ORIGINAL ARTICLE

Divalent Cation Imbalance: A Risk Factor for Diabetic Neuropathy in Type 2 Diabetes Mellitus

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Abstract

Background: Diabetic neuropathy is a common and disabling microvascular complication of type 2 diabetes mellitus (T2DM). Alterations in magnesium and calcium homeostasis have been implicated in neuronal dysfunction and may contribute to the development of neuropathic complications. The present study evaluated the association of serum magnesium and calcium levels with diabetic neuropathy in patients with T2DM.

Methods: This comparative cross-sectional study included 80 patients with T2DM aged 30–60 years. Forty patients with mild diabetic neuropathy were compared with 40 age-matched T2DM patients without neuropathy. Neuropathy was assessed clinically and by vibration perception threshold using a biothesiometer. Fasting blood sugar (FBS), postprandial blood sugar (PPBS), glycated hemoglobin (HbA1c), serum magnesium, and serum calcium levels were measured. Statistical analysis was performed using the Mann–Whitney U test.

Results: Patients with diabetic neuropathy had significantly lower serum magnesium levels than those without neuropathy (1.63 ± 0.37 vs. 1.84 ± 0.55 mg/dL; $p = 0.046$). Serum calcium concentrations were also reduced in the neuropathy group (7.89 ± 0.87 vs. 8.49 ± 0.89 mg/dL; $p = 0.001$). HbA1c levels were higher among patients with neuropathy ($9.86 \pm 2.60\%$ vs. $8.72 \pm 2.35\%$; $p = 0.019$), indicating poorer long-term glycemic control. No significant differences were observed in fasting blood glucose, postprandial blood glucose, body mass index, or waist circumference between the groups.

Conclusion: Lower serum magnesium and calcium levels were associated with diabetic neuropathy in patients with type 2 diabetes mellitus. Combined with poor glycemic control, disturbances in divalent cation homeostasis may contribute to neuropathic progression. Routine assessment of these biochemical parameters could aid in identifying patients at increased risk of diabetic neuropathy and facilitate early preventive interventions.

Keywords: Type 2 diabetes mellitus; diabetic neuropathy; magnesium; calcium; HbA1c.

1. Introduction

Type 2 diabetes mellitus (T2DM) is a major public health concern worldwide, with a steadily increasing prevalence and substantial socioeconomic burden.[1,2] According to the World Health Organization, approximately 830 million individuals are living with diabetes, most of whom reside in low- and middle-income countries.[3] In the United States, approximately 40 million people (or 12% of the population) have diabetes, and over 115 million adults have prediabetes, highlighting the huge burden of glucose metabolism disorders.[4] Chronic hyperglycemia associated with T2DM affects multiple organ systems and contributes to the development of several long-term complications.[5]

About 50% of diabetic patients have diabetic neuropathy (DN), one of the most prevalent and debilitating microvascular consequences.[6,7] According to the Centres for Disease Control and Prevention (CDC), peripheral neuropathy can affect up to 50% of individuals with long-term diabetes.[8] Diabetic neuropathy can cause sensory loss, neuropathic pain, foot ulcerations and amputations, significantly impairing quality of life and increasing healthcare burden.[9]

The pathogenesis of diabetic neuropathy is multifactorial and involves chronic hyperglycemia, oxidative stress, mitochondrial dysfunction, inflammation and neuronal damage.[10] Chronic hyperglycemia promotes oxidative stress through pathways such as AGE formation and polyol pathway activation, leading to endothelial dysfunction and nerve injury.

Recent research has highlighted the role of divalent cations, particularly magnesium and calcium, in glucose metabolism, insulin signalling, neuronal excitability and intracellular signalling pathways.[11] Emerging data suggests that ion homeostasis changes may play a significant role in the development of diabetic complications by influencing oxidative stress pathways, mitochondrial activity, and inflammatory responses.[12]

Magnesium serves as a cofactor for several enzymes involved in carbohydrate metabolism and neuronal stability, whereas calcium is essential for neurotransmitter release and neuronal function.[13] Both ions are essential for maintenance of membrane integrity, synaptic transmission, cellular energy metabolism and neuronal survival.

Hypomagnesemia is frequently observed in patients with T2DM and has been associated with poor glycemic control, insulin resistance, and diabetic complications including neuropathy. [14] Recent clinical studies have additionally demonstrated associations between low magnesium levels and impaired nerve conduction, endothelial dysfunction, and increased inflammatory activity in diabetic patients. [15]

Similarly, calcium dysregulation has been implicated in neuronal apoptosis, mitochondrial dysfunction, and neurodegenerative changes associated with diabetic neuropathy. [16] Abnormal intracellular calcium signaling may contribute to mitochondrial calcium overload, activation of apoptotic pathways, oxidative neuronal injury, and impaired neuronal repair mechanisms.

Despite growing evidence linking mineral imbalance with diabetic complications, limited studies have evaluated the combined role of magnesium and calcium as potential biochemical markers for diabetic neuropathy in patients with T2DM, particularly in the Indian population. Since serum magnesium and calcium estimation are inexpensive, minimally invasive, and routinely available laboratory investigations, they may provide additional value in identifying patients at higher risk for neuropathy before irreversible nerve damage develops. Therefore, the present study was undertaken to assess the association between serum magnesium and calcium levels and diabetic neuropathy in patients with T2DM.

2. Materials and Methods

2.1 Study Design and Setting

The present cross-sectional observational study was conducted in the Department of Biochemistry in collaboration with the Department of General Medicine, Mahatma Gandhi Medical College and Research Institute (MGMCRI), Puducherry. The study was carried out after obtaining approval from the Institutional Human Ethics Committee, and written informed consent was obtained from all participants prior to enrolment.

Patients with chronic kidney disease, liver disease, alcoholism, thyroid disorders, vitamin B12 deficiency, peripheral vascular disease, neurological disorders, other specific types of diabetes mellitus, and individuals receiving magnesium or calcium supplementation were excluded from the study. Relevant clinical history, treatment history, and personal history were documented for all study participants.

2.2 Study Population

A total of 80 subjects with type 2 diabetes mellitus (T2DM) were included in the study after obtaining written informed consent. The study population was divided into two groups consisting of 40 subjects each: Group I included T2DM patients without diabetic neuropathy and Group II included T2DM patients with mild diabetic neuropathy.

Patients with chronic kidney disease, liver disease, alcoholism, thyroid disorders, vitamin B12 deficiency, peripheral vascular disease, neurological disorders, other specific types of diabetes mellitus, and individuals receiving magnesium or calcium supplementation were excluded from the study. Relevant clinical history, treatment history, and personal history were documented for all study participants.

2.3 Assessment of Diabetic Neuropathy

All diabetic cases fulfilling the inclusion criteria were subjected to detailed clinical evaluation for diabetic peripheral neuropathy. Neuropathy assessment included examination for loss of fine touch, crude touch, pain and temperature sensation, ankle reflex, pressure sensation, vibration perception, and ankle-brachial ratio. Vibration perception threshold (VPT) was assessed using a biothesiometer with a vibration output range of 0–50 volts. The threshold of vibration perception was determined by gradually increasing the vibration stimulus until it was perceived by the participant. Based on VPT values, diabetic neuropathy was graded as follows:

- Normal: ≤15 volts
- Mild loss of vibratory perception: 16–20 volts
- Moderate loss of vibratory perception: 21–25 volts
- Severe loss of vibratory perception: >25 volts

Patients with VPT values between 16–20 volts were categorized as having mild diabetic neuropathy and were included in the neuropathy group.

2.4 Sample Collection and Biochemical Analysis

After overnight fasting, 5 mL of venous blood was collected under aseptic precautions from all study participants. Blood samples were analysed for fasting blood glucose, HbA1c, serum magnesium, and serum calcium levels.

Fasting blood glucose was estimated by the glucose oxidase-peroxidase (GOD-POD) method. HbA1c was measured using high-performance liquid chromatography (HPLC). We measured serum magnesium using the xylidyl blue colorimetric method, while serum calcium was measured using the o-cresolphthalein complexone method.

2.5 Statistical Analysis

Data were entered into Microsoft Excel and analysed using SPSS software version 19 and JASP version 9. Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were expressed as frequencies and percentages. Normality of data distribution was assessed using the Shapiro–Wilk test. Student's t-test was used for comparison of normally distributed continuous variables, whereas the Mann–Whitney U test was applied for non-normally distributed variables. Categorical variables were compared using the Chi-square test. Correlation between biochemical parameters and glycemic status was assessed using Spearman correlation analysis. A p-value of ≤ 0.05 was considered statistically significant.

2.6 Data Safety and Confidentiality

Participant data were kept strictly confidential and used only for research purposes. Personal identifiers were excluded from data analysis and reporting, and all records were securely maintained under institutional ethical guidelines.

Results

The present study compared anthropometric and biochemical parameters between T2DM patients without neuropathy and T2DM patients with diabetic neuropathy. The mean BMI and waist circumference did not show statistically significant differences between the two groups ($p>0.05$).

HbA1c levels were significantly elevated in patients with diabetic neuropathy compared to T2DM patients without neuropathy (9.86 ± 2.60 vs 8.72 ± 2.35; $p=0.019$), indicating poorer glycemic control among neuropathy patients. Although fasting blood sugar (FBS) and postprandial blood sugar (PPBS) levels were higher in the neuropathy group, the differences were not statistically significant ($p>0.05$).

Serum magnesium levels were significantly lower in patients with diabetic neuropathy compared to T2DM patients without neuropathy (1.63 ± 0.37 mg/dL vs 1.84 ± 0.55 mg/dL; $p=0.046$). Similarly, serum calcium levels were significantly reduced in the neuropathy group compared to the T2DM group without neuropathy (7.89 ± 0.87 mg/dL vs 8.49 ± 0.89 mg/dL; $p=0.001$).

These findings suggest that poor glycemic control together with reduced serum magnesium and calcium levels may be associated with the development and progression of diabetic neuropathy in patients with type 2 diabetes mellitus.

Table 1: Comparison of anthropometric and biochemical parameters between T2DM patients without neuropathy and with diabetic neuropathy.

Parameters	Study Groups	N	Mean	Std. Deviation	Median	25th percentile	75th percentile	Mann-Whitney test	p-value
BMI	T2DM	40	25.46	4.82	25.85	22.10	28.13	783	0.874
	Neuropathy	40	25.74	3.87	24.90	23.00	27.50		
Waist Circumference	T2DM	40	60.65	11.04	61.00	55.00	68.00	-1.461	0.148t
	Neuropathy	40	63.93	8.89	63.00	59.50	70.00		
HbA1c	T2DM	40	8.72	2.35	7.85	6.70	10.80	501	0.019*
	Neuropathy	40	9.86	2.60	10.40	8.95	11.80		
FBS	T2DM	40	190.05	63.21	172.00	145.25	220.25	692	0.301
	Neuropathy	40	200.75	56.60	196.50	154.50	222.50		
PPBS	T2DM	40	241.33	97.12	228.50	180.00	283.00	733	0.522
	Neuropathy	40	250.73	92.64	249.50	167.75	318.50		
Magnesium	T2DM	40	**1.84**	**0.55**	1.70	1.50	1.93	1007.5	0.046*
	Neuropathy	40	**1.63**	**0.37**	1.50	1.38	1.65		
Calcium	T2DM	40	**8.49**	**0.89**	8.70	8.18	9.13	1136.5	0.001*
	Neuropathy	40	**7.89**	**0.87**	7.90	7.20	8.40		

Table 1 represents the comparison between groups was performed using the Mann–Whitney U test.
*p value ≤0.05 was considered statistically significant.
BMI body mass index, HbA1c glycated haemoglobin, FBS fasting blood sugar, PPBS postprandial blood sugar

Figure 1: Comparison of HbA1c levels between T2DM patients without neuropathy and with diabetic neuropathy

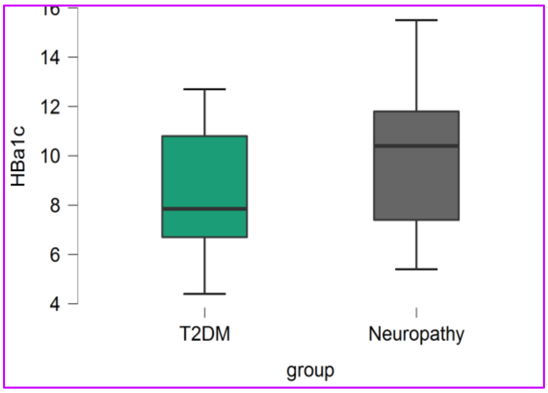

Fig. 1 shows significantly higher HbA1c levels in patients with diabetic neuropathy compared to T2DM patients without neuropathy (p = 0.019), indicating poorer long-term glycemic control in the neuropathy group.

Figure 2: Comparison of serum magnesium levels between T2DM patients without neuropathy and with diabetic neuropathy

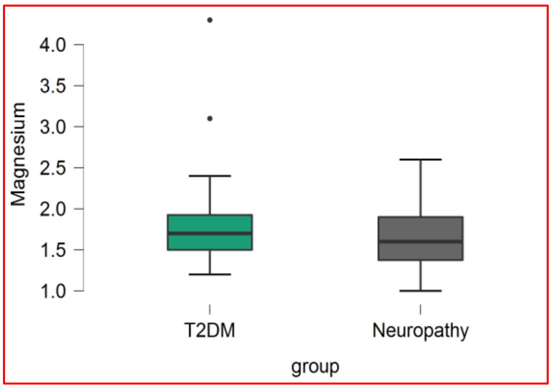

Fig. 2 shows significantly lower serum magnesium levels in patients with diabetic neuropathy compared to T2DM patients without neuropathy (p = 0.046), indicating a possible association between hypomagnesemia and diabetic neuropathy.

Figure 3: Comparison of serum calcium levels between T2DM patients without neuropathy and with diabetic neuropathy

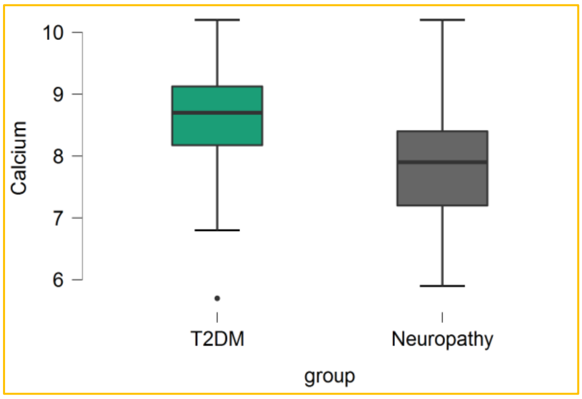

Fig. 3 shows significantly lower serum calcium levels in patients with diabetic neuropathy compared to T2DM patients without neuropathy (p = 0.001), suggesting a potential role of calcium imbalance in the development of diabetic neuropathy.

Discussion

Diabetic neuropathy is one of the most frequent microvascular complications of type 2 diabetes mellitus and is associated with significant morbidity and reduced quality of life. [7,17] Persistent hyperglycemia activates multiple metabolic pathways including advanced glycation end-product formation, polyol pathway activation, and excessive production of reactive oxygen species, ultimately resulting in neuronal degeneration and impaired nerve conduction.[18] In the present study, patients with diabetic neuropathy exhibited lower serum magnesium and calcium levels and higher HbA1c values compared with those without neuropathy. These findings suggest that disturbances in divalent cation homeostasis, together with poor glycemic control, may contribute to the development and progression of neuropathic complications.

Higher HbA1c levels observed among neuropathy patients indicate the importance of long-term glycemic burden in the pathogenesis of diabetic neuropathy. Chronic hyperglycemia promotes oxidative stress, endothelial dysfunction, inflammation, and impaired nerve perfusion, resulting in progressive neuronal injury. [19] Although fasting and postprandial blood glucose concentrations were higher in the neuropathy group, the differences were not statistically significant. This observation suggests that HbA1c, which reflects prolonged glycemic exposure, may be a better indicator of neuropathy risk than single measurements of blood glucose.

Magnesium is an essential intracellular cation involved in glucose metabolism, insulin signaling, ATP synthesis, membrane stabilization, and neuronal excitability.[20] Hypomagnesemia is frequently observed in T2DM patients and has been associated with insulin resistance, poor glycemic control, oxidative stress and microvascular complications. [21] Comparison between groups showed significantly lower serum magnesium concentrations in patients with diabetic neuropathy. Similar observations have been described in previous studies demonstrating a relationship between hypomagnesemia and impaired peripheral nerve function.[20]Our results indicate that magnesium deficiency may contribute to neuropathic changes by enhancing oxidative stress and disrupting intracellular signaling pathways.

Experimental evidence has additionally shown that reduced magnesium levels may facilitate excessive calcium influx through NMDA receptor activation, resulting in mitochondrial dysfunction, excitotoxic neuronal damage, and activation of apoptotic pathways.[22] Recent studies have also reported associations between hypomagnesemia and impaired nerve conduction velocity, endothelial dysfunction, and small fiber nerve injury in diabetic patients.[23]

Neuropathy patients had significantly lower serum calcium levels compared with T2DM patients without neuropathy. Calcium is essential for neurotransmitter release, axonal transport, and preservation of neuronal integrity.[24] Disturbances in calcium homeostasis have been linked to mitochondrial dysfunction, activation of apoptotic pathways, and impaired neuronal regeneration.[25] Experimental studies suggest that chronic hyperglycemia induces intracellular calcium overload, which may accelerate oxidative stress and neurodegenerative changes.[26] Our findings support the possibility that altered calcium signaling represents an additional mechanism involved in diabetic nerve damage

Taken together, our results indicate that disturbances in magnesium and calcium homeostasis, in conjunction with chronic hyperglycemia, may contribute to the progression of diabetic neuropathy. The findings of our study highlight the potential value of serum magnesium and calcium as simple, inexpensive, and readily available biomarkers for identifying patients with type 2 diabetes mellitus who are at increased risk of neuropathic complications.[27] Since these biochemical parameters are routinely measured in clinical laboratories, their assessment may facilitate early recognition of mineral imbalance and timely intervention.

Previous studies have shown that correction of hypomagnesemia through dietary modification and magnesium supplementation can improve glycemic control, insulin sensitivity, and metabolic stability in diabetic patients.[28] Therefore, nutritional interventions aimed at restoring mineral homeostasis, together with strict glycemic control, may help reduce oxidative stress and delay the progression of neuropathic complications.

Overall, our results indicate that reduced serum magnesium and calcium levels are associated with diabetic neuropathy and support the potential utility of these readily available biochemical parameters for early risk assessment and preventive management in patients with type 2 diabetes mellitus.

Limitations of the Study

Several limitations should be considered while interpreting these findings. The sample size was relatively small, and the cross-sectional study design limits causal interpretation. Neuropathy assessment was primarily based on clinical examination and vibration perception threshold analysis, while advanced electrophysiological studies were not performed. In addition, intracellular magnesium and calcium status was not evaluated. Further large-scale longitudinal studies incorporating nerve conduction studies, inflammatory markers, oxidative stress parameters, and molecular biomarkers are warranted to better understand the mechanistic role of divalent cations in diabetic neuropathy.

Important findings that emerged from the present study include:

1. Serum magnesium and calcium levels were significantly reduced in T2DM patients with diabetic neuropathy.
2. Elevated HbA1c levels were strongly associated with neuropathy, indicating the role of poor glycemic control in neuropathy progression.
3. Magnesium and calcium imbalance may serve as potential biochemical markers for early identification of diabetic neuropathy.
4. Early correction of mineral imbalance along with strict glycemic control may help delay or prevent progression of diabetic neuropathy.

Conclusion

Comparison between groups showed lower serum magnesium and calcium concentrations in patients with diabetic neuropathy than in diabetic patients without neuropathy. Neuropathy patients also had higher HbA1c levels, underscoring the importance of long-term glycemic control in the development and progression of nerve damage. Our results indicate that disturbances in divalent cation homeostasis may contribute to neuronal dysfunction and the pathogenesis of diabetic neuropathy in type 2 diabetes mellitus.

Given that serum magnesium and calcium estimation is inexpensive and routinely available, these parameters may serve as useful adjuncts for identifying individuals at increased risk of neuropathic complications and facilitating early preventive measures. Larger prospective studies incorporating electrophysiological and molecular markers are needed to better define the prognostic and therapeutic relevance of these biomarkers in diabetic neuropathy.

Conflict of interest: The authors declare that there are no conflicts of interest related to this study. No financial, personal, or professional relationships have influenced the design, conduct, analysis, or reporting of the research.

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