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Original Research Article

# Prevalence and Clinical Spectrum of Peripheral Neuropathy in Patients with Type 2 Diabetes Mellitus

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**Abstract**

**Background:** Diabetic peripheral neuropathy (DPN) represents the most common microvascular complication of diabetes mellitus, significantly impacting patient quality of life and healthcare burden. Understanding the prevalence and clinical spectrum of DPN in diverse populations is essential for developing effective screening and management strategies.

**Methods:** This cross-sectional study was conducted among 386 patients with type 2 diabetes mellitus (T2DM) attending outpatient clinics of a tertiary care hospital. DPN was assessed using the Michigan Neuropathy Screening Instrument (MNSI), vibration perception threshold (VPT), and nerve conduction studies (NCS). Clinical spectrum, risk factors, and associated complications were analyzed using appropriate statistical methods.

**Results:** The overall prevalence of DPN was 47.2% (n=182). Among affected patients, sensory neuropathy was most common (78.6%), followed by sensorimotor (18.1%) and pure motor neuropathy (3.3%). Patients with DPN had significantly longer diabetes duration (11.8 ± 6.2 vs. 6.4 ± 4.1 years; $p<0.001$) and higher HbA1c levels (8.9 ± 1.8% vs. 7.6 ± 1.4%; $p<0.001$). Multivariate analysis identified diabetes duration >10 years (OR=3.86; 95% CI: 2.42-6.16), HbA1c >8% (OR=2.74; 95% CI: 1.78-4.22), hypertension (OR=1.92; 95% CI: 1.24-2.97), and smoking (OR=2.18; 95% CI: 1.31-3.63) as independent predictors of DPN.

**Conclusion:** Nearly half of T2DM patients have peripheral neuropathy, predominantly sensory type. Longer disease duration, poor glycemic control, hypertension, and smoking are significant modifiable risk factors. Early screening and aggressive risk factor management are essential for preventing this debilitating complication.

**Keywords:** Diabetic Peripheral Neuropathy, Type 2 Diabetes Mellitus, Prevalence, Nerve Conduction Studies, Risk Factors.



## Introduction

Diabetes mellitus constitutes a global health epidemic, with an estimated 537 million adults affected worldwide, and projected to reach 783 million by 2045 [1]. Type 2 diabetes mellitus (T2DM) accounts for approximately 90% of all diabetes cases and is associated with multiple microvascular and macrovascular complications that significantly impact patient morbidity and mortality [2].

Diabetic peripheral neuropathy (DPN) is the most prevalent microvascular complication, affecting up to 50% of diabetic patients during their lifetime [3]. The condition is characterized by progressive damage to peripheral nerve fibers, manifesting as sensory, motor, or autonomic dysfunction [4]. The distal symmetric polyneuropathy pattern, affecting the feet and hands in a "stocking-glove" distribution, represents the most common clinical presentation [5]. The pathophysiology of DPN involves complex metabolic and vascular mechanisms triggered by chronic hyperglycemia. These include polyol pathway activation, advanced glycation end-product accumulation, oxidative stress, protein kinase C activation, and impaired neurotrophic support [6]. Emerging evidence also implicates mitochondrial dysfunction and neuroinflammation in disease progression [7].

The clinical significance of DPN extends beyond sensory symptoms. Patients with DPN face substantially increased risks of foot ulceration, Charcot neuroarthropathy, and lower extremity amputation [8]. Furthermore, painful diabetic

neuropathy affects approximately 20-30% of patients with DPN, causing significant impairment in sleep quality, mood, and daily functioning [9].

Several epidemiological studies have examined DPN prevalence across different populations. The Rochester Diabetic Neuropathy Study reported a prevalence of 66% using comprehensive neurological assessment [10]. However, prevalence estimates vary considerably depending on diagnostic criteria employed, population studied, and screening methods utilized [11]. Studies from Asian populations have reported prevalence rates ranging from 25% to 55%, reflecting potential ethnic and environmental influences [12].

Despite extensive research, significant gaps remain in understanding the clinical spectrum and determinants of DPN in resource-limited settings where diabetes burden is rapidly increasing. Many patients remain undiagnosed until advanced complications develop, highlighting the need for improved screening strategies [13]. Furthermore, the relative contribution of modifiable risk factors beyond glycemic control deserves further investigation to guide comprehensive management approaches.

The present study aimed to determine the prevalence of peripheral neuropathy in patients with T2DM, characterize its clinical spectrum, and identify associated risk factors in a hospital-based population.

## Materials and Methods

**Study Design and Setting:** This cross-sectional observational study was conducted in Outpatient Clinics of a tertiary care teaching hospital.

**Sample Size Calculation:** Based on previously reported DPN prevalence of approximately 45%, with 95% confidence interval and 5% margin of error, the minimum required sample size was calculated as 380 patients. Accounting for incomplete data, a target enrollment of 400 patients was planned.

**Inclusion Criteria:** Adult patients (≥18 years) with established diagnosis of T2DM according to American Diabetes Association criteria, attending outpatient clinics for routine follow-up, and willing to participate were included. Both newly diagnosed and previously known diabetic patients were eligible.

**Exclusion Criteria:** Patients with type 1 diabetes mellitus, secondary diabetes, gestational diabetes, peripheral neuropathy due to other known causes (vitamin B12 deficiency, chronic kidney disease stage 4-5, hypothyroidism, chronic alcoholism, chemotherapy-induced neuropathy, hereditary neuropathies), acute diabetic complications at presentation, and those unable to cooperate with neurological examination were excluded.

**Data Collection:** A structured questionnaire captured demographic information (age, sex, education, occupation), diabetes-related parameters (duration of diabetes, treatment modality, compliance), lifestyle factors (smoking, alcohol consumption, physical activity), and medical history (hypertension, dyslipidemia, cardiovascular disease, retinopathy, nephropathy).

**Clinical Assessment:** All participants underwent comprehensive clinical examination including measurement of height, weight, body mass index (BMI), waist circumference, and blood pressure. Neurological examination assessed deep tendon reflexes (ankle and knee), vibration sense (using 128 Hz tuning fork), position sense, light touch (using 10-g Semmes-Weinstein monofilament), and pinprick sensation.

### Neuropathy Assessment Tools

**Michigan Neuropathy Screening Instrument (MNSI):** This validated questionnaire includes 15 self-administered questions (Part A) and lower extremity examination (Part B). A questionnaire score ≥7 or examination score ≥2.5 was considered positive for neuropathy.

**Vibration Perception Threshold (VPT):** Measured using a biothesiometer at the great toe bilaterally. VPT >25 volts was considered abnormal.

**Nerve Conduction Studies (NCS):** Performed in all patients with positive screening tests and a random sample of screen-negative patients. Motor nerve conduction was assessed in median, ulnar, common peroneal, and tibial nerves. Sensory nerve conduction was assessed in median, ulnar, and sural nerves. DPN was confirmed based on abnormality in at least two nerves with at least one being sural nerve.

**Classification of Neuropathy:** Neuropathy was classified as: (1) Sensory - predominantly sensory symptoms and signs with preserved motor function; (2) Motor - predominantly motor weakness with minimal sensory involvement; (3) Sensorimotor - combined sensory and motor involvement. Severity was graded as mild, moderate, or severe based on NCS findings and clinical examination.

**Laboratory Investigations:** Fasting blood glucose, HbA1c, lipid profile, serum creatinine, estimated glomerular filtration rate (eGFR), and serum vitamin B12 levels were measured using standardized laboratory methods.

**Statistical Analysis:** Data were analyzed using SPSS version 25.0. Continuous variables were expressed as mean ± standard deviation and

compared using independent samples t-test. Categorical variables were expressed as frequencies and percentages, compared using chi-square test. Variables significant on univariate analysis ($p<0.10$) were entered into multivariate binary logistic regression to identify independent predictors of DPN. A p-value <0.05 was considered statistically significant.

## Results

**Study Population Characteristics:** Of 412 patients screened, 386 met inclusion criteria and were enrolled. The mean age was 54.8 ± 11.2 years, with slight male predominance (54.4%, n=210). The mean duration of diabetes was 8.9 ± 5.8 years, and mean HbA1c was 8.2 ± 1.7%. Oral hypoglycemic agents alone were used by 52.3%, insulin alone by 18.1%, and combination therapy by 29.6%.

**Prevalence of Diabetic Peripheral Neuropathy:** The overall prevalence of DPN was 47.2% (n=182). Based on MNSI questionnaire alone, 38.6% screened positive, while MNSI examination identified 42.2%. VPT >25 volts was present in 36.8%. Nerve conduction studies confirmed neuropathy in 89.5% of clinically suspected cases.

**Clinical Spectrum of Neuropathy:** Among the 182 patients with DPN, sensory neuropathy was most common (78.6%, n=143), followed by sensorimotor neuropathy (18.1%, n=33) and pure motor neuropathy (3.3%, n=6). Regarding severity, 42.3% had mild, 38.5% had moderate, and 19.2% had severe neuropathy. Table 1 presents the detailed clinical characteristics of neuropathy.

**Table 1: Clinical Spectrum and Characteristics of Diabetic Peripheral Neuropathy (n=182)**

| Characteristic | n (%) |
|---|---|
| **Type of Neuropathy** | |
| Sensory predominant | 143 (78.6) |
| Sensorimotor | 33 (18.1) |
| Motor predominant | 6 (3.3) |
| **Severity of Neuropathy** | |
| Mild | 77 (42.3) |
| Moderate | 70 (38.5) |
| Severe | 35 (19.2) |
| **Symptom Pattern** | |
| Numbness/tingling | 156 (85.7) |
| Burning sensation | 98 (53.8) |
| Lancinating pain | 67 (36.8) |
| Muscle weakness | 39 (21.4) |
| Difficulty walking | 42 (23.1) |
| **Distribution** | |
| Distal symmetric (feet) | 168 (92.3) |
| Distal symmetric (hands and feet) | 48 (26.4) |
| Asymmetric | 14 (7.7) |
| **Painful Neuropathy** | |
| Present | 72 (39.6) |
| VAS pain score (mean ± SD) | 5.8 ± 2.1 |
| **Foot Complications** | |
| Previous foot ulcer | 28 (15.4) |
| Active foot ulcer | 12 (6.6) |
| History of amputation | 8 (4.4) |

**Comparison of Patients With and Without DPN:** Table 2 demonstrates the comparison of demographic, clinical, and metabolic parameters between patients with and without DPN. Significant differences were observed in age, diabetes duration, glycemic control, and presence of comorbidities.

**Table 2: Comparison of Clinical and Metabolic Parameters between Groups**

| Parameter | DPN Present (n=182) | DPN Absent (n=204) | p-value |
|---|---|---|---|
| Age (years), mean ± SD | 57.4 ± 10.8 | 52.5 ± 11.1 | <0.001 |
| Male sex, n (%) | 104 (57.1) | 106 (52.0) | 0.298 |
| BMI (kg/m²), mean ± SD | 27.8 ± 4.6 | 26.9 ± 4.2 | 0.048 |
| Waist circumference (cm), mean ± SD | 96.4 ± 11.2 | 92.8 ± 10.6 | 0.002 |
| Duration of diabetes (years), mean ± SD | 11.8 ± 6.2 | 6.4 ± 4.1 | <0.001 |
| Diabetes duration >10 years, n (%) | 98 (53.8) | 42 (20.6) | <0.001 |
| HbA1c (%), mean ± SD | 8.9 ± 1.8 | 7.6 ± 1.4 | <0.001 |

| | | | |
|---|---|---|---|
| HbA1c >8%, n (%) | 118 (64.8) | 72 (35.3) | <0.001 |
| Fasting glucose (mg/dL), mean ± SD | 168.4 ± 52.6 | 142.6 ± 44.8 | <0.001 |
| Total cholesterol (mg/dL), mean ± SD | 198.6 ± 42.4 | 186.4 ± 38.2 | 0.004 |
| Triglycerides (mg/dL), mean ± SD | 178.2 ± 86.4 | 152.6 ± 72.8 | 0.002 |
| LDL-C (mg/dL), mean ± SD | 118.4 ± 36.2 | 108.2 ± 32.6 | 0.004 |
| HDL-C (mg/dL), mean ± SD | 42.6 ± 12.4 | 46.8 ± 14.2 | 0.003 |
| eGFR (mL/min/1.73m²), mean ± SD | 72.4 ± 22.6 | 84.6 ± 18.4 | <0.001 |
| Hypertension, n (%) | 128 (70.3) | 98 (48.0) | <0.001 |
| Current smoker, n (%) | 52 (28.6) | 34 (16.7) | 0.004 |
| Diabetic retinopathy, n (%) | 86 (47.3) | 48 (23.5) | <0.001 |
| Insulin therapy, n (%) | 102 (56.0) | 82 (40.2) | 0.002 |

**Risk Factor Analysis:** Multivariate logistic regression analysis was performed to identify independent predictors of DPN. Table 3 presents the results of univariate and multivariate analyses.

**Table 3: Risk Factors Associated with Diabetic Peripheral Neuropathy**

| Risk Factor | Univariate OR (95% CI) | p-value | Multivariate OR (95% CI) | p-value |
|---|---|---|---|---|
| Age >55 years | 2.14 (1.42-3.22) | <0.001 | 1.48 (0.92-2.38) | 0.108 |
| Diabetes duration >10 years | 4.52 (2.94-6.96) | <0.001 | 3.86 (2.42-6.16) | <0.001 |
| HbA1c >8% | 3.38 (2.24-5.10) | <0.001 | 2.74 (1.78-4.22) | <0.001 |
| Hypertension | 2.56 (1.70-3.86) | <0.001 | 1.92 (1.24-2.97) | 0.004 |
| Current smoking | 2.01 (1.24-3.26) | 0.005 | 2.18 (1.31-3.63) | 0.003 |
| BMI >27 kg/m² | 1.58 (1.06-2.36) | 0.024 | 1.24 (0.78-1.96) | 0.362 |
| Dyslipidemia | 1.84 (1.22-2.78) | 0.004 | 1.46 (0.94-2.28) | 0.094 |
| Diabetic retinopathy | 2.92 (1.90-4.48) | <0.001 | 1.68 (1.04-2.71) | 0.034 |
| eGFR <60 mL/min/1.73m² | 2.24 (1.38-3.64) | 0.001 | 1.54 (0.91-2.62) | 0.108 |

**Nerve Conduction Study Findings:** Among the 182 patients with confirmed DPN, sural nerve sensory conduction was abnormal in 94.5%, while peroneal motor conduction was abnormal in 68.1%. Mean sural nerve amplitude was 4.2 ± 2.8 μV (normal >6 μV), and mean conduction velocity was 38.6 ± 8.4 m/s (normal >40 m/s).

## Discussion

The present study demonstrates a DPN prevalence of 47.2% among T2DM patients, consistent with global estimates ranging from 30% to 60% [14]. This substantial burden underscores the importance of systematic screening programs for early detection and intervention. The predominance of sensory neuropathy (78.6%) aligns with the well-established natural history of DPN, wherein small fiber involvement precedes large fiber damage [15].

The finding that diabetes duration exceeds 10 years in 53.8% of patients with DPN reflects the time-dependent nature of nerve damage. The EURODIAB study similarly demonstrated that diabetes duration was the strongest predictor of DPN incidence, with each year of diabetes increasing risk by approximately 3% [16]. This temporal relationship emphasizes the window of opportunity for preventive interventions in early diabetes.

Poor glycemic control emerged as a significant independent predictor, with HbA1c >8% conferring 2.74-fold increased risk. The Diabetes Control and Complications Trial definitively established the role of intensive glycemic control in preventing neuropathy in type 1 diabetes [17]. However, evidence in T2DM has been less conclusive, with studies suggesting that glycemic control alone may be insufficient to prevent neuropathy progression [18].

The association between hypertension and DPN observed in our study supports the vascular hypothesis of diabetic neuropathy pathogenesis. Tesfaye et al. demonstrated that cardiovascular risk factors, including hypertension, independently predicted neuropathy development even after adjustment for glycemic control [19]. This finding has important clinical implications, suggesting that comprehensive cardiovascular risk management may provide neuroprotective benefits beyond glucose lowering alone.

Smoking emerged as a significant modifiable risk factor, doubling the odds of DPN. Cigarette smoking impairs microvascular function, promotes oxidative stress, and directly damages peripheral nerves [20]. This association reinforces the importance of smoking cessation counseling as an integral component of diabetes care.

The prevalence of painful neuropathy (39.6%) in our cohort is higher than some Western studies but comparable to Asian populations [21]. Painful DPN significantly impacts quality of life and requires specific therapeutic approaches. The mean pain score of 5.8 indicates moderate-to-severe pain

requiring pharmacological management in most cases.

The strong association between DPN and diabetic retinopathy (47.3% vs. 23.5%; $p<0.001$) reflects the shared pathophysiological mechanisms underlying microvascular complications [22]. This clustering of complications suggests that patients with one microvascular complication warrant screening for others.

Nerve conduction studies confirmed subclinical abnormalities in many patients, highlighting the importance of electrodiagnostic testing for accurate diagnosis [23]. The predominant involvement of sural sensory nerve is consistent with length-dependent axonal degeneration characteristic of diabetic polyneuropathy.

Study limitations include the cross-sectional design precluding causal inference, potential selection bias inherent to hospital-based populations, and limited assessment of small fiber neuropathy. Future prospective studies incorporating skin biopsy or corneal confocal microscopy may provide additional insights [24].

## Conclusion

This study demonstrates that diabetic peripheral neuropathy affects nearly half of patients with type 2 diabetes mellitus, predominantly manifesting as distal symmetric sensory polyneuropathy. Diabetes duration exceeding 10 years, poor glycemic control with HbA1c above 8%, hypertension, and smoking are significant independent predictors of neuropathy development.

These findings emphasize the importance of early and systematic screening for peripheral neuropathy in all diabetic patients, particularly those with longer disease duration or suboptimal glycemic control.

A comprehensive management approach addressing not only glycemic control but also cardiovascular risk factors and lifestyle modifications is essential. Implementation of structured screening programs using validated tools such as the MNSI can facilitate early detection and intervention, potentially reducing the burden of this debilitating complication and preventing progression to foot ulceration and amputation.

## References


1. Sun H, Saeedi P, Karuranga S, Pinkepank M, Ogurtsova K, Duncan BB, et al. IDF Diabetes Atlas: Global, regional and country-level diabetes prevalence estimates for 2021 and projections for 2045. Diabetes Res Clin Pract. 2022;183:109119. DOI: 10.1016/j.diabres.2021.109119
2. Zheng Y, Ley SH, Hu FB. Global aetiology and epidemiology of type 2 diabetes mellitus and its complications. Nat Rev Endocrinol. 2018;14(2):88-98. DOI: 10.1038/nrendo.2017.151
3. Pop-Busui R, Boulton AJ, Feldman EL, Bril V, Freeman R, Malik RA, et al. Diabetic Neuropathy: A Position Statement by the American Diabetes Association. Diabetes Care. 2017;40(1):136-154. DOI: 10.2337/dc16-2042
4. Feldman EL, Callaghan BC, Pop-Busui R, Zochodne DW, Wright DE, Bennett DL, et al. Diabetic neuropathy. Nat Rev Dis Primers. 2019;5(1):41. DOI: 10.1038/s41572-019-0092-1
5. Tesfaye S, Boulton AJ, Dyck PJ, Freeman R, Horowitz M, Kempler P, et al. Diabetic neuropathies: update on definitions, diagnostic criteria, estimation of severity, and treatments. Diabetes Care. 2010;33(10):2285-2293. DOI: 10.2337/dc10-1303
6. Callaghan BC, Cheng HT, Stables CL, Smith AL, Feldman EL. Diabetic neuropathy: clinical manifestations and current treatments. Lancet Neurol. 2012;11(6):521-534. DOI: 10.1016/S1474-4422(12)70065-0
7. Fernyhough P, McGavock J. Mechanisms of disease: Mitochondrial dysfunction in sensory neuropathy and other complications in diabetes. Handb Clin Neurol. 2014;126:353-377. DOI: 10.1016/B978-0-444-53480-4.00027-8
8. Boulton AJ, Vileikyte L, Ragnarson-Tennvall G, Apelqvist J. The global burden of diabetic foot disease. Lancet. 2005;366(9498):1719-1724. DOI: 10.1016/S0140-6736(05)67698-2
9. Sloan G, Shillo P, Selvarajah D, Wu J, Wilkinson ID, Tracey I, et al. A new look at painful diabetic neuropathy. Diabetes Res Clin Pract. 2018;144:177-191. DOI: 10.1016/j.diabres.2018.08.020
10. Dyck PJ, Kratz KM, Karnes JL, Litchy WJ, Klein R, Pach JM, et al. The prevalence by staged severity of various types of diabetic neuropathy, retinopathy, and nephropathy in a population-based cohort: the Rochester Diabetic Neuropathy Study. Neurology. 1993;43(4):817-824. PMID: 8469345
11. Ziegler D, Papanas N, Vinik AI, Shaw JE. Epidemiology of polyneuropathy in diabetes and prediabetes. Handb Clin Neurol. 2014;126:3-22. DOI: 10.1016/B978-0-444-53480-4.00001-1
12. Vinik AI, Nevoret ML, Casellini C, Parson H. Diabetic neuropathy. Endocrinol Metab Clin North Am. 2013;42(4):747-787. DOI: 10.1016/j.ecl.2013.06.001
13. Boulton AJ, Vinik AI, Arezzo JC, Bril V, Feldman EL, Freeman R, et al. Diabetic neuropathies: a statement by the American

Diabetes Association. Diabetes Care. 2005; 28(4):956-962. DOI: 10.2337/diacare.28.4.956
14. Young MJ, Boulton AJ, MacLeod AF, Williams DR, Sonksen PH. A multicentre study of the prevalence of diabetic peripheral neuropathy in the United Kingdom hospital clinic population. Diabetologia. 1993;36(2):150-154. DOI: 10.1007/BF00400697
15. Malik RA, Veves A, Tesfaye S, Smith G, Cameron N, Zochodne D, et al. Small fibre neuropathy: role in the diagnosis of diabetic sensorimotor polyneuropathy. Diabetes Metab Res Rev. 2011;27(7):678-684. DOI: 10.1002/dmrr.1222
16. Tesfaye S, Stevens LK, Stephenson JM, Fuller JH, Plater M, Ionescu-Tirgoviste C, et al. Prevalence of diabetic peripheral neuropathy and its relation to glycaemic control and potential risk factors: the EURODIAB IDDM Complications Study. Diabetologia. 1996;39(11):1377-1384. DOI: 10.1007/s001250050586
17. The Diabetes Control and Complications Trial Research Group. The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. N Engl J Med. 1993;329(14):977-986. DOI: 10.1056/NEJM199309303291401
18. Ismail-Beigi F, Craven T, Banerji MA, Basile J, Calles J, Cohen RM, et al. Effect of intensive treatment of hyperglycaemia on microvascular outcomes in type 2 diabetes: an analysis of the ACCORD randomised trial. Lancet. 2010;376(9739):419-430. DOI: 10.1016/S0140-6736(10)60576-4
19. Tesfaye S, Chaturvedi N, Eaton SE, Ward JD, Manes C, Ionescu-Tirgoviste C, et al. Vascular risk factors and diabetic neuropathy. N Engl J Med. 2005;352(4):341-350. DOI: 10.1056/NEJMoa032782
20. Clair C, Cohen MJ, Eichler F, Selby KJ, Rigotti NA. The effect of cigarette smoking on diabetic peripheral neuropathy: a systematic review and meta-analysis. J Gen Intern Med. 2015;30(8):1193-1203. DOI: 10.1007/s11606-015-3354-y
21. Abbott CA, Malik RA, van Ross ER, Kulkarni J, Boulton AJ. Prevalence and characteristics of painful diabetic neuropathy in a large community-based diabetic population in the U.K. Diabetes Care. 2011;34(10):2220-2224. DOI: 10.2337/dc11-1108
22. Jaiswal M, Divers J, Dabelea D, Isom S, Bell RA, Martin CL, et al. 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. 2017;40(9):1226-1232. DOI: 10.2337/dc17-0179
23. Perkins BA, Ngo M, Bril V. Symmetry of nerve conduction studies in different stages of diabetic polyneuropathy. Muscle Nerve. 2002;25(2):212-217. DOI: 10.1002/mus.10044
24. Petropoulos IN, Ponirakis G, Khan A, Almuhannadi H, Gad H, Malik RA. Diagnosing Diabetic Neuropathy: Something Old, Something New. Diabetes Metab J. 2018;42(4):255-269. DOI: 10.4093/dmj.2018.0056.