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REVIEW ARTICLE

The Effectiveness of Oral Alpha-Lipoic Acid in Improving Peripheral Neuropathy Symptoms in Type 2 Diabetes Patients

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Abstract:

Background and Objectives: Nearly half of patients with diabetes are affected by diabetic peripheral neuropathy (DPN) during their lifetime. The potential neuroprotective effects of alpha-lipoic acid (ALA) have generated interest, as in diabetic patients these may enhance microcirculation and nerve function, offering a promising therapeutic option. This study aimed to assess the efficacy of oral ALA supplementation in improving symptoms of DPN.

Methods: A quasi-experimental study was carried out at Layla Qasem Specialized Diabetic Centers in Erbil, Iraq, between February 1 and June 30, 2024. A total of 100 patients with type 2 diabetes and confirmed DPN were enrolled (50 in the intervention group and 50 controls). The intervention group received 600 mg of ALA twice daily, while the control group opted not to take the treatment. Data were collected using structured interviews, neurological evaluation with the Michigan Neuropathy Screening Instrument (MNSI: history and physical examination), and the Douleur Neuropathique 4 (DN4) scale for neuropathic pain. Body mass index (BMI) and HbA1c were also measured.

Results: The intervention group showed marked improvement compared with controls: MNSI history (87.5% vs. 40.7%), MNSI physical exam (27.6% vs. 2%), and DN4 scale (70.2% vs. 14%). Glycemic control also improved, with mean fasting blood glucose reduced by 59.06 mg/dL and HbA1c decreased by 1.31% in the intervention group relative to controls.

Conclusion: Supplementation with ALA at 600 mg twice daily significantly alleviated neuropathic symptoms and improved glycemic parameters in patients with type 2 diabetes and peripheral neuropathy.

Keywords: Type 2 Diabetes; Alpha-Lipoic Acid; Michigan Neuropathy Screening Instrument; DN4 scale.

Introduction

Type 2 diabetes mellitus (T2DM) is a long-term progressive disorder characterized by hyperglycemia resulting from insulin resistance and impaired pancreatic β-cell activity [1]. This type of diabetes frequently manifests in individuals who are overweight or obese, as excess body weight exacerbates insulin resistance [2].. The exact etiology of T2DM remains unclear, though it is generally attributed to an interplay of genetic predisposition and environmental influences. The International Diabetes Federation estimates that around 73 million people in the Middle East—approximately one in six adults—are currently living with diabetes. By 2030, this number is expected to rise to 95 million, and by 2045, it is expected to rise to 136 million [3]. Diabetes is associated with numerous complications that reduce quality of life, increase healthcare costs, and contribute to premature mortality. Persistent hyperglycemia leads to widespread damage to various organs, including the kidneys, heart, eyes, and nerves [4]. Diabetic peripheral neuropathy (DPN) is among the most frequent and severe complications, involving both the peripheral and autonomic nervous systems. DPN typically presents as distal symmetric polyneuropathy, beginning in the lower limbs and progressing upward. This condition is classified under peripheral neuropathies, a broad group of nerve disorders. It affects 1-7% of the general population, with increased prevalence in individuals over 50 years old [5]. Treatment of DPN is approached from both preventive and symptomatic perspectives [6]. The most effective preventive strategy, particularly for delaying or slowing disease progression, is tight glycemic control [7]. Alpha-lipoic acid (ALA), which is derived from octanoic acid, is a naturally occurring antioxidant that has shown promise in treating diabetic neuropathy. ALA and its active metabolite, dihydrolipoic acid, improve nerve function by promoting nerve growth factor expression, enhancing microcirculation, and increasing motor conduction

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velocity [8]. It has proven effective in conditions involving oxidative stress, such as diabetic neuropathy, by improving nitric oxide-mediated vasodilation. Recognized by the Food and Drug Administration (FDA) as both safe and effective, ALA stands out as a valuable therapeutic option in managing DPN [9].

This study seeks to determine the effectiveness of oral alpha-lipoic acid in reducing peripheral neuropathy symptoms among patients with type 2 diabetes, using a quasi-experimental approach.

Materials and methods

One hundred patients with type 2 diabetes mellitus (T2DM) diagnosed at least six months prior were enrolled. A quasi-experimental design was conducted at Layla Qasim Specialized Diabetic Center in Erbil, Iraq, from 1st February to 30th June 2024. Convenience The sampling method was used to recruit patients. The expected effect size (Cohen's d) is estimated at approximately 0.525, assuming a standard deviation of 2 [10]. Using a two-tailed alpha level of 0.05 and a power of 80%, the calculated sample size for a two-group comparison (intervention vs. control) is approximately 57 participants per group, totaling 114 participants. However, only fifty patients agreed to receive 600 mg oral alpha lipoic acid (ALA) twice daily for three months (intervention group), while the remaining fifty declined (control group). Patients were included if they were 18 years or older, showed symptoms suggestive of diabetic peripheral neuropathy (DPN) for at least six months, and attended Layla Qasim Specialized Diabetic clinics. Those with significant comorbidities, type 1 diabetes, advanced uncontrolled diabetes (HbA1c >10%), or neuropathy treatments were excluded.

Ethical approval was obtained from the Scientific and Ethical Committee of the Arab Board for Medical Specializations, and participants were asked to sign a written informed consent. Interviews were conducted in Arabic or Kurdish, and patient confidentiality was maintained using coded identifiers.

Data collection involved: Sociodemographic and clinical data, BMI calculation using measured weight and height, and categorized as follows: "underweight (<18.5 kg/m²), normal weight (18.5–24.9 kg/m²), overweight (25.0–29.9 kg/m²), obesity class I (30.0–34.9 kg/m²), obesity class II (35.0–39.9 kg/m²), and obesity class III (≥40.0 kg/m²)." Neuropathy assessments via the DN4 questionnaire (a score ≥4 indicates neuropathic pain [11]). Michigan Neuropathy Screening Instrument (MNSI) (questionnaire + physical exam; score ≥7 is abnormal) [12].

Participants provided blood samples for tests including HbA1c, fasting blood glucose, serum thyroid stimulating hormone, alanine aminotransferase, B12, and creatinine (baseline only). Hemoglobin A1c was analyzed using the Diasys One HbA1CFS method; other labs were done on the Cobas C 111 analyzer.

The intervention group received ALA for three months and was then reassessed using the same tools and procedures to evaluate changes in neuropathy symptoms and glycemic control.

Data were analyzed using SPSS version 26. Statistical tests were selected according to data type and distribution. Group proportions were compared with the chi-square test or Fisher's exact test when expected counts were low. The McNemar test assessed changes in proportions within groups over time. Differences between two independent means were examined using the unpaired t-test, while the Mann–Whitney test compared mean ranks when normality was not met. The Wilcoxon signed-rank test evaluated paired medians at two time points. Data normality was checked with the Shapiro–Wilk test, and nonparametric methods were applied when required. A p-value < 0.05 was considered statistically significant.

Results

One hundred patients with type 2 diabetes and peripheral neuropathy were enrolled, with 50 receiving alpha-lipoic acid (intervention group) and 50 serving as controls. Their mean age was 56.3 ± 9.9 years (range 35–75; median 58), and the largest subgroup (38, 38%) was aged 55–64 years, with no significant difference in age distribution between groups (p = 0.858). Over half (58, 58%) lived in urban areas (p = 1.000), and most were married (75, 75%; p = 0.420). One-third (33.33%) were illiterate, and 28 (28%) had primary education. Daily income was sufficient for 56 (56%) participants, and 60 (60%) were unemployed or housewives. No significant intergroup differences were found for education (p = 0.465), income (p = 0.062), or occupation (p = 0.334) (Table 1).

Lifestyle and clinical factors also showed little variation between groups. Overall, 36% were smokers and 4% consumed alcohol (p = 0.679 and p = 0.617). A family history of diabetic foot was reported in 88%, and 15% had a prior foot ulcer (p = 1.000 and p = 0.779). Diabetes duration ≥15 years was more frequent in the intervention group (30%) than in controls (8%) (p = 0.034). No significant differences were found in adherence to medication (p = 0.161), treatment type (p = 0.137), or comorbidities (p = 0.072) (Table 2).

Neuropathy outcomes improved markedly in the intervention group. The median Michigan score (history) fell from 6 to 3 (p < 0.001), compared with a smaller reduction from 7 to 6 in controls (p < 0.001) (Table 3). The Michigan physical assessment median dropped from 4 to 2 in the intervention group (p < 0.001), while controls remained unchanged at 4 (p = 0.403). Abnormal Michigan history scores declined from 48% to 6% in the intervention group (p < 0.001), versus 54% to 32% in controls (p = 0.003) (Figure 1). Reductions in both history and physical assessment scores were significantly greater in the intervention group (p < 0.001) (Table 4). Abnormal Michigan physical assessment findings decreased from 94% to 68% after intervention (p < 0.001), compared with 98% to 96% in controls (p = 1.000) (Figure 2).

ALA treatment also improved DN4 scores. Significant reductions were observed across all DN4 categories (p < 0.05), whereas controls showed no changes except for "electric shock" (74% to 54%, p = 0.002) (Table 5). The DN4 median dropped from 6 to 2 in the intervention group (p < 0.001), while in controls it decreased slightly from 6.5 to 6 (p = 0.016) (Table 6). Neuropathic pain prevalence declined from 94% to 28% in the intervention group (p < 0.001), compared with 100% to 86% in controls (Figure 3). The median DN4 score reduction was 4 points with ALA versus 0 in controls (p < 0.001) (Table 7).

Glycemic indices also improved significantly. HbA1c and fasting blood sugar decreased more in the intervention group (p < 0.001 and p = 0.006), while BMI reduction approached significance (p = 0.055) (Table 8). Overall improvements in the intervention group were 87.5% for Michigan history, 27.6% for Michigan physical assessment, and 70.2% for DN4, far exceeding the modest changes observed in controls (Figure 4).

Discussion

Diabetic neuropathy affects around 50% of diabetic patients, often causing painful symptoms and serious complications like

foot ulcers and amputations, and its management—particularly of diabetic peripheral neuropathy—remains complex and burdensome, requiring multifactorial interventions [13]. In the present study, the intervention and control groups were well-matched across key sociodemographic and historical variables, such as age, marital status, education level, family history of DM, and medication compliance. These findings are in line with similar baseline matching reported in previous studies by Albeladi et al. [14], Anand Vijayakumar et al. [15], and Mamdouh R. El-Nahas et al. [16], although the latter employed a larger sample size and longer treatment duration. The consistency of baseline characteristics enhances the reliability of assessing alpha lipoic acid (ALA) efficacy in managing DPN. However, it is important to note that in the current study, the duration of diabetes mellitus was significantly longer in the intervention group compared to the control group, which could introduce a confounding variable that may influence the outcomes independently of the intervention itself. This has been acknowledged and addressed as a limitation.

At baseline, the prevalence of diabetic neuropathy, as measured by the Michigan Neuropathy Screening Instrument (MNSI), was slightly higher in the control group (54%) than in the intervention group (48%). Following ALA treatment, the prevalence in the intervention group dropped significantly to 6%, while the control group saw only a modest reduction to 32%, highlighting ALA's effectiveness in symptom reduction. The MNSI scores confirmed these findings, showing significant improvement in the intervention group, a trend consistent with studies by El-Nahas et al. [16], Ziegler et al. [17], and Baghdadi et al. [18], who similarly reported improvement in MNSI scores following ALA use. However, reliance on the MNSI questionnaire alone has limitations; its lower sensitivity may overlook mild cases of DPN, and thus, a combination of patient history and physical assessment is recommended for more accurate diagnosis [19][20][21][22].

The DN4 questionnaire, used to assess neuropathic pain, also showed significant reductions in symptom scores in the ALA group post-treatment, further supporting the analgesic effects of ALA. Comparable improvements were documented in studies by Hsieh et al. [23], Salehi et al. [24], and Abubaker et al. [25], while Papanas et al. [26] noted a less pronounced effect, likely due to differences in study design. When analyzing individual DN4 score components, the intervention group exhibited significant improvements, while the control group had little to no change. These results parallel those of Abubaker et al. [25] and El-Nahas et al. [17], reinforcing ALA's role in reducing neuropathic pain.

Although the control group did not receive alpha-lipoic acid, a modest reduction in neuropathic pain scores was also observed. This improvement may be attributed to the general lifestyle and dietary recommendations provided to all participants, which are known to contribute to better glycemic control and overall metabolic health. However, the degree of symptom reduction in the control group was considerably less pronounced compared to the intervention group, suggesting that the observed improvements in the latter are likely due to the additional effect of ALA rather than lifestyle modification alone. Such findings strengthen the rationale for using ALA as an adjunctive option in managing diabetic peripheral neuropathy.

In terms of metabolic parameters, the intervention group demonstrated significant reductions in fasting blood sugar and HbA1c levels, indicating improved glycemic control, whereas the control group showed minimal changes. These outcomes are consistent with findings from Derosa et al. [27] and Okuroğlu [28], both of whom observed similar biochemical improvements in ALA-treated patients. However, BMI changes post-treatment were not statistically significant in this study, aligning with Garcia et al. [29], who also reported only a marginal reduction in BMI.

The overall improvement in DPN symptoms, as assessed by both the MNSI and DN4 scales, was substantial in the ALA group, with approximately 87% showing marked symptom relief compared to 40% in the control group. This improvement surpasses that reported in studies by Derosa et al. [27] and Okuroğlu [28], likely due to differences in treatment intensity, population characteristics, or assessment tools. Similarly, Mamdouh R. El-Nahas et al. [16] reported a 48% improvement based on Vibration Perception Threshold, while Abubaker et al. [25] observed a 40% improvement using the DN4 scale. Variability in reported outcomes across studies may be attributed to differing methodologies, follow-up durations, and evaluation instruments.

The results of this study should be interpreted in light of its limitations. First, the quasi-experimental design, while practical, introduces the possibility of selection bias, as patients self-selected into the intervention or control group rather than being randomized. This may have led to unmeasured confounding variables influencing the outcomes. For example, although age was partially balanced by chance, with similar percentages of patients in each age group across the intervention and control groups, other factors such as duration of diabetes were not controlled for, which could introduce a potential confounding effect on the outcomes. Second, although the sample size was close to the calculated requirement, it fell short of the estimated 114 participants needed for optimal power, which could have affected the precision of the effect estimates. Third, the study was conducted at a single specialized diabetic center in Erbil, potentially limiting the generalizability of the results to other settings or populations. Additionally, the relatively short follow-up period of three months may not capture long-term efficacy or safety of ALA supplementation. Fourth, the duration of the study was short; a longer follow-up period may provide more accurate data on HbA1c. Finally, although validated tools like DN4 and MNSI were used for neuropathy assessment, the reliance on subjective reporting and clinician-administered scores may introduce observer bias or variability in interpretation. Future randomized controlled trials with larger, more diverse samples and longer follow-up durations are warranted to confirm and extend these findings.

Implication for practice

This study underscores the potential of oral alpha-lipoic acid as an adjunct therapy for diabetic peripheral neuropathy, especially in type 2 diabetes. The marked improvements in neuropathic pain and functional outcomes indicate that ALA may provide additional symptom relief beyond lifestyle modification alone. Given its ease of administration and tolerability, ALA could be incorporated into clinical practice for patients who are either unresponsive to or unable to tolerate conventional pharmacologic treatments. Clinicians should consider evaluating patients with early signs of neuropathy for potential benefit from ALA

supplementation as part of a comprehensive, individualized treatment strategy.

Conclusion

This study demonstrated that oral alpha-lipoic acid supplementation was associated with meaningful improvements in neuropathic symptoms and glycemic control among patients with type 2 diabetes. Improvements were observed across both clinical assessments and patient-reported outcomes, suggesting the potential role of alpha-lipoic acid as an adjunctive option in managing diabetic peripheral neuropathy.

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Table 1. Socio-demographic characteristics of studied patients.

	Intervention No. (%)	Control No. (%)	Total No. (%)	P-value
Age (years)				0.858*
35-44	7 (14.0)	5 (10.0)	12 (12.0)	
45-54	15 (30.0)	13 (26.0)	28 (28.0)	
55-64	18 (36.0)	20 (40.0)	38 (38.0)	
65-75	10 (20.0)	12 (24.0)	22 (22.0)	
Mean (SD)	55.8 (10.1)	56.9 (9.7)	56.3 (9.9)	0.588†
Residency				1.000*
Rural	21 (42.0)	21 (42.0)	42 (42.0)	
Urban	29 (58.0)	29 (58.0)	58 (58.0)	
Marital Status				0.420**
Married	38 (76.0)	37 (74.0)	75 (75.0)	
Single	2 (4.0)	2 (4.0)	4 (4.0)	
Divorced	0 (0.0)	3 (6.0)	3 (3.0)	
Widowed	10 (20.0)	8 (16.0)	18 (18.0)	
Education Level				0.465*
Illiterate	16 (32.0)	17 (34.0)	33 (33.0)	
Primary	16 (32.0)	12 (24.0)	28 (28.0)	
Intermediate	4 (8.0)	10 (20.0)	14 (14.0)	
Secondary	7 (14.0)	5 (10.0)	12 (12.0)	
College and above	7 (14.0)	6 (12.0)	13 (13.0)	
Family income				0.062**
Sufficient for daily needs	32 (64.0)	24 (48.0)	56 (56.0)	
Not sufficient	13 (26.0)	24 (48.0)	37 (37.0)	
Exceeds daily needs	5 (10.0)	2 (4.0)	7 (7.0)	
Occupation				0.334*
Unemployed/housewives	32 (64.0)	28 (56.0)	60 (60.0)	
Unskilled manual workers	4 (8.0)	8 (16.0)	12 (12.0)	
Skilled manual workers	5 (10.0)	6 (12.0)	11 (11.0)	
Non-manual workers	2 (4.0)	5 (10.0)	7 (7.0)	
High-level occupation	7 (14.0)	3 (6.0)	10 (10.0)	
Total	50 (100.0)	50 (100.0)	1 0 0 (100.0)	

*Calculated by Chi-square test. **Calculated by Fisher's exact test. †By unpaired t-test.

Table 2 History characteristics of studied patients.

	Intervention No. (%)	Control No. (%)	Total No. (%)	p-value
Smoking				0.679**
Smoker	19 (38.0)	17 (34.0)	36 (36.0)	
Non-Smoker	30 (60.0)	33 (66.0)	63 (63.0)	
Ex-Smoker	1 (2.0)	0 (0.0)	1 (1.0)	
Alcohol intake				0.617**
Alcoholic	3 (6.0)	1 (2.0)	4 (4.0)	
Non-Alcoholic	47 (94.0)	49 (98.0)	96 (96.0)	
Family history of DM				1.000*
Yes	44 (88.0)	44 (88.0)	88 (88.0)	
No	6 (12.0)	6 (12.0)	12 (12.0)	
History of foot ulcer				0.779*
Yes	8 (16.0)	7 (14.0)	15 (15.0)	
No	42 (84.0)	43 (86.0)	85 (85.0)	
DM Duration (years)				0.034**
<5	4 (8.0)	3 (6.0)	7 (7.0)	
5-9	18 (36.0)	26 (52.0)	44 (44.0)	
10-14	13 (26.0)	17 (34.0)	30 (30.0)	
≥ 15	15 (30.0)	4 (8.0)	19 (19.0)	
Medication regularity				0.161*
Yes	40 (80.0)	45 (90.0)	85 (85.0)	
No	10 (20.0)	5 (10.0)	15 (15.0)	
Treatment types				0.137**
Diet	1 (2.0)	0 (0.0)	1 (1.0)	
Oral antidiabetic (OAD)	37 (74.0)	44 (88.0)	81 (81.0)	
Insulin	2 (4.0)	0 (0.0)	2 (2.0)	
Both OAD and Insulin	10 (20.0)	6 (12.0)	16 (16.0)	
Comorbidities				0.072*
Yes	29 (58.0)	20 (40.0)	49 (49.0)	
No	21 (42.0)	30 (60.0)	51 (51.0)	
Total	50 (100.0)	50 (100.0)	1 0 0 (100.0)	

*Calculated by Chi-square test. **Calculated by Fisher's exact test.

Table 3. Michigan score parameters and Michigan physical assessment scale parameters before and after treatment among the intervention group and control.

	Score before treatment**		Score after treatment**		
Michigan score parameters					
Group	Mean	Median	Mean	Median	p-value
Intervention	6.58	6	3.24	3	0.000*
Control	6.56	7	5.64	6	0.000*
	Score before treatment***		Score after treatment***		
Michigan physical assessment scale parameters					
Intervention	4.42	4.00	2.50	2.00	0.000*
Control	4.11	4.00	4.05	4.00	0.403*

*Calculated by Wilcoxon signed ranks test. **This score was out of 13 points, calculated according to patients' history. ***This score was out of 10 points, calculated according to the doctor's assessment.

Table 4. Comparing the difference in Michigan scores (score before minus score after intervention) between the two study groups.

Difference in score	Intervention	Control	p-value*
Michigan scores			
Mean	3.34	0.92	
Median	4	1	
Mean rank	67.22	33.78	0.000
Michigan physical assessment scores			
Mean	1.92	0.06	
Median	2.00	0.00	
Mean rank	68.22	32.78	0.000

*Calculated by Mann-Whitney test.

Table 5. DN4 symptoms before and after treatment among patients of the intervention group and the control group.

	Before treatment No. (%)	After treatment No. (%)	p-value*
Intervention group			
Burning	41 (82.0)	17 (34.0)	0.000
Painful cold	23 (46.0)	7 (14.0)	0.000
Electric shock	28 (56.0)	9 (18.0)	0.000
Tingling	42 (84.0)	12 (24.0)	0.000
Pins needles	42 (84.0)	14 (28.0)	0.000
Numbness	49 (98.0)	33 (66.0)	0.000
Itching	18 (36.0)	6 (12.0)	0.002

Hypoesthesia to touch	37 (74.0)	17 (34.0)	0.000
Hypoesthesia pin prick	31 (62.0)	12 (24.0)	0.000
Brushing	12 (24.0)	2 (4.0)	0.002
Control Group			
Burning	43 (86.0)	40 (80.0)	0.508
Painful cold	28 (56.0)	22 (44.0)	0.238
Electric shock	37 (74.0)	27 (54.0)	0.002
Tingling	41 (82.0)	41 (82.0)	1.000
Pins needles	48 (96.0)	42 (84.0)	0.070
Numbness	47 (94.0)	42 (84.0)	0.063
Itching	15 (30.0)	12 (24.0)	0.250
Hypoesthesia to touch	41 (82.0)	44 (88.0)	0.375
Hypoesthesia pin prick	27 (54.0)	23 (46.0)	0.344
Brushing	1 (2.0)	1 (2.0)	1.000

*Calculated by McNemar test.

Table 6. DN4 score parameters before and after treatment in each of the intervention and control groups.

	Score before treatment**		Score after treatment**		
Group	Mean	Median	Mean	Median	p-value*
Intervention	6.46	6.00	2.58	2.00	0.000
Control	6.56	6.50	5.88	6.00	0.016

*Calculated by Wilcoxon signed ranks test. **This score was out of 10 points.

Table 7. Comparing the difference in DN4 scores (score before minus score after intervention) between the two study groups.

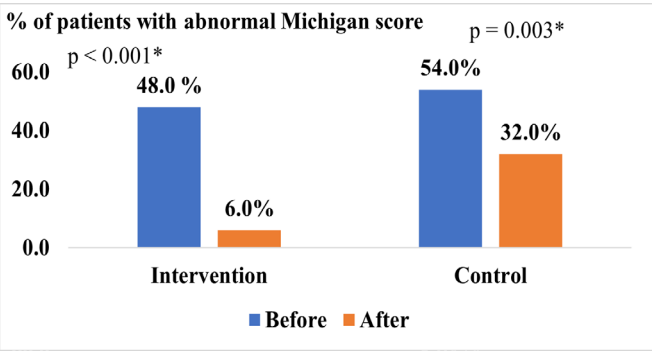
Difference in score	Intervention	Control	p-value*
Mean	3.88	0.68	
Median	4.00	0.00	
Mean rank	68.40	32.60	0.000

*Calculated by Mann-Whitney test.

Table 8. Comparing the difference in blood investigations (reading before minus reading after intervention) between the two study groups.

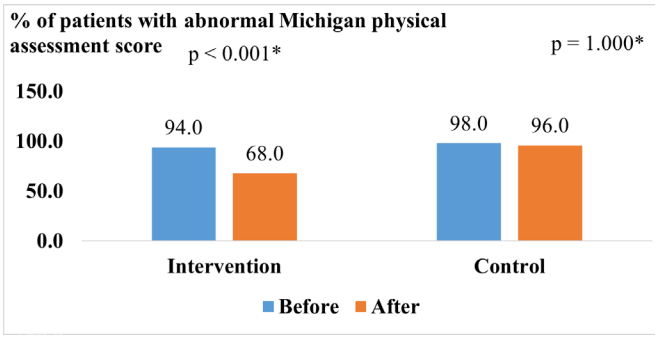
	Intervention			Control			
Difference (before minus after)	Mean	Median	Mean Rank	Mean	Median	Mean Rank	p-value*
HbA1c	1.31	1.24	61.91	0.40	0.35	39.09	0.000
FBS	59.06	55.00	58.49	18.72	11.00	42.51	0.006
BMI	0.76	0.40	56.06	0.23	0.03	44.94	0.055

*Calculated by Mann-Whitney test.



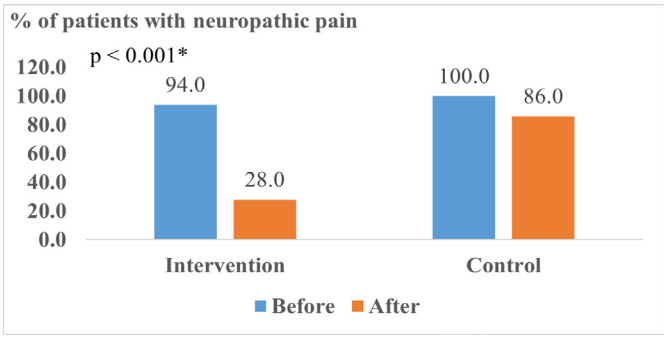
*Calculated by McNemar test.

Figure 1. The proportion of patients with abnormal Michigan scores before and after intervention, in each study group.



*Calculated by McNemar test.

Figure 2. The proportion of patients with abnormal physical Michigan scores before and after intervention, in each study group.



*By McNemar test. Note that the p-value of the control group can't be calculated (one of the percentages was 100%).

Figure 3. The proportion of patients with neuropathic pain (according to DN4 classification) before and after intervention, in each study group.

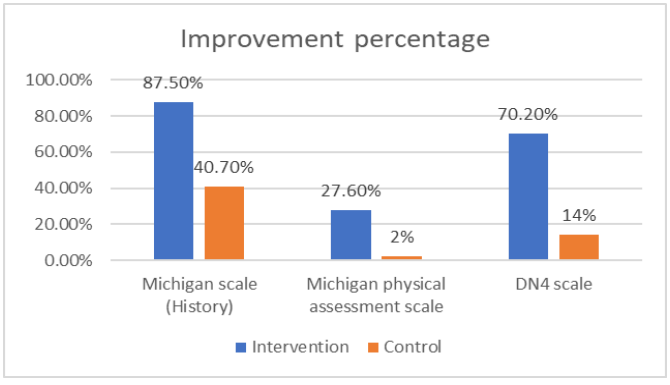


Figure 4. Improvement percentage as assessed by one of the scales, in each of the study groups.