

Association of Serum Magnesium and Distal Symmetric Peripheral Neuropathy Among Filipino Patients with Type 2 Diabetes Mellitus

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Abstract

Objective: This study aims to determine the association of serum magnesium with distal symmetric peripheral neuropathy among persons with type 2 diabetes mellitus (DM).

Methodology: A cross-sectional analytical study among adult Filipinos with Type 2 DM. Logistic regression was used to determine the association of serum magnesium with DSPN diagnosed by the Michigan Neuropathy Screening Instrument. The null hypothesis was rejected at 0.05 α -level of significance.

Results: The average serum magnesium levels were similar between those with versus without DSPN (2.06 ± 0.32 vs 2.05 ± 0.23 , $p = 0.873$); the same was seen for corrected magnesium. There is insufficient evidence to demonstrate a significant statistical difference between those with and without DSPN in relation to glycemic control (HbA1c and FBS). Likewise, there is no significant statistical correlation between serum magnesium levels with HbA1c, FBS, BMI, or duration of diabetes.

Conclusion: This present study could not demonstrate any association between DSPN and serum magnesium, even after adjusting for age, sex, and comorbidity.

Keywords: Magnesium, Distal Symmetric Peripheral Neuropathy, Diabetic Neuropathy

Introduction

Diabetic neuropathies are considered the most common complications of both Types 1 and 2 Diabetes Mellitus (DM). Among its forms, distal symmetric polyneuropathy (DSPN) is highly morbid and is the most diagnosed. DSPN is diagnosed by the presence of a combination of symptoms and signs of neuropathy using the Michigan Neuropathy Screening Instrument (MNSI). Patients are considered positive using cut-off scores of ≥ 4 on the questionnaire and ≥ 1 on the clinical examination.

There is limited data on its prevalence in Southeast Asia, which is believed to be underestimated. In 2000, the Diabcare-Asia project evaluated 2,708 patients in Philippine diabetes care centers and reported a 42% prevalence of DSPN based on medical records. A more recent study was done at the Philippine General Hospital based on abnormality in nerve conduction studies, including 150 patients with diabetes, and showed a 58% DSPN prevalence.³

DSPN limits a patient's functional capacity, especially when associated with pain, leading to poor quality of life. This is associated with a 15-fold increase in falls and fractures, particularly in older patients. Foot ulceration is one of the most serious adverse effects of DSPN and is considered a strong predictor of early mortality. In the most severe cases, amputation may be necessary. Worldwide, it accounts for more hospitalizations than all other complications combined. Due to delay in diagnosis, the hospitalization cost has not decreased and causes a great socioeconomic burden to the patient.¹

Magnesium is the fourth most abundant cation in the human body. It is a co-factor for numerous enzymatic reactions and plays important roles in many biological processes. It regulates the vasomotor tone through the energy-dependent transport of calcium and potassium across cell membranes. Hypomagnesemia is defined as a serum magnesium level below 1.6 mg/dl with a correction factor for albumin level at $+ 0.005 (4 - \text{serum albumin})$

Aberrations in this finely-controlled mechanism may lead to microvascular complications, including peripheral neuropathy.⁵ Recent studies also demonstrate the

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Sample Size for Frequency in a Population	
Population size (for finite population correction factor or fpc)(N):	1000000
Hypothesized % frequency of outcome factor in the population (p):	40.3% +/-10
Confidence limits as % of 100 (absolute +/- %)(d):	10%
Design effect (for cluster surveys-DEFF):	1
Sample Size(n) for Various Confidence Levels	
Confidence Level(%)	Sample Size
95%	93
80%	40
90%	66
97%	114
99%	160
99.9%	261
99.99%	365
Equation	
Sample size $n = [DEFF * Np(1-p)] / [(d^2 / Z^2_{1-\alpha/2} * (N-1) + p * (1-p)]$	
Results from OpenEpi, Version 3, open source calculator--SSPropor	
Print from the browser with ctrl-P	
or select text to copy and paste to other programs.	

Figure 1. Sample Size Computation

independent association of hypomagnesemia with Type 2 DM and the development of related complications.⁷ The prevalence of hypomagnesemia is 2.5% to 15% in the general population, with higher incidence rates of 13.5% to 47.7% in persons with diabetes.⁸ The recommended nutrient intake (RNI) of magnesium is 225 mg, according to the Philippine Dietary Reference Intake (PDRI). Agdeppa and Custodio reported that the mean magnesium intake among Filipino working adults is 183.2 mg or 82% of RNI, which is lower than recommended.⁹ Due to the growing evidence that magnesium has a fundamental role in carbohydrate metabolism, its abundance, low cost, and relatively low side-effect profile, the authors aimed to determine its association with DSPN for its potential therapeutic benefit with appropriate and timely supplementation.⁶

Methodology

This cross-sectional analytical study was approved by the Institutional Review Board of Makati Medical Center (protocol number: MMCIRB 2021-061; date of approval: April 16, 2021).

The inclusion criteria were established Type 2 Diabetes Mellitus diagnosed according to the American Diabetes Association criteria (fasting plasma glucose ≥ 126 mg/dl, or 2-hour prandial glucose ≥ 200 mg/dl during OGTT, or HbA1c ≥ 6.5 %, or random plasma glucose ≥ 200 mg/dl in a patient with classic symptoms of hyperglycemia or hyperglycemic crisis). The following exclusion criteria were reviewed for each participant: (1). Use of drugs known to affect magnesium levels (diuretics, aminoglycosides, amphotericin B, cetuximab, cyclosporine, or digoxin), (2). Co-morbid conditions that may affect serum magnesium levels and somatic sensory function (serum creatinine > 1.5 mg/dl, acute or chronic malabsorption or diarrheal states, history of alcohol consumption, history of vitamin or mineral supplements in the recent past, recent metabolic acidosis, history of stroke, pregnancy, lactation, sepsis, and thyroid or adrenal dysfunction). The purpose and methodology of the research were thoroughly explained, and a written consent form was signed once the patient agreed to

participate in the study before further examination and diagnostic testing were done. Participants were allowed to withdraw from the study at any point, even if consent had already been given.

Detailed history taking and physical examination were done, including the patient demographics, comorbidities, vital signs, and anthropometrics. Blood samples were then obtained for each patient. They were processed at the laboratory department of Makati Medical Center and included the following: serum magnesium level, albumin, creatinine, HbA1c, FBS, and CBC. The Filipino-translated version of the MNSI questionnaire was self-administered on the same day, followed by the clinical examination. A Filipino-translated version of the Michigan Neuropathy Screening Instrument (MNSI) was validated at the Philippine General Hospital in 2016.¹⁰ It has shown a statistically significant correlation to the degree of neuropathy as measured by Electromyography and Nerve Conduction Velocity (EMG-NCV), the gold standard, with a sensitivity of 95.4%. The authors advocate for its use as an important tool, especially in low- to middle-income countries like the Philippines. The questions aimed to identify peripheral neuropathy symptoms were asked. A "Yes" answer to questions 1-6, 8-12, and 14-15 was given one point. A "No" answer for questions 7 and 13 was likewise given one point. All the 15 questions were included in the scoring algorithm.

After administering the questionnaire, the participant was evaluated using the MNSI clinical examination tool, which included foot inspection, Achilles reflexes examination, determination of the vibratory perception threshold, and the monofilament test. One investigator performed the clinical examination only to minimize rater variability. The results of the MNSI questionnaire blinded the authors before the clinical examination. Cut-off scores of ≥ 4 on the questionnaire and ≥ 1 on the clinical examination were considered positive for DSPN screening.

The minimum computed sample required for this study was 103. This was computed using the online software *OpenEpi*TM, ver 4. Based on the study done by Pfannkuche et al., the overall prevalence of peripheral neuropathy among patients with diabetes mellitus is 40.3%. The margin of error used is 10%. At a 95% confidence interval, the result showed that the sample size was 93. There should be an additional 10% to account for the missing data. The final sample size is 104. The computation was made as follows:

$$\begin{aligned}
 N_{final} &= 93 + .10N_{final} \\
 0.90N_{final} &= 93 \\
 N_{final} &= 103.33
 \end{aligned}$$

Descriptive statistics were used to summarize the general and clinical characteristics of the patients. Frequency and proportion were reported for categorical variables. Shapiro-Wilk and Levene's tests were used to determine the normality distribution and homogeneity of variance of continuous variables, respectively. Continuous quantitative data that met the normality assumption was

Table I. Characteristics of Patients with Type 2 Diabetes Mellitus (n=77)

	All (n=77)	Distal Symmetric Peripheral Neuropathy		<i>p Value</i>
		With n=30 (38.96%)	Without n=47 (61.04%)	
Age (years, Mean, Range)	62 (42-95)	65.5 (42-94)	61 (42-95)	0.514*
<60 (Frequency, Range)	31 (40.26)	10 (33.33)	21 (44.68)	
≥60 (Frequency, Range)	46 (59.74)	20 (66.67)	26 (55.32)	
Sex				0.487†
Male (Frequency, %)	45 (58.44)	19 (63.33)	26 (55.32)	
Female (Frequency, %)	32 (41.56)	11 (36.67)	21 (44.68)	
Weight (kg, Mean ± SD)	74.22 ± 17.24	69.98 ± 14.42	76.92 ± 18.47	0.085‡
Height (cm, Mean ± SD)	162.24 ± 10.42	160.45 ± 9.95	163.39 ± 10.77	0.231‡
BMI (Mean, Range)	27.25 (19.1-55.98)	27.18 (19.1-37.84)	27.73 (19.88-55.98)	0.100*
<18.5 (Frequency, %)	0 (0)	0 (0)	0 (0)	
18.5-22.9 (Frequency, %)	17 (22.08)	8 (26.67)	9 (19.15)	
23-24.9 (Frequency, %)	8 (10.39)	3 (13.33)	4 (8.51)	
≥25 (Frequency, %)	52 (67.53)	18 (60)	34 (72.34)	
Smoking status (Frequency, %)				0.939§
Never (Frequency, %)	52 (67.53)	20 (66.67)	32 (68.09)	
Current (Frequency, %)	9 (11.69)	4 (13.33)	5 (10.64)	
Former (Frequency, %)	16 (20.78)	6 (20)	10 (21.28)	
Duration of diabetes (years)				0.746†
Newly diagnosed	19 (24.68)	8 (26.67)	11 (23.4)	
1-4	13 (16.88)	5 (16.67)	8 (17.02)	
5-10	36 (46.75)	11 (36.67)	25 (53.19)	
>10	9 (11.69)	6 (20)	3 (6.38)	
Medications				0.949§
None	21 (27.27)	8 (26.67)	13 (27.66)	
Injectable only	4 (5.19)	2 (6.67)	2 (4.26)	
Oral hypoglycemic agents only	46 (49.74)	17 (56.67)	29 (61.70)	
Combination	4 (5.19)	2 (6.67)	2 (4.26)	
Unrecalled	2 (2.60)	1 (3.33)	1 (2.13)	
Microvascular Complications				0.518§
Diabetic neuropathy (%)	2 (2.6)	0 (0)	2 (4.26)	
Diabetic nephropathy (%)	0 (0)	0 (0)	0 (0)	-
Diabetic retinopathy (%)	1 (1.3)	0 (0)	1 (2.13)	0.999§
Macrovascular Complications				0.699§
Ischemic heart disease (%)	7 (9.09)	2 (6.67)	5 (10.64)	
Cerebrovascular disease (%)	0 (0)	0 (0)	0 (0)	0.641§
Peripheral arterial disease (%)	0 (0)	0 (0)	0 (0)	-
Comorbidities				0.735†
Hypertension (%)	48 (62.34)	18 (60)	30 (63.83)	
Dyslipidemia (%)	8 (10.39)	2 (6.67)	6 (12.77)	0.472§
CAD (%)	2 (2.6)	0 (0)	2 (4.26)	0.518§
Others (%)	17 (22.08)	8 (26.67)	9 (19.15)	0.438†
Mild COVID-19 infection (%)	49 (63.64)	17 (57.67)	32 (68.09)	0.310†

* Using Asia-Pacific Guidelines: <18.5 (Underweight), 18.5-22.9 (Normal), 23-24.9 (Overweight), ≥25 (Obese)

Statistical tests used: * - Mann-Whitney U test; †-Chi-square test; ‡-Independent t-test; §-Fisher's Exact test

summarized using mean and standard deviation, while those that did not were described using median and range.

Continuous data that satisfied the dual assumption was compared using an independent t-test, while those that violated the variance homogeneity were compared using Welch's test. The non-parametric Mann-Whitney U test was used for skewed data. Categorical variables were compared using the Chi-square test. Fisher's Exact Test was used if the expected percentages in the cells were less than 5%.

Logistic regression was used to determine the association of serum magnesium with distal symmetric

peripheral neuropathy. Odds ratios and their corresponding 95% confidence intervals were reported. Missing values were not estimated. The null hypothesis was rejected at 0.05α-level of significance. STATA version 15.0 (StataCorp SE™, College Station, TX, USA) was used for data analysis.

Results

A total of 85 participants were recruited from the outpatient clinic and admitted patients of Makati Medical Center from September 2021 to January 2022, of which eight were excluded; of these, three had elevated creatinine on testing from previous normal baseline, and five withdrew to undergo blood extraction for diagnostic

Table II. Blood Chemistry of Patients with Type 2 Diabetes Mellitus

	All (n=77)	Distal Symmetric Peripheral Neuropathy		<i>p</i>
		With n=30 (38.96%)	Without n=47 (61.04%)	
	Mean ± SD; Median (Range); Frequency (%);			
Serum magnesium	2.06 ± 0.26	2.06 ± 0.32	2.05 ± 0.23	0.873
<1	0 (0)	0 (0)	0 (0)	
1-1.59	2 (2.6)	1 (3.33)	1 (2.13)	
1.6-2	34 (44.16)	12 (40)	22 (46.81)	
≥2	41 (53.25)	17 (56.67)	24 (51.06)	
Corrected magnesium	2.07 ± 0.26	2.09 ± 0.31	2.06 ± 0.23	0.651
<1	0 (0)	0 (0)	0 (0)	
1-1.59	2 (2.6)	1 (3.33)	1 (2.13)	
1.6-2	32 (41.56)	11 (36.67)	21 (44.68)	
≥2	43 (55.84)	18 (60)	25 (53.19)	
Albumin (g/dL)	4.12 (2.18-5.31)	4 (2.18-5.1)	4.18 (3.33-5.31)	0.092*
<4	27 (35.06)	14 (46.67)	13 (27.66)	
≥4	50 (64.94)	16 (53.33)	34 (72.34)	
Creatinine (meq/L)	0.77 (0.45-1.14)	0.83 (0.5-1.14)	0.75 (0.45-1.08)	0.054*
Creatinine clearance by CKD-EPI	[n=75] 94.52 ± 17.23	90.4 ± 19.01	[n=45] 97.27 ± 15.55	0.106*
HbA1c	6.89 (4.86-17.96)	6.77 (4.86-14.4)	7.37 (5.06-17.96)	0.301*
<7	39 (50.65)	17 (56.67)	22 (46.81)	
7-10	24 (31.17)	7 (23.33)	17 (36.17)	
>10	14 (18.18)	6 (20)	8 (17.02)	
FBS	137 (71-281)	127 (71-280.03)	139 (83-281)	.284*
<80	1 (1.3)	1 (3.33)	0 (0)	
80-130	35 (45.45)	15 (50)	20 (42.55)	
>130	41 (53.25)	14 (46.67)	27 (57.45)	

Statistical tests used: * - Mann-Whitney U test; || - Welch's test

Table III. Association of Serum Magnesium with Distal Symmetric Peripheral Neuropathy

	Serum magnesium		Corrected Serum magnesium	
	Odds Ratio (95% CI)	<i>p</i>	Odds Ratio (95% CI)	<i>p</i>
Crude	1.168 (0.20-6.69)	0.861	1.551 (0.27-9.02)	0.625
Adjusted for age	1.064 (0.18-6.31)	0.945	1.426 (0.24-8.59)	0.699
Adjusted for age and sex	0.887 (0.14-5.48)	0.898	1.198 (0.19-7.52)	0.847
Adjusted for COVID-19	1.520 (0.25-39.36)	0.652	2.039 (0.33-12.75)	0.446
Adjusted for age, sex, and any comorbidity	0.984 (0.15-6.25)	0.986	1.321 (0.20-8.51)	0.770
Adjusted for age, sex, and any comorbidity**	1.064 (0.16-6.88)	0.948	1.453 (0.22-9.60)	0.698

**Includes COVID-19 as comorbidity

Table IV. Correlation with serum magnesium

	Correlation coefficient	<i>p</i>
HbA1c	-0.1571	0.172
FBS	-0.0855	0.460
BMI	0.0694	0.549
Duration of diabetes among previously known (n=58)	0.2470	0.062

testing. A total of 77 adult patients with type 2 diabetes mellitus were included in this study, of whom 30 had distal symmetric peripheral neuropathy, and 47 did not have (Table I). The median age was 62 (42-95), while the median BMI was 27.25 kg/m². A little over half were males (58.44%), while the majority had a BMI $\geq$ 25 (67.53%) and never smoked (67.53%).

Nearly half of the patients had Type 2 diabetes mellitus

for 5-10 years (46.75%) and most were taking oral hypoglycemic agents (49.74%). Three had microvascular complications, such as diabetic neuropathy (2, 2.6%) and diabetic retinopathy (1, 1.3%). Seven had macrovascular complications in the form of ischemic heart disease (7, 9.09%), with majority of the patients having hypertension (62.34%).

The average serum magnesium levels were similar

between those with and those without distal symmetric peripheral neuropathy (2.06 ± 0.32 vs 2.05 ± 0.23 , $p = 0.873$) (Table II). The same was seen for corrected magnesium (Table III). We had insufficient evidence to demonstrate a significant statistical difference between those with and without distal symmetric peripheral neuropathy in relation to glycemic control in terms of HbA1c and FBS (Table IV).

We have insufficient evidence to demonstrate a significant statistical association between serum magnesium and distal symmetric peripheral neuropathy, even after adjusting for age, sex, and comorbidity.

There is likewise no significant statistical correlation between serum magnesium levels with HbA1c, FBS, BMI, or duration of diabetes.

Discussion

This present study could not demonstrate any association between DSPN and serum magnesium, even after adjusting for age, sex, and comorbidity. The average serum and corrected magnesium levels were similar between those with and without DSPN. Likewise, there is no statistically significant correlation between serum magnesium levels with HbA1c, FBS, BMI, or duration of diabetes. Possible explanations why no association was found are as follows: (1) magnesium levels are affected by many other factors, like diet, which is not within the scope of this study; (2) serum magnesium, as determined in the study, cannot give an accurate picture of magnesium balance since there is a primarily intracellular distribution of magnesium in the body.

These findings are consistent with the study done by Hyassat et al., which found no association between hypomagnesemia and diabetic neuropathy diagnosed based on symptoms or an abnormality in nerve conduction studies.⁶ Ramadass et al. likewise showed no significant statistical correlation between serum magnesium, fasting blood sugar (FBS), and post-prandial blood glucose (PPBG) in patients with type 2 DM.¹¹ In another study by Rao et al., the mean value of FBS, PPBG, and HbA1c was higher among the group with serum Mg <1.7 mg/dl.¹² In 2015, a prospective case-control study of serum Mg in relation to micro- and macrovascular complications of type 2 DM in India reported no correlation in terms of neuropathy and ischemic heart disease but with an established association with retinopathy and nephropathy.¹³ Likewise, Ghattaura et al. reported a weak association between BMI and hypomagnesemia, and there is no statistical difference in age or duration of diabetes according to Mg levels as reported by Arpaci et al.^{12,14}

In contrast, Chu et al. suggested that low serum magnesium levels may underlie the pathophysiologic features seen in DSPN. Instead of a validated screening tool, they performed nerve conduction studies in all subjects. They found that lower magnesium levels were significantly associated with a lower composite Z amplitude score in patients with diabetes mellitus,

indicating that low serum magnesium levels might affect peripheral nerve function through axonal degeneration.¹⁵ Barbagallo et al. likewise found that serum magnesium concentration is related to insulin response and diabetes. Hypomagnesemia may aggravate insulin resistance, especially in overweight patients, predisposing them to a myriad of metabolic complications. Reduction in the intracellular magnesium concentration results in defective tyrosine-kinase activity and post-receptor impairment in insulin action.¹⁶ The prevalence of hypomagnesemia in persons with diabetes reported in the literature is inconsistent with the present study among the Filipino population. The wide range in reported prevalence of hypomagnesemia likely reflects the difference in the definition of hypomagnesemia, techniques in Mg measurements, and the heterogeneity of the selected patient cohort.¹²

Hypomagnesemia in patients with diabetes can result from poor oral intake, altered gastrointestinal absorption, and enhanced renal excretion. There are various metabolic disturbances suggested to promote urinary magnesium excretion in persons with diabetes, including hypokalemia, hypophosphatemia, and metabolic acidosis. Insulin has been shown to affect magnesium handling in both the thick ascending limb and the distal convoluted tubule. Insulin enhances intracellular magnesium uptake, presumably via tyrosine kinase, and may stimulate cAMP production. This can further potentiate magnesium uptake via other cAMP-dependent hormones, including parathyroid hormone. This way, insulin deficiency or resistance may exacerbate renal magnesium wasting.¹⁸ Due to these findings, magnesium has been suggested as a possible treatment for diabetes. However, randomized controlled trials have conflicting results about the effect of supplementation on glycemic control in diabetes.¹⁹

This study has several limitations. First is the methodology used to screen DSPN using the validated Filipino-translated version of the MNSI questionnaire, and clinical examination with no nerve conduction studies to confirm. There is no single diagnostic test for the detection of DSPN. Due to the lack of agreement on the definition and diagnostic assessment of diabetic neuropathy, several consensus conferences were done to overcome the current problems, the most recent of which has redefined the minimal criteria for diagnosing typical DSPN.¹⁷ Probable DSPN is defined by a combination of symptoms and signs of neuropathy, including any two or more of the following: neuropathic symptoms, decreased distal sensation, or unequivocally decreased or absent ankle reflexes.¹ A few clinical "pearls" can likewise aid the clinician in identifying symptoms atypical for DSPN secondary to diabetes. These include motor greater than sensory symptoms and a reportable asymmetry.¹⁷

Another limitation of the study is using a point determination of serum magnesium to characterize overall magnesium balance in the body. The decrease in serum magnesium concentration usually presents after a severe deficiency of magnesium in the body. It can be

used as a specific indicator of magnesium metabolism because the reduction is closely related to the intracellular magnesium concentration. However, it has a low sensitivity.²⁰ Therefore, the authors recommend combining serum and urine magnesium in future investigations better to determine the overall condition of magnesium *in vivo* and to provide a reference for the diagnosis, management, and prognostication of persons with type 2 diabetes mellitus. The study also failed to identify the exact duration of Type 2 diabetes among study participants, an important factor to consider in patients with DSPN. Future studies can include younger, non-critically ill populations 18 years and older with prediabetes, types 1 and 2 diabetes mellitus, including post-prandial blood glucose and alcoholism in the study variables.

Lastly, the study has a limited sample size. The authors recommend future studies to be done on a larger scale with a detailed plan for collecting sufficient data and longer follow-ups. Peripheral neuropathy in Southeast Asia is associated with multiple possible causes, diabetes being the most common in the region. It can also co-exist with other electrolyte abnormalities, especially in severe cases and vitamin deficiencies which are not within the scope of this study.²¹ This present study also found a higher serum magnesium level with increasing BMI, which could be possibly related to nutritional intake and is an interesting area for future investigation.

Although no association has been found between serum magnesium and DSPN, non-association is not necessarily proven by non-statistically significant results. Given the postulated mechanisms of hypomagnesemia in the setting of diabetes mellitus, the researchers recommend screening those at high risk, including but not limited to patients with poor oral intake, poor gastrointestinal absorption, enhanced renal excretion, various metabolic disturbances, and insulin deficiency or resistance using both serum and urine measurements for enhanced sensitivity. Early detection of abnormality leads to prompt management and patients should be screened regularly for diabetes complications as recommended by the American Diabetes Association. It is critical to increase the patients' awareness of DSPN and a sense of urgency to seek medical consultation. Likewise, the investigators suggest optimizing glycemic control and other modifiable risk factors like hypertension, dyslipidemia, obesity, smoking, alcoholism, physical inactivity, and vitamin B12 status.

Conclusion

This present study was unable to demonstrate any association between DSPN and serum magnesium, even after adjusting for age, sex, and any comorbidity. Despite the limited sample size, the study gives us an idea of the magnesium status of patients with DSPN as diagnosed by MNSI. The average serum magnesium and corrected magnesium levels were similar between those with compared to those without DSPN. There is insufficient evidence to demonstrate a significant statistical difference between those with and without DSPN and

glycemic control in terms of FBS and HbA1c. Likewise, there is no significant statistical correlation between serum magnesium levels with HbA1c, FBS, BMI, or duration of diabetes.

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Appendix

Table A. Michigan Neuropathy Screening Instrument - History

	Frequency (%)	
	No	Yes
1. Are your legs and/or feet numb?	46 (59.74)	31 (40.26)
2. Do you ever have any burning pain in your legs and/or feet?	60 (77.92)	17 (22.08)
3. Are your feet too sensitive to touch?	69 (89.61)	8 (10.39)
4. Do you get muscle cramps in your legs and/or feet?	68 (88.31)	9 (11.69)
5. Do you ever have any prickling feelings in your legs or feet?	50 (64.94)	27 (35.06)
6. Does it hurt when the bed covers touch your skin?	71 (92.21)	6 (7.79)
7. When you get into the tub or shower, are you able to tell the hot water from the cold water?	4 (5.19)	73 (94.81)
8. Have you ever had an open sore on your foot?	71 (92.21)	6 (7.79)
9. Has your doctor ever told you that you have diabetic neuropathy?	72 (93.51)	5 (6.49)
10. Do you feel weak all over most of the time?	77 (100)	0 (0)
11. Are your symptoms worse at night?	71 (92.21)	6 (7.79)
12. Do your legs hurt when you walk?	64 (83.12)	13 (16.88)
13. Are you able to sense your feet when you walk?	5 (6.49)	72 (93.51)
14. Is the skin on your feet so dry that it cracks open?	67 (87.01)	10 (12.99)
15. Have you ever had an amputation?	75 (97.4)	2 (2.6)

Through taking history by using the Michigan Neuropathy Screening Instrument, most respondents did not have numb legs or feet (59.74%), burning pain (77.92%), and prickling feelings in your legs or feet (64.94%) (*Table A*). Most of the respondents did not have feet too sensitive to touch (89.61%), and muscle cramps in their legs and feet (88.31%).

Most of the respondents (92.21%) said it does not hurt when the bed covers touch their skin. Additionally, the majority (94.81%) said that when in the tub or shower, they can tell the hot water from the cold water.

Most respondents (83.12%) do not have legs that hurt when walking, and a majority (93.51%) can sense their feet when walking.

It is worth noting at all respondents said that they did not feel weak all over most of the time, and that majority (92.21%) do not have symptoms that are worse at night.

Most of the respondents (92.21%) do not have open sores on their feet, have skin on their feet so dry that it cracks open (87.01%), have a diagnosis of diabetic neuropathy (93.51%), or ever had an amputation (97.4%).

Table B. Michigan Neuropathy Screening Instrument - Physical Assessment

	Frequency (%)	
	Left	Right
Appearance of feet		
Normal	32 (41.56)	33 (42.86)
Abnormal	45 (58.44)	44 (57.14)
Deformities	1 (2.22)	2 (4.55)
Dry skin, callus	45 (100)	44 (100)
Infection	2 (4.44)	1 (2.27)
Fissure	2 (4.44)	2 (4.55)
Others	0 (0)	0 (0)
Ulceration	0 (0)	2 (2.60)
Ankle Reflexes		
Present	74 (96.10)	75 (97.40)
Present/Reinforcement	3 (3.90)	2 (2.60)
Absent	0 (0)	0 (0)
Vibration perception at great toe		
Absent	67 (87.01)	68 (88.31)
Decreased	8 (10.39)	8 (10.39)
Absent	2 (2.60)	1 (1.30)
Monofilament		
Normal	64 (83.12)	64 (83.12)
Reduced	10 (12.99)	11 (14.29)
Absent	3 (3.90)	2 (2.60)

Through conducting a physical assessment by using the Michigan Neuropathy Screening Instrument, most respondents have left (58.44%) and right (57.14%) feet that do not have a normal appearance (*Table B*). All the feet had dry skin and callus, only 2 right feet (2.60%) had ulceration.

Majority of the left (96.10%) and right (97.40%) had present ankle reflexes. Majority of the left (87.01%) and right (88.31%) feet had vibration perception at the great toe. Lastly, most left (83.12%) and right (83.12%) feet had normal monofilament.