contribution to DPN development [21, 20].

In evaluating sensory nerve conduction velocities, this study observed that the mean conduction velocities of the ulnar, median, and sural nerves were lower compared to previously reported values in similar populations. However, no significant correlation was found between sensory nerve conduction velocities and serum uric acid (SUA) levels. While some studies have suggested a weak positive correlation between SUA and specific nerve conduction velocities, such as in the median nerve, the evidence remains inconclusive. This lack of a strong or consistent relationship indicates that elevated SUA levels may have limited or variable influence on sensory nerve conduction parameters, necessitating further research to better understand its role in diabetic peripheral neuropathy [20, 22].

Motor nerve conduction studies in the ulnar, median, tibial, and peroneal nerves revealed no significant correlations between conduction velocities and serum uric acid (SUA) levels. These findings align with prior research indicating a lack of robust association between SUA levels and motor nerve conduction parameters in diabetic peripheral neuropathy (DPN). While some studies have reported weak positive or negative correlations between SUA and motor nerve conduction velocities, such associations have been inconsistent and inconclusive, underscoring the need for further investigation into the potential role of SUA in motor nerve function within DPN [23, 20].

This study found no significant correlations between sensory or motor nerve amplitudes and serum uric acid (SUA) levels. While some research has suggested a negative or positive correlation between nerve conduction amplitudes and SUA, the findings remain inconsistent. These variations highlight the complexity of the relationship between SUA and nerve conduction parameters, suggesting that the impact of SUA on diabetic peripheral neuropathy (DPN) may differ based on nerve type, patient characteristics, or disease progression [20, 24].

While our study did not find significant correlations between serum uric acid levels and nerve conduction parameters in patients with diabetic peripheral neuropathy, the findings contribute to the ongoing debate regarding the role of SUA in DPN. Oxidative stress, which is influenced by SUA, is likely a factor in DPN progression, but it may not be the sole contributor. Other factors, including glycemic control, duration of diabetes, and vascular health, play a more prominent role in DPN development. Future research with larger, more diverse cohorts and longitudinal studies is needed to better understand the complex interplay between SUA, oxidative stress, and DPN, and to explore potential therapeutic strategies targeting oxidative stress or SUA modulation in preventing or treating DPN [25].

5. Conclusion

In this study, type 2 diabetes patients with peripheral neuropathy had a mean serum uric acid level of 5.15 mg/dL, with 10% of patients exhibiting hyperuricemia. The motor nerve amplitude ranged from 3.1 to 16.1 mV, and the conduction velocity varied between 31.6 to 51.5 m/s. For sensory nerves, the amplitude ranged from 1 to 7.1 mV, and the conduction velocity was between 11.3 to 41.1 m/s. However, no significant correlation was found between nerve conduction parameters and serum uric acid levels in this cohort. Consequently, SUA appears to have a limited role as a marker for nerve damage.

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