Journal of Multidisciplinary Healthcare

Dovepress
Taylor & Francis Group

Open Access Full Text Article

ORIGINAL RESEARCH

Evaluation of the Diagnostic Utility of Combining Electromyography with Clinical and Pathological Indices in the Diagnosis of Diabetic Peripheral Neuropathy: A Retrospective Study

Wenhua Wang, Ziwei Gong, Siyi Wang, Gang Li

Department of Neurology, Wuhan Fourth Hospital, Wuhan, 430000, People's Republic of China

Correspondence: Gang Li, Email ioog124@163.com

Objective: To evaluate the diagnostic value of combining electromyography (EMG) with clinical and pathological parameters in diagnosing diabetic peripheral neuropathy (DPN).

Methods: A retrospective study was conducted on 156 diabetic patients (69 with DPN, 87 without) treated from July 2022 to December 2024. Clinical, biochemical, and EMG parameters were compared between groups. Multivariate logistic regression identified independent predictors of DPN. ROC curve analysis assessed diagnostic performance.

Results: Group A (DPN) had significantly higher levels of glycated hemoglobin, cystatin C, uric acid, and creatinine, and significantly reduced motor and sensory nerve conduction velocities compared to Group B. Logistic regression identified these indicators as independent risk factors. Combined ROC analysis showed higher diagnostic accuracy (AUC = 0.873) than any single indicator.

Conclusion: The combination of EMG with selected clinical and pathological parameters offers high diagnostic accuracy for DPN in diabetic patients.

Keywords: diabetic neuropathies, electromyography, retrospective studies, risk assessment, ROC curve

Introduction

Diabetes mellitus (DM) is a chronic metabolic disorder characterized by persistent hyperglycemia resulting from impaired insulin secretion and/or insulin action, which disrupts carbohydrate, fat, and protein metabolism.[1] According to the World Health Organization (WHO), the global prevalence of DM continues to rise, with projections estimating that the number of DM patients will reach 570 million by 2030.[2] In China, the burden is similarly high, with adult DM prevalence exceeding 12%, accompanied by insufficient disease control in some patients, leading to a growing incidence of diabetes-related complications.[3]

Among the chronic complications of DM, diabetic peripheral neuropathy (DPN) is one of the most common and debilitating, primarily involving distal symmetric polyneuropathy of the lower limbs.[4,5] Clinically, DPN manifests with sensory symptoms such as numbness, pain, and burning sensations, and in advanced stages, motor deficits and gait instability may occur, significantly increasing the risk of foot ulcers, infections, and even amputations.[6] Approximately 50% of DM patients develop DPN within 20 years of disease onset,[7] highlighting the urgency of early and accurate diagnosis.

However, DPN diagnosis remains a challenge, particularly in its early stages or in patients with atypical or mixed symptoms. This is partly because DPN may involve both small and large nerve fibers. Small fiber neuropathy often presents with pain or dysesthesia but is difficult to detect via standard nerve conduction studies, while large fiber involvement is more readily captured through electrophysiological methods such as electromyography (EMG).[7] The limitations of relying solely on EMG or symptom assessment have led to frequent underdiagnosis of early or mixed-type DPN.[8]

Received: 1 March 2025
Accepted: 17 May 2025
Published: 27 June 2025

While EMG remains a key diagnostic tool that quantitatively evaluates motor and sensory nerve conduction velocities (MCV and SCV), its sensitivity in detecting early or small fiber lesions is limited. Recent studies have suggested that certain clinical and pathological indicators—such as glycated hemoglobin (HbA1c), serum creatinine (Cr), uric acid (UA), cystatin C (Cys-C), and lipid metabolism parameters—may serve as additional biomarkers of peripheral nerve damage in DM patients.[9–11]

Therefore, this study aims to evaluate the diagnostic value of a combined approach using EMG and key clinical pathological indicators for the early detection of DPN. Specifically, we sought to verify the effectiveness of this multi-dimensional diagnostic strategy in a Chinese diabetic population and to explore its potential as a practical diagnostic model for clinical application. By integrating electrophysiological data with metabolic and renal biomarkers, we hope to provide a more comprehensive, sensitive, and practical approach for early identification and intervention in diabetic neuropathy.

Materials and Methods

Clinical Data

A retrospective analysis was conducted on the clinical data of 156 diabetes mellitus (DM) patients admitted to our hospital from July 2022 to December 2024. Inclusion criteria: (1) Diagnosed with type 2 diabetes mellitus (T2DM) according to established diagnostic guidelines;[12] (2) Aged between 18 and 75 years, regardless of gender; (3) Duration of diabetes ≥1 year with a stable diabetes treatment regimen prior to enrollment; (4) Diagnosis of diabetic peripheral neuropathy (DPN) confirmed based on clinical criteria,[13] with typical symptoms and signs present in the lower limbs, although standardized scoring systems such as the Toronto Clinical Neuropathy Score (TCNS) or modified TCNS (mTCNS) were not employed due to the retrospective nature and limitations of available medical records, DPN was diagnosed based on widely accepted clinical and electrophysiological criteria, ensuring diagnostic reliability; (5) Provided written informed consent, with both the patient and their legal guardian (when applicable) agreeing to participate in the study. Exclusion criteria: (1) Exclusion of other types of DM (eg, type 1 diabetes, gestational diabetes mellitus) and non-DM-related neuropathy patients; (2) Exclusion of patients with neuropathy caused by acute cardiovascular and cerebrovascular events, acute infections, or other severe diseases; (3) Exclusion of patients with severe heart, liver, kidney, or other major organ dysfunction; (4) Exclusion of patients with severe mental illness, cognitive disorders, or those who cannot understand the purpose and content of the study; (5) Exclusion of pregnant or breastfeeding women; (6) Exclusion of patients with a history of major surgeries involving the nervous system; (7) Exclusion of patients with severe anemia, malignant tumors, or other diseases affecting nerve function. Patients were divided into group A (n=69, with DPN) and group B (n=87, without DPN) based on the presence of DPN. This study was approved by the Wuhan Fourth Hospital Medical Ethics Committee (Approval No. DMSJ24-0019), informed consent was obtained from all study participants, and the research strictly adhered to the ethical guidelines of the Declaration of Helsinki.

Methods

Baseline Data and Clinical Pathological Parameters

Baseline data included gender, age, body mass index (BMI), DM duration, smoking status, alcohol consumption, and hypertension status, which were collected based on patient self-report and/or historical data. Clinical pathological parameters included 24-hour urine protein (24 h UP), urine albumin/creatinine ratio (A/C), urine microalbumin (UMA), serum albumin (Alb), glycosylated hemoglobin (HbA1c), fasting blood glucose (FBG), 2-hour postprandial blood glucose (2 h PBG), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), triglycerides (TG), total cholesterol (TC), lactate dehydrogenase (LDH), alkaline phosphatase (ALP), aspartate aminotransferase (AST), alanine aminotransferase (ALT), total bilirubin (TBil), cystatin C (Cys-C), uric acid (UA), and creatinine (Cr). Patients' total urine within 24 hours was collected, and 24 h UP and UMA were measured using a urine analyzer. Morning urine samples were collected to measure urine albumin and creatinine concentrations using respective reagent kits, followed by calculation of the A/C ratio. Fasting venous blood (20 mL) was drawn in the morning on admission, centrifuged at 3500 r/min (centrifugal radius 8 cm) for 10 minutes to separate the serum for measurement. Levels of Alb, HbA1c, FBG, 2 h PBG, LDL-C, HDL-C, TG, TC, LDH, ALP, AST, ALT, TBil, Cys-C, UA, and Cr were determined using an automatic biochemical analyzer.

EMG Examination

Data collection was performed using a Keypoint 9033A07electromyograph and evoked potential instrument, produced by Natus Manufacturing Limited. All examinations were conducted in a well-ventilated and appropriately temperature-controlled environment to ensure the accuracy of the instruments and the comfort of the patients. During testing, patients were instructed to lie flat and ensure full body relaxation, avoiding any muscle tension or discomfort that could interfere with the results. For MCV measurements, a saddle-shaped electrode stimulator was used to stimulate the common peroneal nerve along the nerve trunk. The nerve conduction velocity was calculated by analyzing the relationship between the nerve conduction speed and the potential transmission time. Each test point was repeated three times, and the average of the three measurements was taken. SCV was measured using a surface electrode, with the superficial peroneal nerve selected for testing. The sensory nerve's electrical stimulation response and conduction time were recorded, and the SCV value was calculated by combining the sensory nerve's length with the distance from the stimulation point to the recording point. Like MCV testing, SCV tests were also repeated three times, with the final result being the average of the three measurements.

Statistical Analysis

GraphPad Prism 8 was used for graphing, and SPSS25.0 was used for statistical analysis. Count data were expressed as n (%), and Chi-square tests were used for analysis. Normally distributed continuous data were expressed as ($\bar{X} \pm S$), and comparisons between two groups were made using independent-sample t-tests. Multivariate logistic regression was used to analyze the independent factors affecting the occurrence of DPN. The diagnostic performance of the combined model was evaluated through ROC curve analysis. Although no direct comparison with TCNS/mTCNS scoring systems was performed, the obtained area under the curve (AUC) values were interpreted in reference to existing literature benchmarks for these systems, providing a contextual framework for the clinical relevance of our findings. A P-value of <0.05 was considered statistically significant.

Results

Comparison of Baseline Data

The baseline data of gender, age, body mass index (BMI), duration of diabetes mellitus (DM), smoking status, alcohol consumption, and hypertension status between the two groups were compared, showing no significant differences (P > 0.05), indicating comparability. See Table 1.

Table 1 Comparison of Baseline Data ($\bar{X} \pm S$, n[%])

	Group A (n=69)	Group B (n=87)	t/x^2	P
Gender	–	–	0.392	0.531
Male	33 (47.83)	46 (52.87)	–	–
Female	36 (52.17)	41 (47.13)	–	–
Age (years)	55.69±6.84	55.21±6.78	0.437	0.662
BMI (kg/m²)	22.51±1.69	22.65±1.54	0.540	0.589
DM Duration (years)	7.23±1.95	6.74±1.87	1.595	0.112
Smoking Status	–	–	0.093	0.759
Yes	23 (33.33)	27 (31.03)	–	–
No	46 (66.67)	60 (68.97)	–	–
Alcohol Consumption	–	–	0.051	0.820
Yes	25 (36.23)	30 (34.48)	–	–
No	44 (63.77)	57 (65.52)	–	–
Hypertension Status	–	–	0.116	0.732
Yes	24 (34.78)	28 (32.18)	–	–
No	45 (65.22)	59 (67.82)	–	–

Abbreviations: BMI, Body Mass Index; DM, Diabetes Mellitus.

Comparison of Clinical Pathological Parameters

The HbA1c, Cys-C, UA, and Cr levels in Group A were higher than those in Group B ($P < 0.05$). The other parameters showed no significant differences ($P > 0.05$). See Table 2.

Comparison of EMG Indicators

The MCV and SCV values in Group A were (42.17 ± 5.08, 40.06 ± 5.03), and in Group B, the values were (46.78 ± 5.16, 46.35 ± 5.34). The MCV and SCV levels in Group A were significantly lower than those in Group B ($P < 0.05$). See Figure 1.

Multivariate Logistic Regression Analysis of Factors Affecting the Occurrence of DPN in DM Patients

The occurrence of DPN in DM patients (0 = no, 1 = yes) was used as the dependent variable. The potential influencing factors obtained from Tables 1 and 2, and Figure 1 were assigned values as independent variables (see Table 3), and a multivariate logistic regression model was established. The results showed that HbA1c, Cys-C, UA, Cr, MCV, and SCV were independent risk factors for DPN in DM patients ($P < 0.05$), as shown in Table 4.

Value of Combined Diagnosis of DPN Using EMG and Clinical Pathological Parameters

ROC curve analysis showed that the AUC for HbA1c, Cys-C, UA, Cr, MCV, SCV, and combined diagnosis were 0.743, 0.727, 0.712, 0.709, 0.784, 0.769, and 0.873, respectively. The AUC, sensitivity, and specificity for combined diagnosis were higher than those for any individual diagnosis, as shown in Table 5 and Figure 2.

Discussion

DPN is a prevalent and debilitating complication of DM, and its early detection is critical to preventing irreversible nerve damage and improving patient outcomes.[14] Despite this importance, current diagnostic methods often face limitations in

Table 2 Comparison of Clinical Pathological Parameters ($\bar{X} \pm S$)

	Group A (n=69)	Group B (n=87)	*t*	*P*
24 h UP (g/24 h)	0.27±0.06	0.25±0.07	1.886	0.061
A/C (mg/mmol)	5.72±1.34	5.48±1.36	1.101	0.272
UMA (mg/L)	41.03±6.45	39.35±5.43	1.765	0.079
Alb (g/L)	42.57±3.22	42.93±2.95	0.726	0.468
HbA1c (%)	8.67±2.52	7.09±1.36	5.003	<0.001
FBG (mmol/L)	8.33±1.54	8.04±1.37	1.242	0.215
2 h PBG (mmol/L)	14.27±2.56	13.85±2.433	1.047	0.296
LDL-C (mmol/L)	2.55±0.42	2.58±0.38	0.467	0.640
HDL-C (mmol/L)	1.07±0.26	1.04±0.25	0.731	0.465
TG (mmol/L)	2.08±0.34	1.99±0.31	1.725	0.086
TC (mmol/L)	4.36±0.85	4.24±0.93	0.831	0.407
LDH (U/L)	172.83±22.48	168.47±21.96	1.218	0.224
ALP (U/L)	72.39±11.27	70.52±19.08	0.720	0.472
AST (U/L)	19.64±3.25	19.98±3.31	0.642	0.521
ALT (U/L)	23.17±3.22	23.72±4.65	0.836	0.404
TBil (μmol/L)	13.19±2.21	13.06±2.14	0.371	0.710
Cys-C (mg/L)	1.16±0.38	0.91±0.15	5.613	<0.001
UA (mmol/L)	337.69±53.52	293.13±45.36	5.626	<0.001
Cr (μmol/L)	78.36±16.15	63.69±14.92	5.880	<0.001

Abbreviations: 24 h UP, 24-hour urine protein; A/C, urine albumin/creatinine ratio; UMA, urine microalbumin; Alb, serum albumin; HbA1c, glycated hemoglobin; FBG, fasting blood glucose; 2 h PBG, 2-hour postprandial blood glucose; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; TG, triglycerides; TC, total cholesterol; LDH, lactate dehydrogenase; ALP, alkaline phosphatase; AST, aspartate aminotransferase; ALT, alanine aminotransferase; TBil, total bilirubin; Cys-C, cystatin C; UA, uric acid; Cr, creatinine.

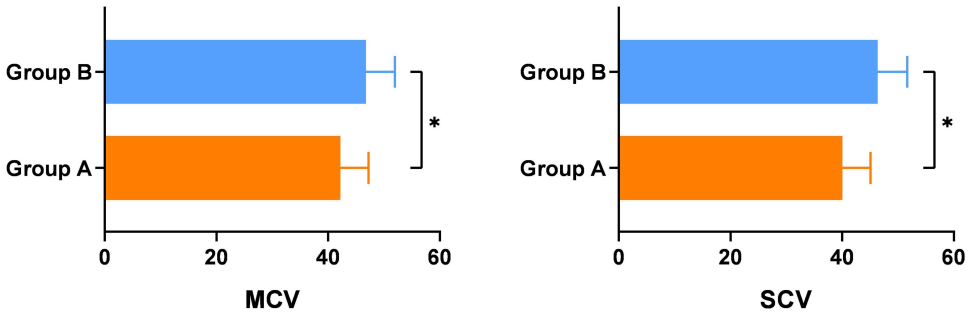

Figure 1 Comparison of EMG Indicators ($\bar{X} \pm S$, m/s).
Note: intergroup comparison, *P < 0.05.
Abbreviations: MCV, Motor nerve conduction velocity; SCV, sensory nerve conduction velocity.

sensitivity and comprehensiveness. In this context, the present study evaluated the diagnostic value of combining EMG with clinical pathological parameters for the early detection of DPN in patients with T2DM.

EMG, particularly through assessments of motor and sensory nerve conduction velocity, remains a fundamental tool in evaluating peripheral nerve function in DM patients.[15] In this study, reduced motor and sensory nerve conduction velocities were observed in patients with DPN compared to those without, aligning with previous reports that nerve conduction slowing is an early manifestation of neuropathy.[16–18] Notably, the involvement of the common and superficial peroneal nerves is recognized as a typical feature of DPN.[19] While EMG provides valuable electrophysiological insight, its diagnostic accuracy can be limited by variability in lesion distribution and examination conditions. Therefore, integrating EMG with relevant biochemical and clinical parameters may enhance its diagnostic performance.

Several studies have emphasized the role of clinical pathological factors in the development of DPN.[20–23] Among them, glycemic control is a critical determinant. Chronic hyperglycemia contributes to nerve damage via oxidative stress, vascular endothelial injury, and other metabolic pathways.[24,25] This study supported previous findings by identifying

Table 3 Variable Assignment Table

Independent Variable	Assignment Method
HbAlc	Original value
Cys-C	Original value
UA	Original value
Cr	Original value
MCV	Original value
SCV	Original value

Table 4 Multivariate Logistic Regression Analysis of Factors Affecting the Occurrence of DPN in DM Patients

Factor	*β*	*SE*	*Wald x²*	*P*	*OR*	*95% CI*
HbAlc	0.303	0.146	4.587	<0.05	1.351	1.024–1.788
Cys-C	0.318	0.171	4.193	<0.05	1.349	1.008–1.854
UA	0.456	0.213	7.654	<0.001	1.345	1.017–1.952
Cr	0.238	0.139	4.175	<0.05	1.263	1.032–1.638
MCV	0.437	0.165	6.874	<0.05	1.536	1.112–2.107
SCV	0.514	0.158	9.511	<0.05	1.647	1.192–2.385

Abbreviations: HbA1c, Glycated hemoglobin; Cys-C, Cystatin C; UA, Uric acid; Cr, Creatinine; MCV, Motor nerve conduction velocity; SCV, Sensory nerve conduction velocity.

https://doi.org/10.2147/JMDH.S525731

Table 5 Value of Combined Diagnosis of DPN Using EMG and Clinical Pathological Parameters

Indicator	Optimal Cutoff Value	AUC	95% CI	P	Sensitivity (%)	Specificity (%)
HbAlc	8.09%	0.743	0.705–0.812	<0.05	75.97	70.36
Cys-C	1.02 mg/L	0.727	0.674–0.797	<0.05	73.52	71.45
UA	307.62 mmol/L	0.712	0.653–0.785	<0.05	69.27	74.16
Cr	70.48 μmol/L	0.709	0.648–0.782	<0.05	76.73	67.35
MCV	45.29 m/s	0.784	0.734–0.856	<0.05	74.21	70.82
SCV	42.47 m/s	0.769	0.727–0.843	<0.05	76.49	68.56
Combined	-	0.873	0.805–0.944	<0.05	83.24	78.39

Abbreviations: HbA1c, Glycated hemoglobin; Cys-C, Cystatin C; UA, Uric acid; Cr, Creatinine; MCV, Motor nerve conduction velocity; SCV, Sensory nerve conduction velocity.

elevated HbA1c levels as an independent risk factor for DPN, highlighting its utility in risk stratification and early warning systems for neuropathic complications.

In addition to glycemic markers, renal dysfunction also emerged as a relevant contributor. The elevated levels of Cys-C, UA, and Cr in DPN patients suggest a possible link between impaired kidney function and neuropathy. Prior research has suggested that reduced renal clearance may promote nerve injury through mechanisms such as impaired nerve perfusion and increased oxidative stress.[26–28] These findings underscore the potential utility of renal function indicators in identifying high-risk diabetic individuals. Future studies may further explore the temporal association and predictive value of these markers in the context of DPN progression.

Contrary to some previous reports, this study did not find significant differences in lipid metabolism indicators between patients with and without DPN. This discrepancy may be attributable to differences in sample characteristics or study design.[29,31] Nevertheless, the influence of lipid dysregulation on nerve integrity—through mechanisms such as altered membrane structure and heightened inflammatory activity—remains biologically plausible and warrants continued investigation.[31]

In this study, the combined diagnostic model using EMG and clinical pathological parameters demonstrated significantly superior performance compared to either method alone, as evidenced by the area under the AUC. This result suggests a synergistic effect that enhances both sensitivity and specificity in diagnosing DPN. By incorporating electrophysiological findings and systemic metabolic indicators, clinicians can achieve a more comprehensive evaluation of neuropathic risk in patients with T2DM. Furthermore, this multidimensional diagnostic approach is feasible in clinical settings and could be integrated into standard diabetes management protocols to improve early screening efforts, enabling the early identification of high-risk DPN patients and timely intervention. By establishing an appropriate "high-risk"

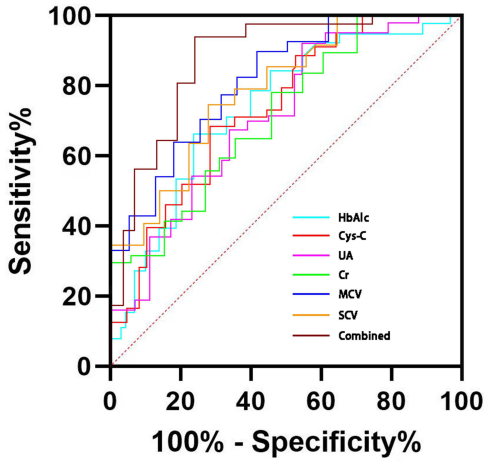

Figure 2 ROC Curve for Combined Diagnosis of DPN Using EMG and Clinical Pathological Parameters.
Abbreviations: HbA1c, Glycated hemoglobin; Cys-C, Cystatin C; UA, Uric acid; Cr, Creatinine; MCV, Motor nerve conduction velocity; SCV, Sensory nerve conduction velocity.

cutoff value, clinicians can better identify high-risk patients in practice and tailor individualized treatment plans. Although this study did not directly compare the combined diagnostic model with existing diagnostic standards, such as TCNS or mTCNS, based on the results of this study, the model may potentially serve as an auxiliary tool to be used in conjunction with current standards in the future to further enhance the diagnostic accuracy of DPN.

Despite its strengths, this study has several limitations. It was a single-center retrospective analysis with a relatively small sample size, which may limit the generalizability of the findings. Prospective, multi-center studies with larger cohorts are necessary to validate the broader applicability of the combined diagnostic model. Additionally, this study focused on a limited set of EMG and laboratory indicators, without incorporating emerging biomarkers such as inflammatory cytokines or advanced imaging techniques. Future research should explore a broader range of diagnostic tools to improve the precision of DPN screening. Furthermore, although the combined diagnostic approach shows promise, its cost-effectiveness and long-term clinical benefits should be evaluated in real-world settings to guide implementation in diverse healthcare environments.

In summary, this study provides preliminary evidence for the potential value of combining EMG with clinical pathological parameters—particularly HbA1c, Cys-C, UA, and Cr—in the early diagnosis of DPN. This integrated approach offers a more comprehensive assessment of neuropathic risk in DM patients and may support more timely and targeted interventions. With further validation, such diagnostic strategies may improve the clinical management and prognosis of individuals with or at risk for DPN.

Disclosure

The authors report no conflicts of interest in this work.

References

1. Zhu XR, Zhang F, Li Y, et al. Prevalence and associated factors of diabetic retinopathy among type 2 diabetes in Dongcheng District and Tongzhou District, Beijing City: a cross-sectional study. *Zhonghua Yu Fang Yi Xue Za Zhi*. 2024;58(9):1324–1330. doi:10.3760/cma.j.cn112150-20240222-00136
2. Zhang SJ, Li Y, Zhou M, et al. Application progress of implementation research on diabetes management. *Zhonghua Yu Fang Yi Xue Za Zhi*. 2023;57(10):1682–1686. doi:10.3760/cma.j.cn112150-20221105-01071
3. Zhang W, Sun H, Liu L, et al. Visualization analysis of research hotspots in pathogenesis of diabetic nephropathy in China. *Zhonghua Yu Fang Yi Xue Za Zhi*. 2023;57(7):1075–1081. doi:10.3760/cma.j.cn112150-20230119-00050
4. Shu Y, Huang Y, Liu J, et al. Research progress on exosomes and exosomal microRNAs in the occurrence and development of diabetic peripheral neuropathy. *Zhonghua Yu Fang Yi Xue Za Zhi*. 2024;58(1):141–147. doi:10.3760/cma.j.cn112150-20230308-00176
5. Lian X, Liu Y, Wang H, et al. Clinical effect of acupuncture combined with medication on diabetic peripheral neuropathy and its influence on serum lipid metabolism. *Zhongguo Zhen Jiu*. 2024;44(5):503–512. doi:10.13703/j.0255-2930.20230711-k0007
6. Avetisov SE, Minasyan YA, Astakhov YS, Akhmedzhanova LT, Fokina AS, Strokov IA. Early diagnosis of diabetic polyneuropathy based on the results of corneal nerve fibers examination. *Vestn Oftalmol*. 2020;136(5 Suppl 2):155–162. doi:10.17116/oftalma2020136052155
7. Chiaramonte R, Romano M, Vecchio M. A systematic review of the diagnostic methods of small fiber neuropathies in rehabilitation. *Diagnostics*. 2020;10(9):613. doi:10.3390/diagnostics10090613
8. Tai HF, Zhang YL, Yang JY, et al. Clinical and neuroelectrophysiological characteristics of primary peripheral nerve hyperexcitability syndrome. *Zhonghua Yi Xue Za Zhi*. 2023;103(25):1925–1930. doi:10.3760/cma.j.cn112137-20230303-00320
9. Moon HI, Lee JW, Kim DW, et al. Diabetic polyneuropathy and the risk of developing carpal tunnel syndrome: a nationwide, population-based study. *Muscle Nerve*. 2020;62(2):208–213. doi:10.1002/mus.26901
10. Song L, Wang Y, Zhao H, et al. Sphingolipid metabolism plays a key role in diabetic peripheral neuropathy. *Metabolomics*. 2022;18(6):32. doi:10.1007/s11306-022-01879-7
11. Chen M, Zhang Y, Wang C, et al. The predictive value of neutrophil-to-lymphocyte ratio and platelet-to-lymphocyte ratio levels of diabetic peripheral neuropathy. *J Pain Res*. 2021;14:2049–2058. doi:10.2147/JPR.S304595
12. Doupis J, Horton ES. Utilizing the new glucometrics: a practical guide to ambulatory glucose profile interpretation. *touchREV Endocrinol*. 2022;18(1):20–26. doi:10.17925/EE.2022.18.1.20
13. Meng Y, Liu J, Chen H, et al. Artificial intelligence based analysis of corneal confocal microscopy images for diagnosing peripheral neuropathy: a binary classification model. *J Clin Med*. 2023;12(4):943. doi:10.3390/jcm12041284
14. Savelieff MG, Feldman EL, Viswanathan V, Jensen TS, Bennett DL, Feldman EL. The global and regional burden of diabetic peripheral neuropathy. *Nat Rev Neurol*. 2025;21(1):17–31. doi:10.1038/s41582-024-01041-y
15. Ashoori M, Nikbakht M, Faraji E, et al. The effectiveness of neurodynamic techniques in patients with diabetic peripheral neuropathy: study protocol for a randomized sham-controlled trial. *Adv Biomed Res*. 2024;13:6. doi:10.4103/abr.abr_180_23
16. Dong D, Liu H. Prevalence of carpal tunnel syndrome in patients with long-term type 2 diabetes mellitus. *Heliyon*. 2022;8(12):e12615. doi:10.1016/j.heliyon.2022.e12615
17. Xiong F, Zhang J, Liu H, et al. Ultrasound characteristics of the cervical vagus nerve in patients with type 2 diabetes and diabetic peripheral neuropathy. *Endokrynol Pol*. 2023;74(5):395–401.

18. Zhang Y, Zhou X, Wu J, et al. Association of sarcopenia and muscle mass with both peripheral neuropathy and nerve function in patients with type 2 diabetes. *Diabet Res Clin Pract*. 2020;162:108096. doi:10.1016/j.diabres.2020.108096
19. Wang Z, Chen Q, Xu J, et al. Clinical efficacy and safety of electro-acupuncture combined with beraprost sodium and α-lipoic acid for diabetic peripheral neuropathy. *Am J Transl Res*. 2022;14(1):612–622.
20. Zheng YH, Wang Y, Lu X, et al. A cross-sectional study on the correlation between inflammatory cytokines, negative emotions, and onset of peripheral neuropathy in type 2 diabetes. *Neuropsychiatr Dis Treat*. 2020;16:2881–2890. doi:10.2147/NDT.S278439
21. Zhong J, Zhao W, Feng M, et al. Metabolomics study of serum from patients with type 2 diabetes: peripheral neuropathy could be associated with sphingosine and phospholipid molecules. *Lipids*. 2025;60(1):3–13. doi:10.1002/lipd.12412
22. Brask-Thomsen PK, Lauridsen CA, Nielsen J, et al. Development and progression of polyneuropathy over 5 years in patients with type 2 diabetes. *Neurology*. 2024;103(3):e209652. doi:10.1212/WNL.0000000000209652
23. Zhu J, Zhang Q, Chen L, et al. Diabetic peripheral neuropathy: pathogenetic mechanisms and treatment. *Front Endocrinol*. 2023;14:1265372. doi:10.3389/fendo.2023.1265372
24. Firouzabadi MD, Vahdatpour B, Nili S, et al. Glycemic profile variability: an independent risk factor for diabetic neuropathy in patients with type 2 diabetes. *Prim Care Diabetes*. 2023;17(1):38–42. doi:10.1016/j.pcd.2022.11.011
25. Wang CS, Li H, Xu J, et al. Diabetic peripheral neuropathy: age-stratified glycemic control. *Front Endocrinol*. 2024;15:1377923. doi:10.3389/fendo.2024.1377923
26. Sinacore DR, Jones MA, Kline PW. Major adverse foot events and functional mobility deficits associated with diabetic neuropathy and nephropathy. *Metab Target Organ Damage*. 2024;4(2):e00095. doi:10.20517/mtod.2024.02
27. Hong X, Wang Y, Zhou H, et al. Stronger association of albuminuria with the risk of vascular complications than estimated glomerular filtration rate in type 2 diabetes. *Kidney Blood Press Res*. 2021;46(5):550–562. doi:10.1159/000515163
28. Li X, Xu D, Li Y, et al. Uric acid in diabetic microvascular complications: mechanisms and therapy. *J Diabetes Complications*. 2025;39(2):108929. doi:10.1016/j.jdiacomp.2024.108929
29. Chang KC, Lin HY, Chiang ST, et al. The association between hyperlipidemia, lipid-lowering drugs and diabetic peripheral neuropathy in patients with type 2 diabetes mellitus. *PLoS One*. 2023;18(6):e0287373. doi:10.1371/journal.pone.0287373
30. Zhong M, Gao X, Li J, et al. Change in urine albumin-to-creatinine ratio and risk of diabetic peripheral neuropathy in type 2 diabetes: a retrospective cohort study. *Diabetes Metab Syndr Obes*. 2021;14:1763–1772. doi:10.2147/DMSO.S303096
31. Stino AM, Smith AG, Singleton JR, et al. Evolving concepts on the role of dyslipidemia, bioenergetics, and inflammation in the pathogenesis and treatment of diabetic peripheral neuropathy. *J Peripher Nerv Syst*. 2020;25(2):76–84. doi:10.1111/jns.12387