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Silent diabetic peripheral neuropathy and symptom-examination discordance among Malaysian adults with type 2 diabetes mellitus: Findings from the Seremban Diabetes Cohort

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Abstract

Aims

To determine the burden of silent diabetic peripheral neuropathy (DPN), quantify symptom-examination discordance, and evaluate the diagnostic performance of symptom severity among Malaysian adults with type 2 diabetes mellitus (DM).

Methods

Eligible adults with type 2 DM recruited into the Seremban Diabetes Cohort underwent standardized neurological examinations including 10g monofilament, Ipswich touch test, vibration, pinprick and ankle reflex assessments. Neuropathic symptoms were quantified using an exploratory 0–18 ordinal study-specific severity score meant specifically for this study. Receiver operating characteristics (ROC), Youden index, threshold performance, and discordance analyses were performed.

Results

From 773 participants, 120 (15.5%) had examination-confirmed DPN. Symptom severity exhibited modest discriminatory performance (AUC = 0.64). The optimal severity threshold (> 8) exhibited a sensitivity of 42.2% and specificity of 84.4%. Increasing the severity threshold increased the specificity at the expense of sensitivity. Discordance analysis showed 44.8% of neurologically confirmed DPN patients were asymptomatic or minimally symptomatic while 25.0% of patients complained of neuropathic symptoms without overt clinical signs of DPN.

Conclusion

Almost half of clinically confirmed DPN were asymptomatic or minimally symptomatic, limiting the sensitivity of symptom-based screening alone. Standardized clinical and ancillary examinations should complement symptom assessment to improve early detection of DPN.

1. Introduction

Diabetic peripheral neuropathy (DPN) has been defined by the Americal Diabetes Association (ADA) as “the presence of symptoms and/or signs of peripheral nerve dysfunction in people with diabetes after exclusion of other causes” [1]. It remains the commonest complications affecting more than 30% of Type 2 DM patients with the prevalence rate increasing with diabetes duration as noted in a recent systematic review on global prevalence of diabetes-related neuropathy and vascular complications [2]. Not only does it impose risks to affected individuals, but it also renders significant cost to the healthcare system and economic structure [1].

Symptomatology plays an important role in alerting patients and physicians to the possibility of neurological complications. However, symptoms alone are unreliable as there is no clear overlap between symptoms and examination confirmed DPN [3]. Some patients present with significant symptoms without clinically objective DPN [4, 5]. More alarmingly, patients might develop clinical DPN without overt symptomatology, a silent PDN, by as high as 50% of the population [6].

Tesfaye et al regarded the existence of neurological abnormalities without discernible symptoms or signs as subclinical DPN [7]. This silent condition is worrisome since patients are unknowingly at increased risk of repeated foot injuries, ulceration and even amputations [1]. The true burden of DPN may be underrepresented because of this proportion of patients [8].

Despite increasing recognition that many patients with DPN are asymptomatic, the degree of discordance between patient-reported symptoms and objective neurological examination is incompletely characterised, particularly in Southeast Asian populations. We therefore investigated this burden of silent DPN, quantified the discordance between symptoms and examination findings, and evaluated the diagnostic performance of symptom severity.

2. Methods

2.1 Subjects

Subjects were recruited from the Seremban Diabetes Cohort (SeDia) recruitment clinic between 01 January 2024 and 31 December 2025 using consecutive sampling. Inclusion criteria comprised adults aged ≥ 18 years with Type 2 DM who were able to participate in foot examinations. Patients with pre-existing neurological disorders, such as Parkinson disease and cerebrovascular accidents, as well as those with major lower limb amputations were excluded from this study.

2.2 Study design

This study was conducted within an existing cohort recruitment framework; therefore, formal a priori sample size calculation was not performed. The full SeDia protocol may be accessed at https://sedia.my. [9] Baseline patient characteristics consisting of age, sex, duration of DM, and body mass index (BMI) were extracted from the SeDia patient database. Participants were asked regarding neuropathic symptoms of the feet such as numbness, tingling, pain, temperature sensation, etc. An exploratory study-specific symptom severity scoring approach was used to quantify participant-reported neuropathic symptoms. Participants were asked to rate overall neuropathic symptoms for each foot using an ordinal Likert scale ranging from 0 to 9, where higher score indicated greater symptom severity. Scores from both feet were summed to generate a total symptoms severity score ranging from 0 to 18.

Neurological examinations incorporated multiple validated bedside tests recommended for the assessment of DPN, consisting of 10g monofilament examination, Ipswich touch test, tuning fork vibratory sensation, pinprick examination, and ankle reflexes [10, 11]. Because no single bedside

examination demonstrates perfect diagnostic accuracy, DPN patient classification was based on a composite assessment integrating abnormalities across complementary neurological modalities. Participants were classified as having DPN according to the SeDia clinical assessment protocol if they demonstrated either abnormal monofilament testing, abnormal Ipswich Touch Test findings, or concurrent abnormalities in vibration, pinprick sensation and ankle reflex.

2.3 Data analysis

The total symptom severity score (0–18) was analysed as a continuous variable for primary diagnostic performance analysis. Data management and statistical analyses were performed on Microsoft 365 Excel using complete-case data for the variables of interest. Participants with missing symptom severity scores or incomplete DPN assessments were excluded from analyses requiring both variables. The relationship between neuropathic symptoms and examination-confirmed DPN was evaluated using complementary analytical approaches. Discordance analysis was performed to quantify the agreement between symptom status and objective neurological findings. The diagnostic performance of the study-specific symptom severity score was then assessed using receiver operating characteristic (ROC) analysis with estimation of the area under the curve (AUC), followed by Youden index and threshold performance analyses. Baseline characteristics were tabulated accordingly. Normally distributed continuous variables were compared using independent-samples t-test, whereas non-normally distributed continuous variables were compared using the Mann-Whitney U-test and presented as median (interquartile range). Categorical variables were compared using Pearson's chi-square test. A p-value of < 0.05 was considered as statistically significant.

3. Results

A total of 773 participants with Type 2 DM were included and analysed, of which 120 (15.5%) met the examination criteria for DPN. (Table 1)

Table 1
Baseline characteristic of participants according to diabetic peripheral neuropathy status.

Variable	Total cohort	DPN	Non-DPN	p-value
Total participants	773	120	653	
Age (years)	61.4 ± 10.9	65.4 ± 9.6	60.0 ± 11.0	< 0.001
Female sex (n, %)	429 (55.5)	54 (45.0)	375 (57.4)	0.016
DM duration (years)	11.8 ± 8.9	16.3 ± 10.2	11.0 ± 8.4	< 0.001
BMI (kg/m^2)	28.3 ± 5.4	27.8 ± 4.7	28.4 ± 5.5	0.249
Symptom severity score (Interquartile range)	0 (0–6)	4.5 (0–10)	0 (0–4)	< 0.001

Continuous variables are presented as mean ± standard deviation. Categorical variables are presented as frequencies and percentages. Symptom score presented as median (interquartile range) and comparison performed using Mann-Whitney U test. DPN – diabetic peripheral neuropathy. BMI – body mass index.

Patients with DPN were significantly older, had a longer duration of DM and demonstrated a higher symptom burden than those without DPN, whereas BMI did not differ significantly between groups.

To further evaluate the ability of symptom severity to discriminate between participants with and without DPN identified on clinical examination, an ROC analysis was performed. It demonstrated modest discriminatory performance of symptom severity scores for detecting DPN, with area under curve (AUC) of 0.64.

The ROC curve lay only modestly above the line of no discrimination, indicating that symptom severity alone provided limited ability to distinguish participants with and without neurologically confirmed DPN.

The optimal discriminatory threshold was explored using Youden Index analysis, which was found to be 0.267, corresponded to a severity cutoff point of 8, providing a sensitivity of 42.2% and specificity of 84.4%. A threshold performance analysis was also performed (Table 2).

Table 2
Diagnostic performance of selected symptom severity score thresholds for detecting diabetic peripheral neuropathy.

Symptom score cutoff	Sensitivity (%)	Specificity (%)	Clinical interpretation
≥ 4	55.2	70.5	Sensitivity-prioritized threshold
≥ 5	50.0	75.9	Balanced threshold
≥ 8	42.2	84.4	Highest overall discriminatory threshold (Youden index – see above)
≥ 10	32.8	88.7	Specificity-prioritized threshold

Although a higher symptoms threshold improved specificity, this occurred at the expense of progressively lower sensitivity, suggesting that increasing symptom burden alone does not adequately identify many individuals with DPN diagnosed clinically.

Finally, a discordance analysis (Table 3) was performed in the 747 participants with complete symptom severity and DPN assessment data. Twenty-six participants were excluded because of incomplete symptom severity data required for discordance analysis.

Table 3
Concordance and discordance between symptom status and objective diabetic peripheral neuropathy.

Classification	n, (% of total cohort)
True positive (TP)	64 (8.6)
True negative (TN)	444 (59.4)
Silent DPN	52 (7.0)
Symptomatic non-DPN	187 (25.0)

The analysis showed that among the 116 participants with neurologically confirmed DPN, 44.8% were asymptomatic or minimally symptomatic. Additionally, 187 participants reported neuropathic symptoms despite the absence of objective DPN. These findings demonstrate a considerable discordance between patient -reported symptom burden and objective clinical findings, with a substantial proportion of DPN cases remaining clinically silent despite abnormal clinical examination results.

4. Discussion

Nearly one in two patients with examination-confirmed DPN were either asymptomatic or minimally symptomatic. This finding has important clinical implications for DM screening because reliance on symptom enquiry alone may delay recognition of DPN in a substantial proportion of patients. This burden of silent DPN explains the modest diagnostic performance observed in our study, where symptom severity demonstrated an AUC of 0.64 and a sensitivity of only 42.2% at the optimal threshold. Additionally, the overlap in symptom severity between participants with and without DPN suggests that symptom burden alone cannot reliably distinguish individuals with clinically confirmed neuropathy.

Neuropathic symptoms appear heterogeneous [6, 12] extending beyond pain alone, encompassing numbness, altered temperature perception and other sensory disturbances, and may not appear despite the duration of DM [7, 13]. Of 1119 Type 2 DM patients that Maatta et al [14] evaluated over a 13-year period, 40% of their participants never experienced neuropathic symptoms and of the 150 patients who were diagnosed with DPN, 49.7% were asymptomatic, a proportion that is similar to our findings. Sorenson et al quoted an even higher proportion of patients (88.3%) with insensate feet who presented with no symptoms [5]. In a retrospective review of 520 patients, Gao et al found almost 60% of their cohort to be asymptomatic based on the Diabetic Neuropathy Symptoms score (DNS = 0) and of that number, 45.7% of them had subclinical DPN based on an abnormal NCS (polyneuropathy 54,2%, mononeuropathy 37.3%, mononeuropathy multiplex 14.1%). Thus, more than one in four patients (142/520 = 27.3%) would have been missed if screening was symptom-based alone [13]. Unmar et al found the prevalence rate of subclinical DPN to be 17.1%, associating the condition with age, height, diabetic control, and the presence of vasculopathy [15]. This led some authors to suggest that symptoms alone have a poor diagnostic accuracy in cross-sectional studies [14].

Within our local context, Ping et al evaluated 425 patients in 2017 and found almost half of the cohort had undiagnosed DPN using the neuropathy symptom score (NSS) and only 0.2% of them had DPN on clinical examination [16], therefore placing more emphasis on symptom identification. However, their study primarily focused on symptomatic neuropathy and therefore did not specifically evaluate the burden of silent DPN.

Previous studies have attempted to categorize DPN symptom severity, and most were related to pain [7, 17, 18]. However, we found that our patients were disturbed by other symptoms as well, such as numbness and cold. Furthermore, some patients have difficulty expressing the exact sensation (or lack thereof) they experience, complicating symptom identification [17, 19]. These observations support the use of an ordinal symptom severity approach that captures the overall symptom burden rather than relying solely on a binary symptom classification. The lack of a standardized symptom categorization appears to support this [20], and our findings demonstrate that a substantial proportion of patients with examination confirmed DPN were either asymptomatic or minimally symptomatic, emphasizing the inherent limitations of symptom-based assessment.

The observed discordance that we noted is plausible. Although DPN results from progressive peripheral nerve injuries from multiple fronts [3, 7, 21], the relationship between structural damage and symptom perception is complex and incompletely understood. Some patients may develop bedside neurological examination abnormalities before clinically significant symptoms become apparent, while others may experience symptoms without clinical neurological deficit. This heterogeneity may be due to differences in extent and distribution of nerve fibre involvement, individual perception, and the multifactorial pathophysiology of DPN. Although some authors have described the progressive loss of the different sensations with increasing duration of DM [8], the exact temporal sequence of small- and large sensory fibre involvement is not completely understood [14, 22]. Consequently, neurological abnormalities identified during examinations may not align with patient-reported symptom burden. Our findings reinforce the concept that symptoms and neurological examinations should be viewed as complementary rather than interchangeable components of DPN assessment which might include the use of neurophysiological testing [8, 22, 23]. Litoriya et al noted that asymptomatic patients exhibited abnormal nerve conduction parameters compared to healthy subjects [24], underscoring the usefulness of early NCS and other point of care rapid tests for DPN detection [25, 26].

In many low- and middle-income countries, DPN screening is frequently limited by resource availability, clinic workload and access to specialized investigations. Symptom enquiry therefore remains an important first-line assessment. Our findings suggest that symptom-based screening alone may substantially underestimate DPN burden and support routine incorporation of standardized neurological examination into diabetes care pathways [27, 28], [29]. Even among established physicians, accurate diagnosis of DPN may be variable using symptoms and signs either alone or in combination [30].

This study has several strengths. Firstly, symptom burden was quantified using an ordinal severity approach rather than simple symptom presence or absence thus allowing evaluation across multiple

clinically relevant thresholds. Secondly, diagnostic performance was comprehensively assessed using ROC, Youden index and threshold analyses. Thirdly, discordance analysis extended conventional diagnostic performance metrics by demonstrating the substantial burden of silent DPN underlying the modest sensitivity of symptom-based assessments. By integrating ROC analysis with discordance analysis, this study provides an explanation for the limited sensitivity of symptom-based screening that is not apparent from diagnostic performance metrics alone. Finally, the relatively large, consecutively recruited cohort reflects clinical practice and enhance the applicability of our findings.

We acknowledge some limitations of our study. Participants were volunteers from a cohort recruitment clinic and may not fully represent the broader diabetic population, and because of the cross-sectional design of the study, we could not establish temporal relationships between symptom development and neurological abnormalities. Additionally, DPN classification was based on a composite neurological examination instead of formal neurophysiological testing, which may have resulted in some misclassification of neuropathy status. The symptom severity score used in this study was study-specific and exploratory in nature, and formal psychometric validation was not performed.

5. Conclusions

Although neuropathic symptom might indicate an increase likelihood of DPN, many patients remain asymptomatic despite clinical confirmed neuropathy rendering them at risk of delayed diagnosis and preventive care. Symptom-based screening alone demonstrated limited sensitivity because a substantial proportion of patients with neurologically diagnosed DPN were asymptomatic or minimally symptomatic. A structured and robust neurological examination should therefore complement symptom assessment during routine screening.

Declarations

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Ethical approval

This study was performed in accordance with the ethical principles outlined in the Declaration of Helsinki. Institutional ethical approval was obtained from the Malaysian Medical Research and Ethical Committee (MREC 22-01568-0D2) and IMU University Joint Committee for Research and Ethics (IMU R 228-2022). Each patient provided written consent for participation in the study.

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Conflicts of Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Declaration of generative AI and AI-assisted technologies in the manuscript preparation process

During the preparation of this work, the authors used AI (ChatGPT) was used for proofreading and language editing of the manuscript to improve readability. After using this tool, the authors reviewed and edited the content as needed and take full responsibility for the content of the published article.

Availability of data and materials

Data and materials relating to this study can be made available upon request and release from data manager of Seremban Diabetes Cohort.

CRediT authorship contribution statement

Zairul-Nizam ZF: conceptualization, data acquisition, data analysis and curation, writing – original draft and editing, final approval. Sulaiman LH: conceptualization, project administration and funding acquisition, supervision, writing – review and editing, final approval. Zakaria ZF: project administration, data analysis, writing – review and editing, final approval.

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Figures

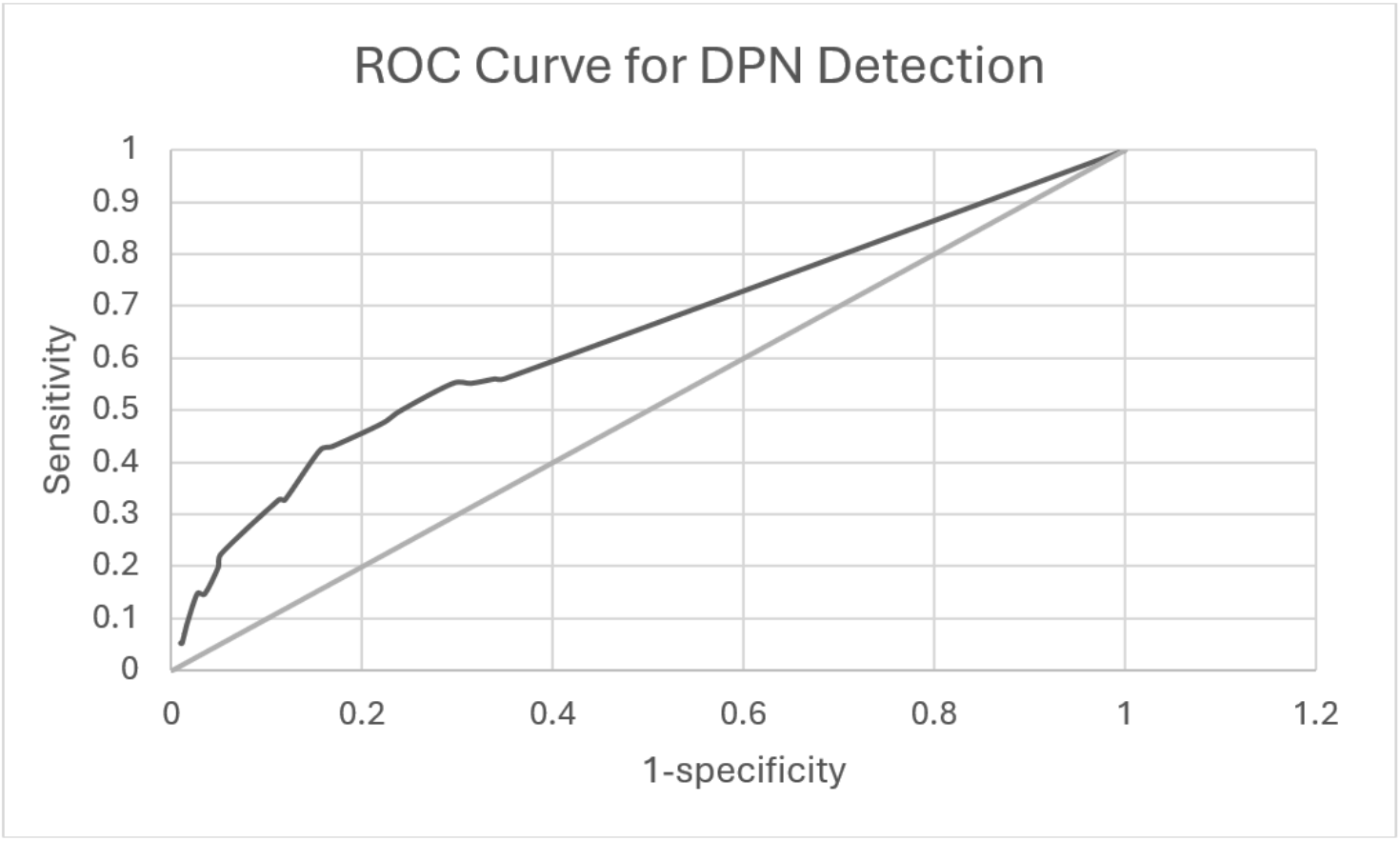


Figure 1

Receiver operating characteristics (ROC) curve of symptom severity score for detecting diabetic peripheral neuropathy.