Research article

Magnitude and associated factors of peripheral neuropathy among diabetes patients attending Jimma University Medical Center, Southwest Ethiopia

Daba Abdissa [*], Rebuma Sorsa, Asfaw Gerbi, Nigusse Hamba, Zelalem Banjaw

Department of Biomedical Sciences, College of Medical Sciences, Institute of Health Science, Jimma University, Oromia, Ethiopia

ARTICLE INFO

ABSTRACT

Background: Chronic complications of diabetes (DM) are a major cause of mortality and morbidity. Of these, diabetic peripheral neuropathy (DPN) is the most common. Screening using validated tools for DPN is crucial to prevent consequent complications. One of the useful tools for DPN screening in clinical practice is the Michigan Neuropathy Screening Instrument (MNSI). However, there is limited information on DPN in the study area. Hence, the aim of this study was to assess the prevalence of DPN and its determinants among patients with type one DM (T1DM) attending Jimma University Medical Center (JUMC) from January 2 to March 31, 2020.
Methods: An institution based cross-sectional study was conducted and DPN was assessed using MNSI. Data were collected using pretested structured questionnaire and entered into EPI data version 3.1 and exported to SPSS version 20 for analysis. A variable having a p-value of <0.25 in the bivariable logistic regression analysis were subjected to multivariable logistic regression analysis to avoid confounding variable's effect. Adjusted odds ratios (AOR) were calculated at 95% confidence interval (CI) and considered significant with a p-value of ≤0.05 in the final model.
Results: A total of 217 study participants with T1DM who met inclusion criteria were recruited consecutively during the study period. Their mean age was 43 ± 15.5 years and the overall prevalence of DPN was 37.3% among study participants. The independent predictors of DPN identified by multivariable logistic regression analysis were increasing age [age of 40–49 years (AOR = 3.80; 95% CI: 1.30, 10.60), age of ≥50 years (AOR = 6.50; 95% CI: 2.50, 16.50)], smoking habit [current smoker (AOR = 3.40, 95% CI: 1.20, 9.50; former smoker (AOR = 2.70; 95% CI: 1.60, 6.80)] and comorbid hypertension (AOR = 2.40; 95% CI: 1, 5.40).
Conclusion: The magnitude of DPN among DM patients at JUMC was high. Early detection and appropriate management is vital particularly for these with increasing age, comorbid hypertension and smoking habit.

1. Introduction

Diabetes mellitus (DM) is one of the most common metabolic disorders and is a major health problem throughout the world. It is estimated that it affects 9% of adults worldwide [1]. In Africa, 39 million people were living with DM in 2017 and in 2045; that number is expected to rise to 82 million. As the prevalence and incidence of DM increases, a corresponding increase in the incidence of complications is expected [1, 2].

The most common and distressing of all the chronic complications of DM is diabetic peripheral neuropathy (DPN) [3]. It affects both type 1 and type 2 DM, but in type1DM (T1DM) it develops faster with serious complications due to the early onset of DM [4, 5]. DPN is defined as the presence of signs and/or symptoms of peripheral nerve dysfunction in patients with DM after the ruling out of other causes [6, 7]. The

prevalence of DPN varies among T1DM. In one study, the prevalence of DPN among adults with T1DM at baseline was 6% and increased to 30 at 13–14 years of follow-up [8]. Furthermore, in the Australian cohort of 1, 433 adolescents with T1DM the prevalence of DPN was 21% [9]. In Ethiopia, few studies report the prevalence of DPN in patients' withT1DM. These include 33.3% in Addis Ababa [10], 51.2% in Bahirdar [11] and 16.4% in Jimma [12].

DPN is a main cause of impairment as a result of foot ulceration, disruption of the gait, neuropathic pain and frequent hospitalizations. It accounts for more hospital admissions than all other complications combined and is responsible for 50–75% of non-traumatic amputations [13, 14]. According to previous studies, appropriate interventions can reduce ulcers and amputations by 60% and 85%, respectively, in patients with high-risk groups [15].

---

* Corresponding author.
E-mail address: dhaabaa4@gmail.com (D. Abdissa).

Due to the fact that DPN is mostly irreversible, primary prevention is essential. Studies suggest that the main risk factors responsible for DPN among T1DM patients were longer duration of DM, advanced age, poor glycemic control, cigarette smoking and increased body mass index (BMI) [16, 17, 18].

Screening is necessary to identify DPN in its early stages, as its subclinical signs may precede its development. The American Diabetes Association (ADA) recommends all adults with DM should be screened for DPN at the time of DM diagnosis, and yearly thereafter [19]. Michigan Neuropathy Screening Instrument (MNSI) is one of a simple, validated, sensitive, and specific tool for the screening of DPN among DM patients [20]. However, screening practices has not yet received due attention and there are limited information regarding the burden of DPN among T1DM in Ethiopia including our study area, Jimma. Moreover, most of the studies focused on only type 2 DM. Therefore, the aim of this study was to assess the magnitude and associated factors of DPN using MNSI among T1DM patients attending their follow-up at Jimma University Medical Center (JUMC), Southwest Ethiopia. This study will show the scope of problem in the study area and information collected from this study will provide baseline data for further studies and provide for early identification of the problem and provides input for appropriate interventions.

2. Methods and materials

2.1. Study area, period and design

A hospital based cross-sectional study was conducted from January 2 to March 31, 2020 at JUMC, in Jimma town, Jimma Zone, Oromia region, Southwest Ethiopia. It is one of the largest hospitals in our country serving very large catchment area in the Southwestern part of Ethiopia. It offers a variety of specialized clinical services including follow-up care for patients with DM, hypertension (HTN), and other chronic conditions.

2.2. Selection of study subjects

A total of 217 adult patients with T1DM who met the inclusion criteria were consecutively recruited over a 3-month period. Individuals <18 years of age, type 2 DM, pregnant women, participants with HIV, alcohol use disorder, critically ill and psychiatric disorders were excluded. For those who had repeated clinic visits during the study period, data were collected during their first visits.

2.3. Data collection tool and procedure

Data were collected using a pretested, structured interviewer-administered questionnaire which was developed after reviewing several different related studies [11, 12, 21, 22]. It was first prepared in English, then translated to local languages and back translated to English to ensure the translated version gave the required meaning by language experts in both cases. Data were collected through face to face interviews, physical examination and patient record reviews. The questionnaire consisted of four sections: socio-demographic, behavioral characteristics, clinical characteristics and MNSI.

2.4. Physical measurements

Anthropometric measurements: Height was measured by using a stadiometer to the nearest 0.1 cm (cm), by standing upright on a flat surface. Body weight measured while wearing light clothes by an adjusted weight scale to the nearest 0.1 kg. BMI was calculated as weight in kilograms divided by height in meters squared and other clinical variables were retrieved from patient chart review. World health organization STEPS wise approach for chronic disease risk factor surveillance were used to assess behavioral characteristics [22].

Assessment of DPN: DPN was assessed using the MNSI, which is a validated, noninvasive screening tool for DPN with a sensitivity of 80% and a specificity of 95% [20]. It has two complementary sections: MNSI history version and MNSI examination version which was done by trained data collectors.

MNSI History Version scoring: It encompasses 15 items of which only 13 items were used to assess symptoms of DPN. 'Yes' responses to questions 1–3, 5, 6, 8, 9, 11, 12, 14, and 15 were each counted as one point and 'No' responses to questions 7 and 13 likewise counted as one point. Item number 4 and item number 10 were not included in scoring. Thus, the total score ranges from 0 to 13 points and a score that is ≥ **7** indicated the presence of DPN by MNSI history version [21].

MNSI examination version procedure and scoring: It is a brief physical examination of feet involving: inspection of feet appearance, vibration perception at the distal great toe using 128-hertz C-tuning fork, examination of ankle reflex using standard triangular rubber-headed hammer and 5.07/10-g Semmes-Weinstein monofilament test. Scores were assigned as follows as per MNSI protocol; appearance of feet (normal = 0, abnormal = 1), ulceration (absent = 0, present = 1), ankle reflexes (absent = 1, present with reinforcement = 0.5, present = 0), vibration perception (absent = 1, reduced = 0.5, present = 0) and finally monofilament test. The monofilament test was examined on 9 sites on plantar surface of the foot and one on the dorsum feet and scored as (8 correct responses out of 10 scored 0, 1–7 correct scored 0.5, and no correct answers scored as 1). Each foot was independently assessed and the score of both feet were added together [21].

Detailed training on each procedure was given to data collectors to reduce intra- and inter-examiner difference; in addition each physical examination was crosschecked by 2 supervisors independently. Data were collected by 3 medical interns with supervision of two supervisors and principal investigator.

2.5. Operational definitions

DPN: was defined if the patient's history version of MNSI questionnaire score was ≥7 and/or if the examination version of MNSI scores was ≥2.5.

Controlled fasting blood sugar: Fasting blood sugar (FBS) of 80–130 mg/dl [23].

Critically ill: Patients who are unable to communicate during data collection period.

Type 1 diabetes patient: Patients diagnosed with T1DM who were on follow-up at JUMC.

Alcohol use disorder: in this study was defined as consumption of ≥6 standard drinks and ≥4 standard drinks on a single occasion in men and women respectively.

Standard drink: is any drink containing about 10grame of alcohol [22].

2.6. Statistical analysis

Collected data were checked for completeness and any error by supervisor and principal investigator on daily basis and initially checked for completeness and consistency before and after data entry. Then entered into the Epi-Data version 3.1 and exported to the SPSS version 20 for analysis. Descriptive statistics such as means, percentages, and standard deviations were calculated as necessary. A bivariable logistic regression between outcome variable and a number of independent variables were examined and those independent variables whose p-values less than <0.25 in bivariable analysis were shifted into the multivariable logistic model. Multivariable logistic regression was performed using a backward method to identify independent predictors. Finally, variables which had independent association with dependent variable were identified on basis of adjusted odds ratio (AOR), with 95% confidence interval (CI) and a p-value of ≤0.05. The Hosmer and Lemeshow goodness of fit test were checked and gave a p-value of 0.707, signifying fitness of the model.

2.7. Data quality assurance

To ensure reliable data collection, training was given to the data collectors and supervisors on the purposes of the study, measurement techniques, chart review contents and interview methods by principal investigator. The English version of the tool were translated to the local language and again translated back to English. The original and translated questionnaire was checked for consistency and the discrepancies reviewed and resolved accordingly. The questionnaire was pretested on T1DM patients at nearby Shenen Gibe hospital to check for the validity of the questionnaire. The collected were checked carefully on a daily basis for accuracy, completeness, and clarity and continuous supervision was made by the supervisors and principal investigator throughout the data collection period.

2.8. Ethical approval and consent to participate

Ethical clearance was obtained from the Jimma University Institutional Review Board (IRB). A supportive formal letter was written to JUMC outpatient unit manager. Then respondents were well informed about the purpose and benefit of the study. Then information was collected after oral informed consent from each participant was obtained. Information was recorded anonymously and confidentiality was assured throughout the study period. The study was conducted in accordance with the Declaration of Helsinki.

3. Results

3.1. Socio-demographic characteristics of participants

A total of 217 participants were involved in this study with a 100% response rate. Their mean age was 43 ± 15.5 years. More than half (55.8%) of the respondents were males and almost one fourth (26.7%) of them had family history of DM. Near to half of the respondents (45.6%) were Muslim in religion and nearly three-quarters (72.8%) of them were married (Table 1).

3.2. Behavioral and clinical characteristics of participants

The mean duration of DM was 5.1 ± 4.5 years and nearly three-quarters (73.7%) were in the normal BMI category. Regarding the participants behavioral characteristics nearly half (47.5%) were physically active and some (11.5%) of participants were current smokers (Table 2).

3.3. Prevalence of DPN

In this study, the prevalence of DPN was assessed with the combined MNSI history and examination scores. The prevalence of DPN based on the symptom score alone was 19 (8.8%), while that of the sign score was 72 (33.2%). The overall prevalence of DPN using combined MNSI history and MNSI examination version among the study population was 81 (37.3%).

3.3.1. Factors independently associated with DPN

Variables that were significantly associated with DPN on bivariable logistic regression analysis were re-entered into multivariable logistic regression model as independent variables for outcome variable. In this manner, three of the seven candidate variables were found significant predictors of overall prevalence of DPN. The factors that were identified to be significantly associated with the development of DPN were; increasing age, comorbid HTN and smoking habit.

Diabetic patients in their age 40–49 years were 3.8 times more likely to develop DPN compared to patients younger than 30 years [AOR = 3.80; 95%CI: 1.30,10.60, p = 0.011]. Likewise, participants of age ≥50 years were 6.5 times more likely to develop DPN compared to patients whose ages were younger than 30 [AOR = 6.50; 95%CI:2.50,16.50,

Table 1. Socio-demographic characteristics of participants at JUMC 2020, Ethiopia (n = 217).

Variables	Category	Number	Percentage
Sex	male	121	55.8
	female	96	44.2
Residence	urban	108	49.8
	rural	109	50.2
Family history of DM	yes	58	26.7
	no	159	73.3
Age (years)	<30	65	30
	30 to 39	21	9.7
	40 to 49	48	22.1
	≥50	83	38.2
Marital status	married	158	72.8
	single	33	15.2
	others*	26	12
Ethnicity	Oromo	120	55.3
	Amhara	63	29
	Others†	34	15.7
Religion	muslim	99	45.6
	orthodox	81	37.3
	protestants	37	17.1
Educational status	illiterate	56	25.8
	primary	107	49.3
	Secondary and above	54	24.9
Occupational status	house wife	51	23.5
	farmer	58	26.7
	private worker	54	24.9
	governmental employer	48	22.1
	Others‡	6	2.8
Average monthly income (ETB)	<1000	59	27.2
	1000 to 2999	95	43.8
	≥3000	63	29

*Widowed, divorced; † Yem, Keffa, Tigre; ‡ Retired, unemployed; ETB = Ethiopian birr.

Table 2. Clinical and behavioral characteristics of participants at JUMC 2020, Ethiopia (n = 217).

Variables	Category	Number	Percentage
Duration of DM	<5 years	123	56.7
	5–10 years	63	29
	≥10 years	31	14.3
BMI (Kg/m²)	18.5 to 24.9	160	73.7
	<18.5	21	9.7
	≥25	36	16.6
Alcohol intake	current	26	12
	former	17	7.8
	never	174	80.2
Smoking	current	25	11.5
	former	32	14.
	never	160	73.7
Physical exercise	physically active	103	47.5
	physically inactive	114	52.5
Fasting blood sugar (mg/dl)	<130	157	72.4
	≥130	60	27.6
Comorbid hypertension	yes	42	19.4
	no	175	80.6

P = ≤0.001]. Participants who were currently active smokers were 3.4 times more likely to develop DPN as compared with those who never smoked [AOR = 3.40; 95%CI: 1.20, 9.50, p = 0.02]. Likewise, former

smokers were 2.7 times more likely to develop DPN as compared with those who never smoked [AOR = 2.70; 95%CI: 1.60, 6.80, p = 0.042]. Finally, participants with comorbid HTN were 2.4 times more likely to develop DPN as compared with their counterparts [AOR = 2.40; 95% CI: 1,5.40, p = 0.039] (Table 3).

4. Discussion

This study examined the prevalence and predictors of DPN in T1DM. In this study, the prevalence of DPN according to the MNSI history version was 8.8%, which is lower than the prevalence identified in the MNSI examination of 33.2%. This difference demonstrates the importance of the MNSI examination version for early detection of DPN and the limitations associated with the MNSI history version. Therefore, DM patients should be screened for DPN even in the absence of neuropathic symptoms. The overall prevalence of DPN using combined MNSI history and examination version was found to be 37.3% [95%CI: 30.90,43.80]. Therefore, a combination of MNSI history and examination versions is suitable for early detection of DPN in DM patients. This result was consistent with other studies reporting prevalence of 33.3% in Addis Ababa, Ethiopia [10], 34.2% in Austria [24], and 42.8% in Porto, Portugal [25].

However, the results of the current study were higher than those of studies conducted in Spain, the United States, and China, reporting a prevalence of 12.9%, 7% and 21.92%, respectively [26, 27, 28]. Possible differences may be due to differences in study design, study populations, existing medical facilities, and different diagnostic criteria for assessing DPN. Another study, from Jimma, Ethiopia by Worku et al. 2010 reported 16.4% [12]. The possible explanation of this discrepancy was they used patient record review to assess the prevalence of DPN. In our study, DPN was evaluated using MNSI. This can increase the detection of neuropathy in the patient.

On the other hand, our finding was lower than the study done in Danish which reported prevalence of 62% [29]. The observed higher DPN prevalence in Danish study could be due to the longer duration of DM and reliance on a single measure of neuropathy as opposed to our use of the MNSI, which includes both history and different physical assessment parts. In the same way study done in Iran and Egypt reported 57.5% and 61.7% respectively [30, 31]. This difference in prevalence may be

due to different diagnostic methods, duration of illness, sample selection, and study population. Furthermore, study done in Bahirdar, Ethiopia reported the prevalence of DPN among T1DM was 51.2% [11]. These discrepancies can be due to the sample size and sampling method used.

Consistent with previous studies, the current findings show that age of greater than 40 years was associated with increased risk of DPN [27, 32, 33]. This may be due to the fact that physiological well-being deteriorates with age and the nervous system becomes more and more susceptible to long-term metabolic stress.

Furthermore, in agreement with other previous studies, our study revealed smoking habit was significantly related with increased risk of developing DPN [25, 27, 34]. The possible explanation for this relation can be due to cigarette smoking increases risk for DPN through its toxic effects on the neurons and neuronal ischemia from increased inflammation, endothelial damage and oxidative stress [35].

Finally, this study demonstrated that participants with comorbid HTN were 2.4 times more likely to develop DPN as compared with their counterparts [25, 27, 29]. This association may be justified by rodent studies showing that coexisting HTN with DPN was attributed to damage to the myelin sheath around the axon [36].

5. Conclusion

The magnitude of DPN among T1DM patients at JUMC was high. It increases with the age of >40 years, presence of comorbid HTN and smoking habit. The findings suggest the need for appropriate interventions among identified high risk groups in order to decrease consequent complications.

5.1. Limitations of the study

1st since, we enrolled participants from just one institution, this study couldn't be generalized to all DM patients in the country. 2nd lack of nerve conduction study which is the definitive and standard techniques for detecting neuropathy. 3rd the recall bias might be affect MNSI history score. 4th it was not possible to identify all predictors of DPN; also these results may not apply to those with type 2 DM. Lastly, the nature of the cross-sectional study design did not indicate a temporal relationship and

Table 3. Independent predictors of DPN among diabetic patients at JUMC 2020, Ethiopia (n = 217).

Variables	Category	DPN		Bivariable analysis		Multivariable analysis	
		Yes	No	P- value	COR (95%CI)	P-value	AOR (95%CI)
Age (years)	<30	8	57	1	1	1	
	30–39	4	17	.442	1.67 [.45,6.30]	.787	1.20 [.29,5.20]
	40–49	23	25	≤0.001	6.50 [2.60,16.60]	.011*	3.80 [1.30,10.60]
	≥50	46	37	≤0.001	8.80 [3.80,20.90]	≤0.001*	6.50 [2.50,16.50]
Sex	male	52	69	0.054	1.70 [.98,3.06]	.105	1.70 [.88,3.50]
	female	29	67	1	1		1
Residence	urban	45	63	.189	1.40 [.83,2.50]	.325	1.40 [.70,2.80]
	rural	36	73	1	1	1	1
BMI(Kg/m²)	18.5to24.9	53	107	1	1	1	1
	<18.5	6	15	.676	.80 [.30,2.20]	.781	.80 [.20,2.90]
	≥25	22	14	.002	3 [1.50,6.70]	.344	1.50 [.60,3.80]
Smoking	current	16	9	0.001	4.30 [1.70,10.30]	.020*	3.40 [1.20,9.50]
	former	18	14	0.004	3 [1.40,6.70]	.042*	2.70 [1,6.80]
	never	47	113	1	1	1	1
Comorbid hypertension	yes	27	15	≤0.001	4 [1.98,8.20]	.039*	2.40 [1,5.40]
	no	54	121	1	1	1	1
Duration of DM (years)	<5	46	77	1	1	1	1
	5–10	19	44	.328	.70 [.40,1.40]	.146	.50 [.30,1.20]
	≥10	16	15	.152	1.70 [.80,3.90]	.650	1.20 [.50,3.30]

* value statistically significant; AOR- Adjusted Odds ratio; COR-Crude odds ratio; CI-Confidence interval; 1-reference.

other uncommon causes of neuropathy like vasculitis and vitamin B deficiency were not excluded.

Declarations

Author contribution statement

Daba Abdissa: Conceived and designed the experiments; Performed the experiments; Wrote the paper.

Rebuma Sorsa and Asfaw Gerbi: Performed the experiments; Analyzed and interpreted the data; Contributed reagents, materials, analysis tools or data.

Nigusse Hamba and Zelalem Banjaw: Performed the experiments; Wrote the paper.

Funding statement

Data availability statement

Data will be made available on request.

Declaration of interests statement

The authors declare no conflict of interest.

Additional information

No additional information is available for this paper.

Acknowledgements

References

[1] World Health Organization, Global Status Report on Noncommunicable Diseases 2014. Geneva, 2014. http://www.who.int/nmh/publications/ncd-status-report-2014/en/.

[2] P. Saeedi, I. Petersohn, P. Salpea, B. Malanda, S. Karuranga, N. Unwin, S. Colagiuri, L. Guariguata, A.A. Motala, K. Ogurtsova, J.E. Shaw, Global and regional diabetes prevalence estimates for 2019 and projections for 2030 and 2045: results from the International Diabetes Federation Diabetes Atlas, Diabetes Res. Clin. Pract. 157 (2019 Nov 1) 107843.

[3] A.J. Boulton, A.I. Vinik, J.C. Arezzo, V. Bril, E.L. Feldman, R. Freeman, R.A. Malik, R.E. Maser, J.M. Sosenko, D. Ziegler, Diabetic neuropathies: a statement by the American Diabetes Association, Diabetes Care 28 (4) (2005 Apr 1) 956–962.

[4] E. Otto-Buczkowska, Z. Kazibutowska, J. Sołtyk, L. Machnica, Neuropathy and type 1 diabetes mellitus, Pediatr. Endocrinol. Diabetes Metab. 14 (2) (2008) 109–116.

[5] M. Louraki, C. Karayianni, C. Kanaka-Gantenbein, M. Katsalouli, K. Karavanaki, Peripheral neuropathy in children with type 1 diabetes, Diabetes Metabol. 38 (4) (2012 Oct 1) 281–289.

[6] R. Pop-Busui, A.J. Boulton, E.L. Feldman, V. Bril, R. Freeman, R.A. Malik, J.M. Sosenko, D. Ziegler, Diabetic neuropathy: a position statement by the American Diabetes Association, Diabetes Care 40 (1) (2017 Jan 1) 136–154.

[7] L. Johannsen, T. Smith, A.M. Havsager, C. Madsen, M.J. Kjeldsen, N.J. Dalsgaard, D. Gaist, H.D. Schröder, S.H. Sindrup, Evaluation of patients with symptoms suggestive of chronic polyneuropathy, J. Clin. Neuromuscul. Dis. 3 (2) (2001 Dec 1) 47–52.

[8] C.L. Martin, J.W. Albers, R. Pop-Busui, DCCT/EDiC research Group, Neuropathy and related findings in the diabetes control and complications trial/epidemiology of diabetes interventions and complications study, Diabetes Care 37 (1) (2014 Jan 1) 31–38.

[9] M.C. Eppens, M.E. Craig, J. Cusumano, S. Hing, A.K. Chan, N.J. Howard, M. Silink, K.C. Donaghue, Prevalence of diabetes complications in adolescents with type 2 compared with type 1 diabetes, Diabetes Care 29 (6) (2006 Jun 1) 1300–1306.

[10] G. Jarso, A. Ahmed, Y. Feleke, The prevalence, clinical features and management of periphral neuropathy among diabetic patients in Tikur Anbessa and St. Paul's Specialized University Hospitals, Addis Ababa, Ethiopia, Ethiop. Med. J. 49 (4) (2011 Oct) 299–311.

[11] G. Jember, Y.A. Melsew, B. Fisseha, K. Sany, A.Y. Gelaw, B. Janakiraman, Peripheral Sensory Neuropathy and associated factors among adult diabetes mellitus patients in Bahr Dar, Ethiopia, J. Diabetes Metab. Disord. 16 (1) (2017 Dec) 1–8.

[12] D. Worku, L. Hamza, K. Woldemichael, Patterns of diabetic complications at jimma university specialized hospital, southwest Ethiopia, Ethiop J Health Sci 20 (1) (2010) 33–39.

[13] W. Jeffcoate, K. Bakker, World diabetes day: footing the bill, Lancet 365 (9470) (2005 Apr 30) 1527.

[14] A.J. Boulton, R.A. Malik, J.C. Arezzo, J.M. Sosenko, Diabetic somatic neuropathies, Diabetes Care 27 (6) (2004 Jun 1) 1458–1486.

[15] G. Ning, Progress in the diagnosis of diabetic peripheral neuropathy, Chin. J. Pract. Intern. Med. 7 (2007) 487–489.

[16] K. Van Acker, D. Bouhassira, D. De Bacquer, S. Weiss, K. Matthys, H. Raemen, C. Mathieu, I.M. Colin, Prevalence and impact on quality of life of peripheral neuropathy with or without neuropathic pain in type 1 and type 2 diabetic patients attending hospital outpatients clinics, Diabetes Metabol. 35 (3) (2009 Jun 1) 206–213.

[17] S. Tesfaye, A.J. Boulton, P.J. Dyck, R. Freeman, M. Horowitz, P. Kempler, G. Lauria, R.A. Malik, V. Spallone, A. Vinik, L. Bernardi, Diabetic neuropathies: update on definitions, diagnostic criteria, estimation of severity, and treatments, Diabetes Care 33 (10) (2010 Oct 1) 2285–2293.

[18] X. Liu, Y. Xu, M. An, Q. Zeng, The risk factors for diabetic peripheral neuropathy: a meta-analysis, PLoS One 14 (2) (2019 Feb 20), e0212574.

[19] American Diabetes Association, Microvascular complications and foot care: standards of medical care in diabetes—2019, 11, Diabetes Care 42 (Supplement 1) (2019 Jan 1) S124–S138.

[20] W.H. Herman, R. Pop-Busui, B.H. Braffett, C.L. Martin, P.A. Cleary, J.W. Albers, E.L. Feldman, DCCT/EDIC Research Group, Use of the Michigan neuropathy screening instrument as a measure of distal symmetrical peripheral neuropathy in type 1 diabetes: results from the diabetes control and complications trial/epidemiology of diabetes interventions and complications, Diabet. Med. 29 (7) (2012 Jul) 937–944.

[21] University of Michigan, How to Use the Michigan Neuropathy Screening Instrument. Michigan. http://diabetesresearch.med.umich.edu/peripherals/profs/documents/svi/MNSI_howto.pdf.

[22] STEP Wise Approach to Surveillance (STEPS) [webpage on the Internet]. Geneva: World Health Organization. Available from: www.who.int/chp/steps.

[23] D. Care, 6. Glycemic targets: standards of medical care in diabetes—2019, Diabetes Care 42 (Supplement 1) (2019 Jan 1) S61–70.

[24] I. Walter-Höliner, D.S. Barbarini, J. Lütschg, A. Blassnig-Ezeh, U. Zanier, C.H. Saely, B. Simma, High prevalence and incidence of diabetic peripheral neuropathy in children and adolescents with type 1 diabetes mellitus: results from a five-year prospective cohort study, Pediatr. Neurol. 80 (2018 Mar 1) 51–60.

[25] M. Barbosa, A. Saavedra, S. Oliveira, L. Reis, F. Rodrigues, M. Severo, R. Sittl, C. Maier, D.M. Carvalho, Prevalence and determinants of painful and painless neuropathy in type 1 diabetes mellitus, Front. Endocrinol. 10 (2019 Jun 28) 402.

[26] J. Cabezas-Cerrato, Neuropathy Spanish Study Group of the Spanish Diabetes Society (SDS. The prevalence of clinical diabetic polyneuropathy in Spain: a study in primary care and hospital clinic groups, Diabetologia 41 (11) (1998 Oct 1) 1263–1269.

[27] M. Jaiswal, J. Divers, D. Dabelea, S. Isom, R.A. Bell, C.L. Martin, D.J. Pettitt, S. Saydah, C. Pihoker, D.A. Standiford, L.M. Dolan, Prevalence of and risk factors for diabetic peripheral neuropathy in youth with type 1 and type 2 diabetes: SEARCH for Diabetes in Youth Study, Diabetes Care 40 (9) (2017 Sep 1) 1226–1232.

[28] Q. Pan, Q. Li, W. Deng, D. Zhao, L. Qi, W. Huang, L. Ma, H. Li, Y. Li, X. Lyu, A. Wang, Prevalence of and risk factors for peripheral neuropathy in Chinese patients with diabetes: a multicenter cross-sectional study, Front. Endocrinol. 9 (2018 Nov 5) 617.

[29] B.S. Olsen, J. Johannesen, A.K. Sjølie, K. Borch-Johnsen, P. Hougaard, B. Thorsteinsson, S. Pramming, K. Marinelli, H.B. Mortensen, T.D. Of Diabetes, I. Childhood, Metabolic control and prevalence of microvascular complications in young Danish patients with Type 1 diabetes mellitus, Diabet. Med. 16 (1) (1999 Jan) 79–85.

[30] Y.A. Al-Taweel, R.M. Fahmi, N. Shehta, T.S. Elserafy, H.M. Allam, A.F. Elsaid, Frequency and determinants of subclinical neuropathy in type 1 diabetes mellitus, Egypt. J. Neurol. Psychiatr. Neurosurg. 53 (4) (2016 Oct 1) 232.

[31] V. Toopchizadeh, S. Shiva, N.Y. Khiabani, R. Ghergherechi, Electrophysiologic pattern and prevalence of subclinical peripheral neuropathy in children and adolescents with type I diabetes mellitus in Iran, Saudi Med. J. 37 (3) (2016 Mar) 299.

[32] X.H. Bao, V. Wong, Q. Wang, L.C. Low, Prevalence of peripheral neuropathy with insulin-dependent diabetes mellitus, Pediatr. Neurol. 20 (3) (1999 Mar 1) 204–209.

[33] M.C. Monti, J.T. Lonsdale, C. Montomoli, R. Montross, E. Schlag, D.A. Greenberg, Familial risk factors for microvascular complications and differential male-female risk in a large cohort of American families with type 1 diabetes, J. Clin. Endocrinol. Metabol. 92 (12) (2007 Dec 1) 4650–4655.

[34] C. Clair, M.J. Cohen, F. Eichler, K.J. Selby, N.A. Rigotti, The effect of cigarette smoking on diabetic peripheral neuropathy: a systematic review and meta-analysis, J. Gen. Intern. Med. 30 (8) (2015 Aug 1) 1193–1203.

[35] B. Eliasson, Cigarette smoking and diabetes, Prog. Cardiovasc. Dis. 45 (5) (2003) 405–413.

[36] J.A. Gregory, C.G. Jolivalt, J. Goor, A.P. Mizisin, N.A. Calcutt, Hypertension-induced peripheral neuropathy and the combined effects of hypertension and diabetes on nerve structure and function in rats, Acta Neuropathol. 124 (4) (2012 Oct 1) 561–573.