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SYMPOSIUM

Plantar Pressure Distribution Patterns of Individuals with Prediabetes in Comparison with Healthy Individuals and Individuals with Diabetes

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Abstract

Background:

Since elevated mechanical stress along with loss of plantar protective sensation are considered relevant factors in skin breakdown resulting in diabetic foot ulcerations, the assessment of plantar pressure is important for the prevention of diabetic foot complications. Prediabetes subjects are at risk of chronic hyperglycemia complications, among them neuropathy, but information about plantar loading in this population is not available. We aimed to compare baropodometric parameters of individuals with prediabetes versus healthy persons and persons with diabetes mellitus (DM).

Methods:

Baropodometric data from 73 subjects (15 with prediabetes (pre-DM), 28 with type 2 DM, 30 healthy) aged between 29 and 69 years of both genders were registered through a pressure platform with self-selected gait speed and first-step protocol. Peak plantar pressure, stance time, percentage of contact time, percentage of contact area and pressure-time integral were assessed in five plantar foot regions: heel, midfoot, metatarsals, hallux, and toes 2 to 5. Groups were compared by one-way analysis of variance with Scheffé *post hoc* ($\alpha = 0.05$).

Results:

Age, body mass index, gender, and arch height index did not differ between groups. Pre-DM and DM subjects presented increased peak pressure and pressure-time integral in metatarsals ($p = .010$; $p > .001$), as well as increased percentage of contact time in midfoot ($p = .006$) and metatarsals ($p = .004$) regions when compared with healthy subjects. Stance time was significantly higher ($p = .017$) in DM subjects.

Conclusions:

Pre-DM subjects seem to exhibit an altered plantar pressure distribution pattern similar to that often found in DM subjects.

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Abbreviations: (ADA) American Diabetes Association, (ANOVA) analysis of variance, (BMI) body mass index, (DM) diabetes mellitus, (DMG) type 2 diabetes mellitus group, (HG) healthy group, (IGT) impaired glucose tolerance, (MNSI) Michigan Neuropathy Screening Instrument, (NCT) nerve conduction test, (PDG) prediabetes group, (SD) standard deviation

Keywords: diabetic foot, gait, hyperglycemia, impaired glucose tolerance, secondary prevention

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Introduction

When a chronic above-normal glycemic status does not reach the values established for the diagnosis of diabetes mellitus (DM), it can be classified as impaired fasting glucose or impaired glucose tolerance (IGT), depending on the criteria used for the DM diagnosis.[1] Both conditions are officially referred to as prediabetes (pre-DM) by the American Diabetes Association (ADA)[1] and represent a high-risk factor for DM progression.[2]

Based on the 2011 worldwide IGT estimation, 6.4% of adults between 20 and 79 years old had this condition,[3] which involves chronic above-normal blood glucose levels, leading to microvascular complications, neuropathy, and cardiovascular disease,[4] as occurs in DM.[5] By 2030, an IGT prevalence of 7.1% is expected.[3]

Neuropathy is a common complication of chronic hyperglycemia, affecting 50% to 70% of DM cases.[6] Early hyperglycemia or insulin resistance is sufficient to damage small-diameter peripheral distal axons.[7] Thus, both sensorimotor peripheral neuropathy and autonomic neuropathy occur in pre-DM subjects, in a lower prevalence and with less intensity than that seen in DM, due to the same pathophysiological mechanisms related to glucose metabolism impairment and, as in early diabetes, in a subclinical undiagnosed condition.[8]

Some studies have demonstrated that the sensorimotor loss arising from peripheral neuropathy is associated with muscle imbalance with consequences for motor coordination, abnormal gait, delays in muscle activation patterns,[9,10] and alterations of plantar pressure distribution pattern.[11–17] Although these are well-documented alterations in DM subjects with peripheral neuropathy,[11–18] baropodometric parameters are altered in early type 2 DM subjects without clinical neuropathy or other foot complications. An explanation for this could be a subclinical undiagnosed neuropathic status.

Several factors related to chronic hyperglycemia effects on tissues due to nonenzymatic glycation of structural proteins,[18] such as foot deformities,[19] limited joint mobility,[12,20] and changes in the structure of the fat pad,[21] have been identified as contributing toward the gait pattern modifications[22] and plantar pressure distribution alterations. This leads to increased pressure on the heels and under the metatarsal heads, which are strongly associated with plantar ulceration.[13]

In DM patients, assessment of baropodometric parameters is important for the prevention of foot complications, because elevated mechanical stress along with loss of plantar protective sensation are considered the most relevant factors in skin breakdown, resulting in diabetic foot ulcerations.[21] However, since pre-DM individuals are at risk for chronic hyperglycemia complications, among them neuropathy,[23] it raises the question about what the baropodometric parameters of these individuals would be.

Considering the aforementioned, the objective of this study was to compare the baropodometric gait parameters of pre-DM subjects with those of type 2 DM and healthy subjects without clinical signs and symptoms of neuropathy and other foot complications.

Methods

Participants

A total of 87 subjects were assessed, and 73 who fulfilled the inclusion criteria were assigned to three groups according to glycemic status. Pre-DM and DM endocrinology outpatients of the Hospital de Clínicas de Porto Alegre, Rio Grande do Sul, Brazil, were screened for type 2 DM or pre-DM. Healthy subjects were recruited following a call for volunteers made among relatives of graduate students and professors in the Federal University of Rio Grande do Sul, Brazil.

Inclusion criteria for all the groups were volunteers aged 29 to 69 years old of both genders. The type 2 diabetes mellitus group (DMG) required diagnosis of type 2 DM for more than 2 and less than 10 years; the prediabetes group

(PDG) required diagnosis of prediabetes for more than 1 and less than 5 years. Both diagnoses were given by the endocrinology outpatient physician according to ADA criteria.[1] The healthy group (HG) required negative diagnosis of DM or pre-DM (confirmed by fasting plasma glucose test carried out in the previous 3 months) and no history or suspicion of pathologies that may potentially cause neuropathy.

Exclusion criteria for all groups were any type of foot deformities, clinical signals and symptoms of neuropathy established by a Michigan Neuropathy Screening Instrument (MNSI) score ≥4,[24] history of plantar ulceration, lower limb ischemic vascular disease, history of back or lower limb orthopedic surgery or trauma, rheumatic diseases, central neurological disorders, visual alteration not correctable with lenses, impaired cognitive ability to understand the procedures, incapacity to walk unaided, and presence of symptoms such as vertigo. Subjects were excluded if they presented a difference between relative leg length >10 mm assessed by physical evaluation. In a suspected inconspicuous foot deformity, a radiographic exam was requested.

The principles of the Declaration of Helsinki[25] were applied, and all patients gave written informed consent to participate. The study was approved by the Ethics Committee of the Hospital de Clínicas de Porto Alegre in decision 09-445, March 2010.

Clinical Signs and Symptoms of Peripheral Neuropathy

A trained collaborator, blinded to the subject group, performed the MNSI test, a validated peripheral neuropathy screening tool[24] that evaluates Achilles reflex, vibration sensitivity test on the hallux dorsum with a 128 Hz tuning fork, tactile sensitivity of the plantar aspect of the hallux with a 10 g nylon monofilament (Sory®, Bauru, Brazil), foot deformities through inspection, and a questionnaire about symptoms. Subjects were excluded from the study when an MNSI score was ≥4 (ranging from 0 to 10), which is considered positive for clinical neuropathy.

Physical Evaluation

All subjects underwent a physical examination consisting of body mass index (BMI) assessment through weight and height measurement, screening for foot deformities, relative legs length measurement (the length from the greater trochanter of the femur to the lateral malleolus), measurement of ankle active range of motion for plantar flexion and dorsiflexion, with an analog goniometer (Carci®, São Paulo, Brazil). The subjects presenting foot deformities (prominent metatarsal heads, clawing of the toes, hallux valgus, hallux rigidus, ankle equinus), hyperkeratosis, history of foot conditions requiring professional treatment, or difference between relative legs length >10 mm were excluded because these were considered confounding factors for baropodometric pattern.

Subclinical Peripheral Sensorimotor Neuropathy and Cardiac Autonomic Dysfunction

Assessment of subclinical peripheral sensorimotor neuropathy was made through a nerve conduction test (NCT) by a certified physician. The protocol included the functional test of motor and sensory nerves of the four segments, as well as myography with needle electrode in suspected cases of axonal injury or root involvement, as recommended by the American Association of Electrodiagnostic Medicine.[26] To record and analyze the data, an electromyographic device with two channels (Neurosoft®, Ivanovo, Russia) and dedicated software (NeuroMep®, Ivanovo, Russia) were used. Subclinical peripheral neuropathy definition followed the Epidemiology of Diabetes Intervention and Complications Research Group criteria.[27,28] The evaluator was blinded to the subject group (DMG or PDG). All subjects in the HG and also the subjects in the DMG or PDG who were excluded after the previous assessments did not perform this test for ethical recommendations.

To assess the presence of cardiac autonomic dysfunction, heart rate variability tests were performed and comprised three spectral indices in the frequency domain and four Ewing tests (Valsalva maneuver, orthostatic test, deep breathing test, and orthostatic hypotension test).[29] The electrocardiogram was recorded using electrocardiography equipment (Neurosoft) and dedicated software for heart rate variability analysis (Poly-Spectrum®, Ivanovo, Russia). A questionnaire concerning autonomic dysfunction symptoms was applied, and presence of cardiac autonomic dysfunction was considered when more than two results were abnormal.[29]

Baropodometry Assessment

Baropodometry was performed using a pressure platform Emed-X (Novel GmbH, Munich, Germany) with 1 sensor/cm^2, 400 samples/s, pressure measurement uncertainty ± 5 kPa, flushed to a rubber walkway approximately 7 m long, along which the subject walked barefoot at a self-selected speed. For arch height index assessment, the subject performed a static evaluation standing for 15 s with one foot over the platform and the other over the walkway. One record of static baropodometric data was performed for each foot. For dynamic assessment of baropodometric variables the first-step gait protocol was adopted because of its adequate reproducibility[30] and validity and for foot protection of subjects with feet at risk.[31] Ten successful trials were recorded for each foot. A trial was considered successful if the subject made a clean pressure plate contact using the most habitual gait, without targeting. Measurements were obtained in both walking directions across the platform to minimize the time of data acquisition.

Data Analysis

The values of baropodometric variables peak plantar pressure, stance time, percentage of contact time, percentage of contact area and pressure-time integral were obtained using Novel Scientific (Novel GmbH) software. The plantar region was automatically divided into five regions of interest: heel, midfoot, metatarsals, toes 2 to 5, and hallux. Mean and standard deviation (SD) values for each variable, for each group, were obtained from the average values of the 10 records of both feet for each subject to minimize subject variability of baropodometric data. The arch height index was calculated according to the Cavanagh and Rodgers[32] method, by "geometry" function, based on static baropodometric record.

Statistical Analysis

Data were reported as mean and SD or absolute and relative frequencies. Normality of data distribution was verified by Shapiro–Wilk test. Age, BMI, arch height index, ankle active range of motion for plantar flexion and dorsiflexion movements, and baropodometric data were compared between groups by one-way analysis of variance (ANOVA) and Scheffé *post hoc* test to adjust size samples differences. An analysis of covariance through univariate linear model with Bonferroni confidence interval adjustment was performed to compare baropodometric parameters between groups adjusted for BMI and stance time, as they are considered confounding variables. Between-group comparisons of peak plantar pressure, pressure-time integral, percentage of contact time, and percentage of contact area were adjusted for BMI. Between-group comparison of peak plantar pressure was adjusted for stance time too. The initial ANOVA analysis with Scheffé *post hoc* test were considered, as no significant interference of BMI or stance time was found in the adjusted analysis. The homogeneity of gender among groups was assessed by Chi-square test. Correlation between baropodometric variables and ankle active range of motion was verified by Pearson correlation. A significance level of 5% was adopted. Statistical tests were performed using SPSS software (version 17.0, SPSS Inc., Chicago, IL).

Results

From 87 subjects who consented to participate in the study, 73 fulfilled the entry criteria and were divided into three groups: 15 subjects in the PDG, 28 subjects in the DMG, and 30 subjects in the HG. Causes for exclusion of 14 subjects are reported in **Table 1**.

Table 1.
Number of Subjects Excluded from the Sample Regarding Exclusion Criteria

Cause of exclusion	HG	DMG	PDG
MNSI score ≥4	0	4	0
Foot deformity	1	2[a]	1
Relative legs length >10 mm	1	1	0
Lower limb lesion in the past 6 months	3	0	0
Lower limb ischemic vascular disease	0	2	0
Symptom of vertigo at the day of baropodometric assessment	1	0	0
Total of subjects excluded from the sample by group	6	7	1
Total of subjects excluded from the total sample	14		

[a] The subjects presented more than one cause of exclusion.

Subject Characteristics

The groups were homogeneous regarding gender, age, BMI, and arch height index. The DMG presented an active range of motion for ankle dorsiflexion significantly lower than the other two groups (**Table 2**).

Table 2.
Participant Characterization[a]

	Gender, male/ female	Age, years	BMI, kg/m^2	Arch height index	Active plantar flexion,°	Active dorsiflexion,°
HG (30)	11 (36.3) / 19 (73.7)	51.5 (11.9)	26.8 (3.4)	0.24 (0.06)	35 (4)	25 (2)[b]
DMG (28)	7 (26.7) / 21 (73.3)	54.4 (7.7)	27.9 (3.3)	0.25 (0.03)	33 (4)	21 (2)[c]
PDG (15)	4 (26.7) / 11 (73.3)	54.8 (9.7)	29.3 (3.0)	0.25 (0.02)	34 (2)	24 (4)[b]
p values	0.655[d]	0.227[e]	0.058[e]	0.370[e]	0.208[e]	< 0.001[e]

[a] Data are absolute (relative frequency) or mean (SD). Statistical significance when $p < .05$.
[b] Significant differences after *post hoc* Scheffé in relation to c.
[c] Significant differences after *post hoc* Scheffé in relation to b.
[d] Chi-squared.
[e] One-way ANOVA.

Subclinical Peripheral Neuropathy and Cardiac Autonomic Dysfunction

Regarding subclinical peripheral neuropathy condition assessed by electrophysiological measurements, 52.0% of DMG subjects and 20.0% of PDG subjects presented abnormal electrophysiological results, whereas 43.3% of DMG subjects and 39.9% of PDG subjects presented altered cardiac autonomic test.

Baropodometric Parameters of Prediabetes Subjects Compared with Healthy Subjects and Diabetes Subjects

Peak plantar pressures were significantly higher in metatarsal plantar regions in the PDG and DMG compared with the HG, without significant differences between the DMG and the PDG. Significant differences pertaining to the other plantar regions were not found between groups (**Table 3**).

The stance time was significantly higher in the DMG than in the other groups. As the stance time is related to gait velocity, and in this study the gait velocity was self-selected, we preferred to present the contact time of each plantar region as a percentage of the contact time (**Table 3**).

The percentage of contact time was significantly higher in the hallux in the DMG compared with the other groups. The percentage of contact time of the metatarsals and midfoot plantar regions was significantly higher in the PDG and DMG compared with the HG. This variable was not significantly different between the groups in the heel and toes 2 to 5 (**Table 3**).

Midfoot, metatarsals, toes, and hallux plantar regions have significantly higher values for pressure-time integral in the PDG and DMG compared with the HG, while there was no significant difference between the PDG and the DMG. There was no significant difference between the groups for other plantar regions (**Table 3**).

There was no statistically significant difference between the groups regarding the percentage of contact area at any plantar region (**Table 3**).

No significant positive or negative correlation was found between baropodometric variables and active range of motion for ankle plantar flexion and dorsiflexion for any of the three studied groups.

Discussion

Our main result was that the PDG plantar pressure distribution pattern seemed to be similar to the DMG pattern, in which peak pressures, pressure-time integral, and percentage of contact time were elevated in the metatarsals and midfoot regions compared with the HG. As far as we know, there were no previous studies describing baropodometric variables or gait performance in pre-DM individuals, leading us to believe that the similarity of the patterns found

Table 3.
Baropodometric Parameters of the Healthy Group, the Type 2 Diabetes Mellitus Group, and the Prediabetes Group[a]

Plantar region	HG (30)	DMG (28)	PDG (15)	*p* values
Peak pressure, kPa				
Heel	316 (59)[b]	326 (84)[b]	311 (64)[b]	0.896
Midfoot	144 (31)[b]	161 (43)[b]	173 (43)[b]	0.051
Metatarsal	406 (69)[b]	482 (109)[c]	509 (109)[c]	0.010
Toes 2 to 5	104 (38)[b]	98 (75)[b]	99 (36)[b]	0.305
Hallux	279 (95)[b]	283 (105)[b]	336 (107)[b]	0.209
Stance time, ms	924 (130)[b]	1038 (144)[c]	983 (101)[b]	0.017
Contact time, % roll over process				
Heel	65 (7)[b]	67 (6)[b]	66 (6)[b]	0.569
Midfoot	70 (7)[b]	73 (6)[c]	76 (3)[c]	0.006
Metatarsal	88 (2)[b]	91 (3)[c]	90 (3)[c]	0.004
Toes 2 to 5	52 (1)[b]	59 (14)[b]	56 (18)[b]	0.313
Hallux	6.5 (1)[b]	7.3 (9)[c]	6.9(1)[d]	0.041
Pressure-time integral, kPa/s				
Heel	115 (27)[b]	135 (44)[b]	124 (39)[b]	0.205
Midfoot	61 (21)[b]	80 (30)[c]	87 (24)[c]	0.004
Metatarsal	156 (31)[b]	222 (64)[c]	205 (53)[c]	<0.001
Toes 2 to 5	76 (32)[b]	99 (44)[c]	116 (55)[d]	0.002
Hallux	30 (4)[b]	36 (19)[c]	33 (16)[d]	0.039
Contact area, % total contact area				
Heel	25 (2)[b]	25 (4)[b]	24 (2)[b]	0.403
Midfoot	20 (3)[b]	20 (5)[b]	22 (2)[b]	0.090
Metatarsal	37(2)[b]	38 (3)[b]	37 (2)[b]	0.206
Toes 2 to 5	7 (2)[b]	6 (2)[b]	6 (2)[b]	0.205
Hallux	8 (1)[b]	8 (1)[b]	8 (2)[b]	0.868

[a] Data reported as mean (SD). Statistical significance when $p < .05$.
[b,c,d] Different letters in the same line are used to denote where the between groups significant difference was found after *post hoc* Scheffé. Equal letters denote no significant difference between groups.

between the PDG and DMG could be related to the hyperglycemic status when factors such as age, BMI, and foot characteristics were controlled between groups.

The increase of plantar pressure values for the anterior plantar region in DM subjects has been associated with loss of protective sensation[12–18] as well as foot deformities[19] and vascular complications.[13,14] Nevertheless, before clinical manifestation of neuropathy in early type 2 DM subjects with no vascular and foot complications, Pataky and coauthors[33] found an increase in plantar pressure under the hallux and the fifth metatarsal head, whereas it was significantly lower in the heel when compared with the healthy controls. They concluded that an anterior displacement of weight-bearing during walking as well as an increased contact time of the plantar surface in DM patients without evidence of

any complications could be a premature sign of peripheral neuropathy, which the clinical examination or quantitative sensory testing were not able to identify.

In our study, we evaluated DM subjects diagnosed for 2 to 10 years and pre-DM subjects diagnosed for 1 to 5 years, without loss of foot protective sensation, while some of them presented altered sensorimotor NCT results. Considering the results of Pataky and coauthors,[33] it is possible that the similarity of the patterns found between these two groups could be related to a subclinical neuropathy.

Although MNSI score, calibrated tuning fork, classical NCTs, and vibration and temperature perception thresholds are the most commonly used tests in clinical practice, they might not detect neuropathy in pre-DM people.[34] On the other hand, distal intra-epidermal nerve fiber density, quantitative sudomotor testing, total sweat volume, arm-to-foot sweat responses, deep tendon reflexes, and temperature sensation are sensitive markers of sensorimotor neuropathy in early DM and pre-DM patients.[35,36]

It is suggested that small demyelinated fibers might be implicated in IGT and early diabetic neuropathy.[37,38] Therefore, sensory impairment is more pronounced than motor impairment in pre-DM.[38,39] However, barefoot gait depends on the speed and quality of information from sensory plantar receptors and joint proprioceptors, and this could lead to changes in the reciprocal motor activation, altering the dynamics of movement of these individuals,[18] which could be related to the pattern of plantar distribution found in the PDG and the DMG.

The cardiac autonomic dysfunction was also present in the DMG but mainly in the PDG, corroborating the strong evidence for the association between autonomic impairment and prediabetes.[34] The evaluation of cardiac autonomic dysfunction brings information about the neurovegetative system, which plays an important role in the development of plantar lesions. Alterations on peripheral vasomotor and sudomotor function lead to dryer skin, which, along with mechanical stress caused by increased values of plantar pressure, increases the risk for foot complications.[40]

The reduction in feet joint mobility, as a consequence of the tissue stiffness that affects joints as well as the decline in strength and muscle activation,[18] is associated with the increased metatarsal load. Such alterations are commonly found in individuals with DM and peripheral neuropathy.[20] In our study, only the DMG presented significantly decreased active range of motion for ankle dorsiflexion, but no positive or negative significant correlation with baropodometric parameters were found.

Another factor contributing to plantar stress is the plantar time of ground contact.[20,21] Previous studies have already demonstrated that individuals with DM with[41] or without chronic neuropathy and foot deformities have a significantly slower barefoot walking speed than healthy age–gender-matched individuals.[42] A cautious walking pattern is adopted, decreasing walking speed by adapting temporal gait variables such as step time, cadence, or an increased double support time during barefoot walking, which results in a decrease in peak plantar pressures that could be more expressive in a faster gait.[41,42] In contrast with the DMG, the PDG did not decrease their walking speed but increased the contact time of their midfoot and metatarsal regions, overloading them during gait.

Foot deformities, changes in posture, and arch height are confounding factors of plantar pressure distribution patterns even in healthy individuals.[43,44] For this reason, we excluded individuals with deformities in the feet and those who had relative difference between lower limbs length. In addition, we have sought to maintain the incidence of arch height index homogeneous between the groups. This screening resulted in no differences between groups for any of the evaluated plantar regions regarding the percentage of contact area.

The main limitation of our study is the sample size, which was not sufficient to allow a regressive statistical analysis to verify the effect of subclinical neuropathy influences in the plantar pressure distribution pattern. Nevertheless, none of the subjects presented clinical neuropathy characterized by loss of foot protective sensation.

The fact that PDG baropodometric parameters have similarities with those of the DMG has been described here for the first time. However, our study does not allow generalizations, because it is necessary to explore other factors

beyond subclinical neuropathy. Further studies of gait dynamics or exploring factors associated to structural tissue protein glycation in pre-DM individuals are necessary to understand the similarity of the distribution patterns of plantar pressure in DM and pre-DM and individuals. In addition, studies assessing baropodometric parameters of pre-DM during shod conditions are also necessary, considering that this is the status of the feet during most of daily life activities.

Orientation is an important aspect in the management and prevention of foot complications, and baropodometric assessment is a technological tool that helps in these processes. Pre-DM individuals are susceptible to peripheral neuropathy and may already present changes in the values of plantar pressure and its distribution pattern. This fact reinforces the importance of early diagnosis and treatment of this condition and brings attention to foot care in these patients.

Conclusion

In this study, pre-DM subjects presented plantar pressure distribution patterns similar to those of DM subjects, except for the stance time, which did not differ from healthy subjects. As far as we know, this fact has not been previously described and reinforces the importance of early diagnosis and treatment of both DM and pre-DM and conditions, as well as the attention to foot care in these patients.

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References:

1. American Diabetes Association. Diagnosis and classification of diabetes mellitus. Diabetes Care. 2013;36 Suppl 1:S67–74.
2. Nathan DM, Davidson MB, DeFronzo RA, Heine RJ, Henry RR, Pratley R, Zinman B; American Diabetes Association. Impaired fasting glucose and impaired glucose tolerance: implications for care. Diabetes Care. 2007;30(3):753–9.
3. International Diabetes Federation. Diabetes atlas. 5th ed. 2012 Update sheet. http://www.idf.org/diabetes-atlas-2012-update-out-now. Accessed April 15, 2013.
4. Shaw JE, Sicree RA, Zimmet PZ. Global estimates of the prevalence of diabetes for 2010 and 2030. Diabetes Res Clin Pract. 2010;87(1):4–14.
5. Singleton JR, Smith AG. Therapy insight: neurological complications of prediabetes. Nat Clin Pract Neurol. 2006;2(5):276–82.
6. Shaw JE, Zimmet PZ. The epidemiology of diabetic neuropathy. Diabetes Rev. 1999;7(4):245–52.
7. Singleton JR, Smith AG. Neuropathy associated with prediabetes: what is new in 2007? Curr Diab Rep. 2007;7(6):420–4.
8. Buysschaert M, Bergman M. Definition of prediabetes. Med Clin North Am. 2011;95(2):289–97.
9. Andersen H, Nielsen S, Mogensen CE, Jakobsen J. Muscle strength in type 2 diabetes. Diabetes. 2004;53(6):1543–8.
10. Petrofsky J, Macnider M, Navarro E, Lee S. Motor control and gait characteristics in people with type 1 and type 2 diabetes without sensory impairment in the foot. Basic Appl Myol. 2005;15(2):75–86.
11. Boulton AJ, Vinik AI, Arezzo JC, Bril V, Feldman EL, Freeman R, Malik RA, Maser RE, Sosenko JM, Ziegler D; American Diabetes Association. Diabetic neuropathies: a statement by the American Diabetes Association. Diabetes Care. 2005;28:956–62.
12. Cavanagh PR, Derr JA, Ulbrecht JS, Maser RE, Orchard TJ. Problems with gait and posture in neuropathic patients with insulin-dependent diabetes mellitus. Diabet Med. 1992;9(5):469–74.
13. Payne C, Turner D, Miller K. Determinants of plantar pressures in the diabetic foot. J Diabetes Complications. 2002;16(4):277–83.
14. Boulton AJ, Hardisty CA, Betts RP, Franks CI, Worth RC, Ward JD, Duckworth T. Dynamic foot pressure and other studies as diagnostic and management aids in diabetic neuropathy. Diabetes Care. 1983;6(1):26–33.
15. Luger E, Nissan M, Karpf A, Steinberg E, Dekel S. Dynamic pressures on the diabetic foot. Foot Ankle Int. 2001;22(9):715–9.
16. Caselli A, Pham H, Giurini JM, Armstrong DG, Veves A. The forefoot-to-rearfoot plantar pressure ratio is increased in severe diabetic neuropathy and can predict foot ulceration. Diabetes Care. 2002;25(6):1066–71.
17. Melai T, Ijzerman TH, Schaper NC, de Lange TL, Willems PJ, Meijer K, Lieverse AG, Savelberg HH. Calculation of plantar pressure time integral, an alternative approach. Gait Posture. 2011;34(3):379–83.
18. Allet L, Armand S, Golay A, Monnin D, De Bie RA, De Bruin ED. Gait characteristics of diabetic patients: a systematic review. Diabetes Metab Res Rev. 2008;24(3):173–91.

19. Bus SA, Yang QX, Wang JH, Smith MB, Wunderlich R, Cavanagh PR. Intrinsic muscle atrophy and toe deformity in the diabetic neuropathic foot: a magnetic resonance imaging study. Diabetes Care. 2002;25(8):1444–50.

20. Rao S, Saltzman CL, Yack HJ. Relationships between segmental foot mobility and plantar loading in individuals with and without diabetes and neuropathy. Gait Posture. 2010;31(2):251–5.

21. Zou D, Mueller MJ, Lott DJ. Effect of peak pressure and pressure gradient on subsurface shear stresses in the neuropathic foot. J Biomech. 2007;40(4):883–90.

22. Wrobel JS, Najafi B. Diabetic foot biomechanics and gait dysfunction. J Diabetes Sci Technol. 2010;4(4):833–45.

23. Bongaerts BW, Rathmann W, Heier M, Kowall B, Herder C, Stöckl D, Meisinger C, Ziegler D. Older subjects with diabetes and prediabetes are frequently unaware of having distal sensorimotor polyneuropathy: the KORA F4 study. Diabetes Care. 2013;36(5):1141–6.

24. Lunetta M, Le Moli R, Grasso G, Sangiorgio L. A simplified diagnostic test for ambulatory screening of peripheral diabetic neuropathy. Diabetes Res Clin Pract. 1998;39(3):165–72.

25. World Medical Association. Declaration of Helsinki: ethical principles for medical research involving human subjects, as amended by the 52nd WMA Assembly, Edinburgh, Scotland, October 2000; Note of clarification in paragraph 29 added by the WMA General Assembly, Washington DC; 2002.

26. England JD, Gronseth GS, Franklin G, Miller RG, Asbury AK, Carter GT, Cohen JA, Fisher MA, Howard JF, Kinsella LJ, Latov N, Lewis RA, Low PA, Sumner AJ; American Academy of Neurology; American Association of Electrodiagnostic Medicine; American Academy of Physical Medicine and Rehabilitation. Distal symmetric polyneuropathy: a definition for clinical research: report of the American Academy of Neurology, the American Association of Electrodiagnostic Medicine, and the American Academy of Physical Medicine and Rehabilitation. Neurology. 2005;64(2):199–207.

27. Souza A, Nery CA, Marciano LH, Garbino JA. Avaliação da neuropatia periférica: correlação entre a sensibilidade cutânea dos pés, achados clínicos e eletroneuromiográficos. Acta Fisiatr. 2005;12(3):87–93.

28. Albers JW, Herman WH, Pop-Busui R, Martin CL, Cleary P, Waberski B; Diabetes Control and Complications Trial (DCCT)/Epidemiology of Diabetes Intervention and Complications (EDIC) Research Group. Subclinical neuropathy among Diabetes Control and Complications Trial participants without diagnosable neuropathy at trial completion: possible predictors of incident neuropathy? Diabetes Care. 2007;30(10):2613–8.

29. Rolim LC, Sá JR, Chacra AR, Dib SA. Diabetic cardiovascular autonomic neuropathy: risk factors, clinical impact and early diagnosis. Arq Bras Cardiol. 2008;90(4):e24-31.

30. Peters EJ, Urukalo A, Fleischli JG, Lavery LA. Reproducibility of gait analysis variables: one-step versus three-step method of data acquisition. J Foot Ankle Surg. 2002;41(4):206–12.

31. Bus SA, de Lange A. A comparison of the 1-step, 2-step, and 3-step protocols for obtaining barefoot plantar pressure data in the diabetic neuropathic foot. Clin Biomech (Bristol, Avon). 2005;20(9):892–9.

32. Cavanagh PR, Rodgers MM. The arch index: a useful measure from footprints. J Biomech. 1987;20(5):547–51.

33. Pataky Z, Assal JP, Conne P, Vuagnat H, Golay A. Plantar pressure distribution in Type 2 diabetic patients without peripheral neuropathy and peripheral vascular disease. Diabet Med. 2005;22(6):762–7.

34. Tabák AG, Herder C, Rathmann W, Brunner EJ, Kivimäki M. Prediabetes: a high-risk state for diabetes development. Lancet. 2012;379(9833):2279–90.

35. Smith AG, Russell J, Feldman EL, Goldstein J, Peltier A, Smith S, Hamwi J, Pollari D, Bixby B, Howard J, Singleton JR. Lifestyle intervention for pre-diabetic neuropathy. Diabetes Care. 2006;29(6):1294–9.

36. Grandinetti A, Chow DC, Sletten DM, Oyama JK, Theriault AG, Schatz IJ, Low PA. Impaired glucose tolerance is associated with postganglionic sudomotor impairment. Clin Auton Res. 2007;17(4):231–3.

37. Tesfaye S, Boulton AJ, Dyck PJ, Freeman R, Horowitz M, Kempler P, Lauria G, Malik RA, Spallone V, Vinik A, Bernardi L, Valensi P; Toronto Diabetic Neuropathy Expert Group. Diabetic neuropathies: update on definitions, diagnostic criteria, estimation of severity, and treatments. Diabetes Care. 2010;33(10):2285–93.

38. Papanas N, Ziegler D. Prediabetic neuropathy: does it exist? Curr Diab Rep. 2012;12(4):376–83.

39. Ziegler D, Rathmann W, Dickhaus T, Meisinger C, Mielck A; KORA Study Group. Prevalence of polyneuropathy in pre-diabetes and diabetes is associated with abdominal obesity and macroangiopathy: the MONICA/KORA Augsburg Surveys S2 and S3. Diabetes Care. 2008;31(3):464–9.

40. Low PA, Vernino S, Suarez G. Autonomic dysfunction in peripheral nerve disease. Muscle Nerve. 2003;27(6):646–61.

41. Courtemanche R, Teasdale N, Boucher P, Fleury M, Lajoie Y, Bard C. Gait problems in diabetic neuropathic patients. Arch Phys Med Rehabil. 1996;77(9):849–55.

42. Ko M, Hughes L, Lewis H. Walking speed and peak plantar pressure distribution during barefoot walking in persons with diabetes. Physiother Res Int. 2012;17(1):29–35.

43. Gravante G, Pomara F, Russo G, Amato G, Cappello F, Ridola C. Plantar pressure distribution analysis in normal weight young women and men with normal and claw feet: a cross-sectional study. Clin Anat. 2005;18(4):245–50.

44. Teyhen DS, Stoltenberg BE, Collinsworth KM, Giesel CL, Williams DG, Kardouni CH, Molloy JM, Goffar SL, Christie DS, McPoil T. Dynamic plantar pressure parameters associated with static arch height index during gait. Clin Biomech (Bristol, Avon). 2009;24(4):391–6.