ORIGINAL PAPER

doi: 10.5455/medarh.2019.73.262-267
MED ARCH. 2019 AUG; 73(4): 262-267
RECEIVED: JUN 12, 2019 | ACCEPTED: AUG 12, 2019

Influence of Diabetic Distal Symmetric Polyneuropathy on the Performance of the Musculoskeletal System of Lower Leg and Foot

Snjezana Novakovic Bursac[1], Slavica Jandric[2], Goran Talic[1]

[1]Institute for Physical Medicine and Rehabilitation „Dr Miroslav Zotovic“, Banja Luka, Bosnia and Herzegovina

[2]Medical faculty, Universitiy of Banja Luka, Banja Luka, Bosnia and Herzegovina

Corresponding author: Snjezana Novakovic Bursac, MD, MMs. Institute for physical medicine and rehabilitation „Dr Miroslav Zotovic”, Banja Luka, Bosnia and Herzegovina. E-mail address: snjezana.nb@ms.zotovicbl.org. ORCID ID https://orcid.org/0000-0001-7499-8537.

ABSTRACT

Introduction: Complications on the lower extremities are a major cause of morbidity, disability, emotional and physical suffering in people with diabetes. Diabetic neuropathy (DN) is the most frequent complication of both types of diabetes. Lack of performance of the musculoskeletal system of lower leg and foot can results in high focal plantar pressures with increased ulceration risk in patients with neuropathy. **Aim:** To determine the impact of the severity of distal symmetric polyneuropathy (DSPN) on the foot and ankle muscle strength and the range of motion (ROM) at ankle joint (AJ), subtalar joint (SJ) and first metatarsophalangeal joint (I MTP). **Methods:** A cross-sectional study was conducted among 100 diabetic patients. The level of DSPN was assessed using the Neuropathy Disability Score. Function of ten foot and ankle muscles has been evaluated by manual muscle testing. Muscle strength was scored by semiquantitative grading system used in the Michigan Diabetic Neuropathy Score. ROM at the AJ, SJ and I MTP was measured with goniometer. **Results:** The average patients age was 61.91±10.74 and diabetes duration 12.25±8.60 years. DSPN was present in 45% of patients. The average strength of foot and ankle muscles expressed by muscle score was 11.56±5.08. The average ROM at AJ was 47.85°, at SJ 35.10° and at I MTP 72.70°. Correlations between the severity of the DSPN and muscle function, ROM at AJ, SJ and I MTP were statistically significant. ROM at SJ and I MTP declines significantly with progression of neuropathy but not significant at AJ. **Conclusion:** The severity of DSPN is significantly associated with foot and ankle muscle weakness and ROM at the SJ and the I MTP, but not significantly with the ROM at the AJ.

Keywords: Diabetes Mellitus, complications, muscles.

1. INTRODUCTION

Diabetes mellitus (DM), or diabetes is the global epidemic of the 21st century and it is now the fourth leading cause of death in most developed countries (1). Diabetes complications on the lower extremities are a major cause of morbidity, disability, emotional and physical suffering in people with DM (2) generating huge economic costs for patients, their families and the entire society (3).

Diabetic neuropathy (DN) is the most frequent complication of both types of DM and is present to some degree in more than 50% of diabetic persons older than 60 years (4, 5). DN affects different sets of lower-limb nerve fibers and leads to a variety of clinical manifestations (5-7). Distal symmetrical polyneuropathy (DSPN) is thought to be the most common variety of DN. The typical DSPN is a chronic, symmetrical, length-dependent sensorimotor polyneuropathy. Internationally agreed definition of DSPN for clinical practice is as follows: the presence of symptoms and/or signs of dysfunction of peripheral nerves in patients with DM after excluding the other possible causes (7).

Limited joint mobility (LJM) at ankle joint (AJ), subtalar joint (SJ), and first metatarsophalangeal joint (I MTP) results in high focal plantar pressures with increased ulceration risk in patients with neuropathy (8). Range of motion (ROM) restriction associated with a lack of protective sensation and foot deformities may even increase the force and mechanical stress exposure under the patient's foot (9). LJM is often overlooked because it causes small disability and is therefore thought to be of little clinical consequence. Determination of the foot and ankle joint mobility is a simple and rather exact test to identify diabetic patients with

an at-risk foot and, might be useful as a screening tool in diabetic patients to identify those with an at-risk foot because of its price and simplicity (10, 11).

Motor dysfunction in patients with DM can be detected as muscle weakness as well as atrophy of muscular tissue. It is usually found distally in the extremities, primarily in the lower extremities and it is believed to be caused primarily by DN (12). Foot muscle atrophy is closely related to severity of DN. Since DN shows a centripetal pattern of progression, quantification of the more distally situated foot muscles could possibly serve as an early marker for motor dysfunction in DN (13).

The association among ROM restriction, muscle strength and function loss can lead to altered foot rollover during gait, as their integrity is needed to enable proper load absorption (9). Elevated plantar pressure coupled with a longer period of time spent in support phase in DN patients contributes to the susceptibility for skin damage through the prolonged mechanical load on tissue leading to skin breakdown and ulceration (14).

Keeping in mind serious consequences that complications of DM on the lower extremities make on a personal and social level, inevitably raises the question what can be done to reduce their rate and severity. The implementation of simple and low-cost strategies can reduce the rate of diabetic complication on the lower extremities (15, 16) . Proper metabolic control of both types of DM may delay the onset and progression of diabetic complications (17).

2. AIM

To determine the impact of the severity of DSPN on the foot and ankle muscle strength and the ROM at AJ, STJ and I MTP.

3. METHODS

A cross-sectional study was conducted among diabetic patients (both type DM) registered at the family physicians in the Public Primary Health Centre Banja Luka, Bosnia and Herzegovina, during the 2014. The study included 100 diabetic patients. The sample was formed in a way that the patients who approached family doctor for a prescription for insulin or oral antidiabetic drugs in 10 family medicine ambulances were over the time successively asked to enter the study. The survey included: review of medical records, history-taking, measurement and testing.

Medical records were source of personal data, data on DM-type, therapy and HbA1c value not older than 6 months. History-taking data were entered in the anamnestic list and included information about the duration and the past treatment of DM (18, 19). The clinical examinations of muscles and joints were performed routinely by the same observer.

The level of DSPN was assessed by using the Neuropathy Disability Score (NDS) (20, 21, 22). The NDS was derived from examination of ankle reflexes using a tendon hammer, vibration perception on the great toe using a 128-Hz tuning fork, pin-prick perception using standard neurotips on the dorsal surface of the great toe, and temperature perception on the dorsal surface of the metatarsal heads using warm and cool rods. The sensory modalities were scored as either present (score of 0) or reduced/absent (score of 1) for each side, and reflexes as normal (score of 0), present with reinforcement (score of 1), or absent (score of 2) per side. The maximum score is 10, whereas a score of 0 represents a totally normal peripheral nervous system examination, a score of 3-5 represents mild neuropathy, a score of 6-8 moderate neuropathy and a score of 9-10 represents severe neuropathy (21, 23, 24). Patients were diagnosed as having DSPN if they had NDS ≥6 (24).

Muscle function of the foot and ankle muscles has been evaluated by manual muscle testing (MMT) on the dominant leg applying the scoring system as used in the Michigan Diabetic Neuropathy Score (MDNS) (9, 22, 25). MMT means assessing ability of the muscle to produce active movement against the examiner's resistance. Muscle weakness was scored as 0 for normal muscle strength, 1 for mild, 2 for severe weakness, and 3 for complete loss of strength. A muscle score (MS) was, therefore, obtained for each set of muscles examined. Higher values for this score represented increased muscle weakness (25, 26). In the positions described for manually clinical assessment (27) the function of the following muscles was evaluated: triceps surae, tibialis anterior, interosseus, lumbrical, flexor hallucis brevis, extensor digitorum brevis, extensor digitorum longus, flexor digitorum brevis, extensor hallucis longus and extensor hallucis brevis (9).

Joint mobility at the AJ, SJ and I MTP was measured with a goniometer on the dominant leg (10, 11, 28, 29).

At the AJ range of motion (ROM) was measured with the patient supine and goniometer set with immobile prong in line with calf, mobile prong in line with external edge of the foot and center of goniometer above the joint center. The maximum range of active talar flexion and extension in was measured and the sum of the values was recorded as ROM at the AJ (10).

At the SJ ROM was measured with the patient pronate; a vertical line was marked on the patient's skin from heel to midcalf; goniometer set with immobile prong in line with the line on the calf, mobile prong in line with the line on the heel and center of goniometer above the joint center; the maximum range of active calcaneal inversion and eversion were measured with a goniometer and the sum of the values was recorded as ROM at the SJ (10, 11).

At the I MTP range of active extension to plantar flexion was measured with the patient in the supine position; horizontal line was drawn from the first toe to the heel in line with medial edge of the foot; goniometer center set above the joint center, immobile prong in line with proximal part of drawn line and mobile prong in line with the distal part of the line; the sum of maximal extension and flexion was recorded as ROM at I MTP (10, 11).

The statistical analyses were done using the software package "IBM SPSS Statistics". To test the statistical significance between variables the ANOVA and the Tukey post-hoc test were applied. The relationship and the relationship strength of various parameters were assessed by

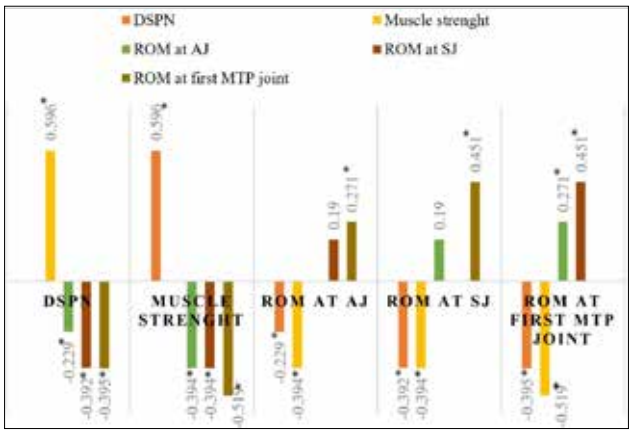

Figure 1. Pearson's correlation between the severity of the DSPN and muscle function, ROM at AJ, SJ and I MTP joint. Pearson coefficient is statistically significant in all relationships that were observed, except for relations between ROM at AJ and SJ. *p<0,05. DSPN–distal symmetrical polyneuropathy, AJ – ankle joint, SJ – subtatalar joint, I MTP – first metatarsophalangeal joint, ROM–range of motion

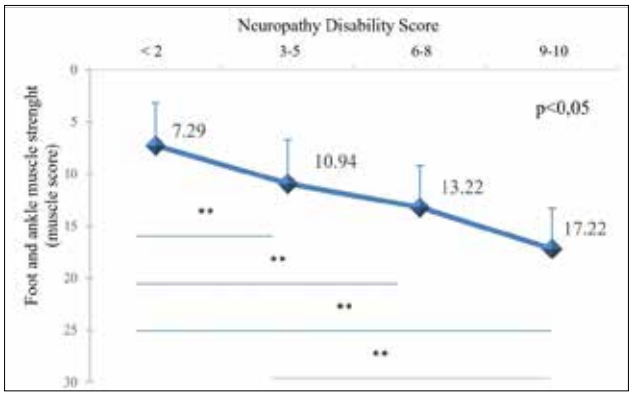

Figure 2. Foot and ankle muscle strength in groups of patients with different stages of diabetic neuropathy. Strength of ankle and foot muscle significantly declines with progression of neuropathy; p<0,05. ** post-hoc Tukey test p<0,05

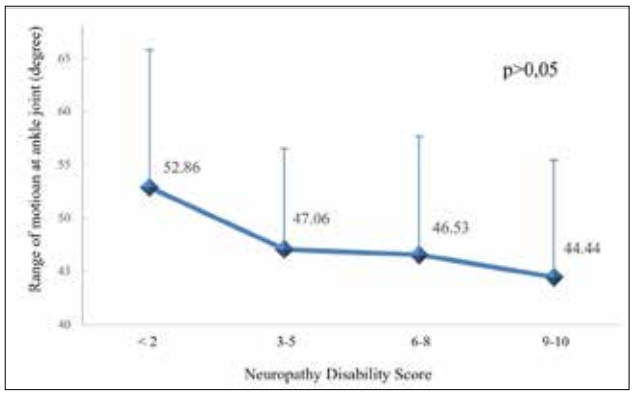

Figure 3. Range of motion at ankle joint in groups of patients with different stages of diabetic neuropathy. Range of motion at ankle joint in groups of patients with different stages of neuropathy declines with progression of neuropathy, but not significantly; p>0,05.

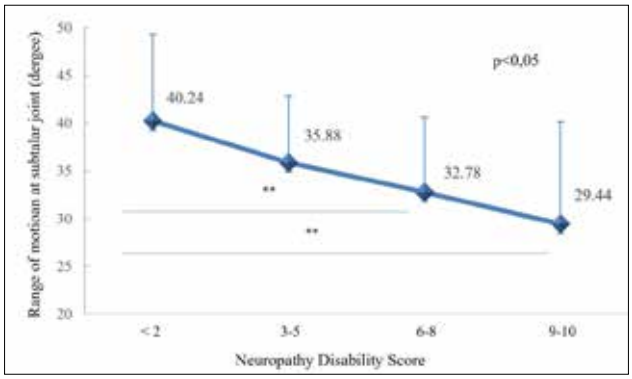

Figure 4. Range of motion at subtalar joint in groups of patients with different stages of diabetic neuropathyRange of motion at subtalar joint in groups of patients with different stages of neuropathy significantly declines with progression of neuropathy; p<0,05. ** post-hoc Tukey test p<0,05

Pearson´s correlation. The cut off for the results significance was p<0.05.

4. RESULTS

In the study group were more women (53%) than men (47%). The average age of the patients was 61,91±10,74 years. The majority of patients (94%) had DM type 2 and type 1 DM had 6% of them. The average diabetes duration was 12,25± 8,60 years. The even number of patients was treated with insulin and antidiabetic drugs (46%), and combined therapy with insulin and the antidiabetic drugs had 8% of patients.

DSPN was present in 45% of patients. The average foot and ankle muscles expressed by MS was 11,56±5,05. Average ROM at AJ was 47,85°±11,1, at SJ 35,10°±8,55 and at I MTP 72,70°±21. There were statistically significant correlations between the severity of the DSPN and muscle function, ROM at AJ, SJ and I MTP. Pearson coefficient is statistically significant in all relationships that were observed, except for relations between ROM at AJ and SJ that is shown at Figure 1.

The average muscle strength in the group of patients without neuropathy (NDS < 2) was 7,28±4,12. Muscle strength in the group of patients who had mild neuropathy (NDS 3-5) was 10,94±4,23, in the group pf patients who had moderate neuropathy (NDS 6-8) was 13,22±4,01 and in the group of patients who had severe neuropathy (NDS 9-10) muscle strength was 17,22±3,93. Strength of ankle and foot muscle significantly declines with progression of neuropathy that is shown at Figure 2.

The average ROM at AJ in the group of patients without neuropathy (NDS < 2) was 52,86°±12,9. ROM at AJ in the group of patients who had mild neuropathy (NDS 3-5) was 47,06°±9,5, in the group pf patients who had moderate neuropathy (NDS 6-8) was 46,53°±11,10 and in the group of patients who had severe neuropathy (NDS 9-10) ROM at AJ was 44,44°±11. ROM at AJ declines with progression of neuropathy but not significantly as it is shown at Figure 3.

The average ROM at SJ in the group of patients without neuropathy (NDS < 2) was 40,24°±9,1. ROM at SJ in the group of patients who had mild neuropathy (NDS 3-5) was 35,88°±7, in the group pf patients who had moderate neuropathy (NDS 6-8) was 32,78°±7,9 and in the group of patients who had severe neuropathy (NDS 9-10) ROM at SJ was 29,44°±10,7. ROM at SJ significantly declines with progression of neuropathy that is shown at Figure 4.

The average ROM at I MTP in the group of patients without neuropathy (NDS < 2) was 89,05°±13,6. ROM at

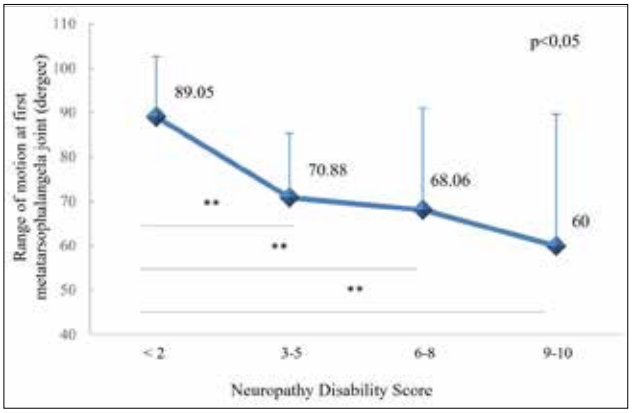

Figure 5. Range of motion at first metatarsophalangeal joint in the groups of patients with different stages of diabetic neuropathy. Range of motion at first metatarsophalangeal joint in the groups of patients with different stages of diabetic neuropathy significantly declines with progression of neuropathy; p<0,05. ** post-hoc Tukey test p<0,05

I MTP in the group of patients who had mild neuropathy (NDS 3-5) was 70,88°±14,4, in the group pf patients who had moderate neuropathy (NDS 6-8) was 68,06°±23 and in the group of patients who had severe neuropathy (NDS 9-10) ROM at I MTP was 60°±29,7. ROM at I MTP significantly declines with progression of neuropathy that is shown at Figure 5.

5. DISCUSSION

Muscle strength of the foot and ankle muscles in this study has been evaluated in ten muscles applying the scoring system as used in the MDNS. Mean MS of evaluated muscles was 11,56±5,05 and represents mild muscle weakness in the study group. Only 13% of patients had preserved muscle strength, 23% of patients had severe muscle weakness, none of them had complete loss of strength, while the most of patients (64%) had mild muscle weakness. Data from this study are consistent with the results of research that has been done by Andersen [12,30], Andreassen [20], Giacomozzi [31] and Camargo [32] that confirmed the existence of a decrease in muscle strength in people with DM, especially in the region of ankle and knee. The muscle weakness in the lower leg can be explained by the presence of the typical DSPN that is length-dependent and more sever in the distal part of the leg [5].

This study has proved a strong relationship between the presence of the DSPN and the foot and ankle muscle weakness. This relationship was confirmed by numerous studies as led by Andreassen [29,33] and by Andersen [12,13,30]. Muscle weakness is associated with the atrophy of ankle and foot muscles due to denervation caused by loss of motor axons combined with insufficient reinnervation more than demyelination process [20], and the same was found by Balducci [34]. Van Shie and his associates also found that the muscle weakness in patients with DM is caused by incomplete reinnervation after axonal degeneration [25]. Andreassen found a correlation between isokinetic muscle strength in ankle and DSPN evaluated by standardized clinical examination, while in the prospective study found that people with DM have a more rapid decline in muscle strength over time compared with those suffering from DM without neuropathy and healthy individuals. Decrease in muscle strength in patients with DM is 3-4% per year and is related to the severity of neuropathy [20]. In another study Andreassen and his associates, following long-term effects of DN on the muscular system, confirmed that in patients with DN there is an apparent distal-proximal gradient with a significant loss of muscle mass in distal segment of the lower extremities, and that in patients with DM there is a correlation between the presence of neuropathies and decreased muscle strength [33].

Data from this study have shown that the average ROM at ankle joint reduces with the increase of the NDS score, i.e., with the severity of DSPN but not significantly, while the relationship between the severity of the DSPN and the ROM at subtalar and I MTP is significantly correlated that is consistent with data from the literature. By her research on ankle mobility during foot rollover in DN patients Sacco confirmed that people with DSPN have reduced active ankle range of motion and dynamic ankle flexion at heel–strike as well as reduced amplitude (flexion–extension) when compared to non-diabetic subjects [35]. Lazaro-Martinez has found that in patients with DM neuropathy presence significantly affects the ROM at I MTP [36]. Hajrasouliha has proved that ROM at ankle joint is one of the late complications of DM [37]. ROM at AJ and I MTP is essentially seen as a risk factors for foot ulceration because it plays an important role in the redistribution of pressure during the support phase of gait [10]. Sacco has found that anterior areas of the foot in patients with DN had altered role receiving higher loads at push-off phase that were probably due to smaller ankle flexion at support phase. This data may explain the higher loads in anterior areas of the foot observed in DN patients [35] and give an opportunity to minimize the consequences of the DM complication on the lower extremities through an active approach in prevention and treatment using evidence based physical and medical exercise therapy [38,39]. In patients with DSPN physiotherapy discreetly changed the foot rollover towards a more physiological process, supported by improved plantar pressure distribution and better functional condition of the foot ankle complex [39]. Specific gait and balance training in combination with functionally oriented strengthening may improve gait and balance, muscle strength, and increase the joints mobility in patients with DM [39,40].

6. CONCLUSION

The severity of diabetic symmetric polyneuropathy is significantly associated with foot and ankle muscle weakness and range of motion at the subtalar joint and the first metatarsophalangeal joint, but not significantly with the range of motion at the ankle.

- **Acknowledgments:** We would like to thank to colleagues from the Public Primary Health Centre Banja Luka and the Institute for physical medicine and rehabilitation „Dr Miroslav Zotović“ for help and support in carrying out study.

- **Authors contribution:** Each author gave substantial contributions to the conception or design of the work in acquisition, analysis, or interpretation of data for the work. Each author had a part in article preparing for drafting or revising it critically for important intellectual content, and each author gave final approval of the version to be published and agreed to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.
- **Declaration of patient consent:** The authors certify that they have obtained all appropriate patient consent forms.
- **Conflict of interest:** There are no conflicts of interest.
- **Financial support and sponsorship:** Nil.

REFERENCES

1. International Diabetes Federation and International Working Group on the Diabetic Foot. Diabetes and Foot Care: Time to Act. Brussels (Belgium): International Diabetes Federation, 2005.
2. International Diabetes Federation. The diabetic foot: amputations are preventable. Position Statement. Brussels, Belgium: International Diabetes Federation, 2015.
3. American Diabetes Association. Preventive foot care in diabetes. Diabetes Care 2004; 27(1): 63-64.
4. Singh N, Armstrong DG, Lipsky BA. Preventing foot ulcers in patients with diabetes. JAMA. 2005; 293: 217-222.
5. Tesfaye S, Boulton AJ, Dyck PJ, Freeman R, Horowitz M, Kempler P. et al, Toronto Dibaetic Neuropathy Expert Group. Diabetic Neuropathies: Update on Definitions, Diagnostic Criteria, Estimation of Severity, and Treatments. Diabetes Care. 2010; 33: 2285-2293.
6. International Working Group on the Diabetic Foot. International Consensus on the Diabetic Foot: Practical Guidelines on the Management and the Prevention of the Diabetic Foot [CD-ROM]. Amsterdam, the Netherlands: International Working Group on the Diabetic Foot, 2005.
7. Boulton AJ, Vinik AI, Arezzo JC, Bril V, Feldman EL, Freeman R, et al. American Diabetes Association. Diabetic neuropathies: a statement by the American Diabetes Association. Diabetes Care. 2005; 28(4): 956-962.
8. Frykberg RG, Zgonis T, Armstrong DG, Driver VR, Giurini JM, Kravitz SR. et al. Diabetic foot disorders. A clinical practice guideline (2006 revision). J Foot Ankle Srg. 2006; 45(5): 1-66.
9. Sartor CD, Watari R, Pássaro AC, Picon AP, Hasue RH. et Sacco ICN. Effects of a combined strengthening, stretching and functional training program versus usual-care on gait biomechanics and foot function for diabetic neuropathy: a randomized controlled trial. BMC Musculoskeletal Disorders.2012; 13: 36.
10. Zimny S, Schatz H, Pfohl M. The role of limited joint mobility in diabetic patients with an at-risk foot. Diabetes Care. 2004; 27: 942-946.
11. Fernando DJ, Masson EA, Veves A, Boulton AJ. Relationship of limited joint mobility to abnormal foot pressures and diabetic foot ulceration. Diabetes Care. 1991; 14(1): 8-11.
12. Andersen H. Motor dysfunction in diabetes. Diabetes Metab Res Rev. 2012; 028(1): 89-92.
13. Andersen H, Gjerstad MD, Jakobsen J. Atrophy of foot muscles: a measure of diabetic neuropathy. Diabetes Care. 2004; 27(10): 2382-2385.
14. Fernando M, Crowther R, Lazzarini P, Sangla K, Cunningham M, Buttner P, Golledge J. Biomechanical characteristics of peripheral diabetic neuropathy: A systematic review and meta-analysis of findings from the gait cycle, muscle activity and dynamic barefoot plantar pressure. Clin Biomech. 2013; 28(8): 831-845.
15. World Health Organization. Diabetes, Fact Sheet no. 312 [updated 2016 Mar, cited 2016 April 8]. Available from: http://www.who.int/mediacentre/factsheets/fs312/en/
16. Game FL, Hinchliffe RJ, Apelqvist J, Armstrong DG, Bakker K, Hartemann A, et al. Specific guidelines on wound and wound-bed management 2011. Diabetes Metab Res Rev. 2012; 28(Suppl 1): 232-233.
17. Brownlee M. The pathobiology of diabetic complications: a unifying mechanism. Diabetes.2005; 54(6): 1615-1625.
18. Boyko EJ, Ahroni JH, Stensel V, Forsberg RC, Davignon DR, Smith DG. A prospective study of risk factors for diabetic foot ulcer. The Seattle Diabetic Foot Study. Diabetes Care.1999; 22(7): 1036-1042.
19. Tapp RJ, Shaw JE, de Courten MP, et al.Foot complications in type 2 diabetes: an Australian population-based study. Diabet Med. 2003; 20: 105-113.
20. Andreassen CS, Jakobsen J, Andersen H. Muscle weakness: a progressive late complication in diabetic distal symmetric polyneuropathy. Diabetes. 2006; 55(3): 806-812.
21. Tapp RJ, Zimmet PZ, Harper CA, et al.,Australian Diabetes Obesity and Lifestyle Study Group Diabetes care in an Australian population: frequency of screening examinations for eye and foot complications of diabetes. Diabetes Care. 2004; 27: 688-693.
22. Feldman EL, Stevens MJ, Thomas PK, Brown MB, Canal N, Greene DA. A practical two-step quantitative clinical and electrophysiological assessment for the diagnosis and staging of diabetic neuropathy. Diabetes Care. 1994; 17(11): 1281-1289.
23. van Houtum WH, Rauwerda JA, Ruwaard D, Schaper NC, Bakker K. Reduction in diabetes-related lower-extremity amputations in The Netherlands: 1991-2000. Diabetes Care. 2004; 27(5): 1042-1046.
24. Young MJ, Boulton AJM, Macleod AF, Williams DRR, 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.
25. van Schie CHM, Vermigli C, Carrington AL, Boulton A. Muscle weakness and foot deformities in diabetes: relationship to neuropathy and foot ulceration in caucasian diabetic men. Diabetes Care. 2004; 27: 1668-1673.
26. Bokan V. Muscle weakness and other late complications of diabetic polyneuropathy. Acta Clin Croat. 2011; 50(3): 351-355.
27. Lacote M. Chevalier AM, Miranda A, Bleton JP, Stevenin P. Clinical evaluation of muscle function. Edinburgh, London, Melbourn and New York: Churchill Livingstone, 1987.
28. Jandrić S. Osnovi fizikalne medicine i rehabilitacije. Laktaši: Grafomarkt; 2009.
29. Stevanović M, Stevanović M. Merenje obima pokreta u zglobovima. Beograd: Zavod za rehabilitaciju „Dr Miroslav Zotović"; 1975.
30. Andersen H, Nielsen S, Mogensen CE, Jakobsen J. Muscle strength in type 2 diabetes. Diabetes. 2004; 53(6): 1543-1548.
31. Giacomozzi C, D'Ambrogi E, Cesinaro S, Macellari V, Uccioli L.Muscle performance and ankle joint mobility in long-term patients with diabetes. BMC Musculoskeletal Disor-

ders. 2008; 9: 99.
32. Camargo MR, Barela JA, Nozabieli AJ, Mantovani AM, Martinelli AR, Fregonesi CE. Balance and ankle muscle strength predict spatiotemporal gait parameters in individuals with diabetic peripheral neuropathy. Diabetes Metab Syndr. 2015; 9(2): 79-84.
33. Andreassen CS, Jakobsen J, Ringgaard S, Ejskjaer N, Andersen H. Accelerated atrophy of lower leg and foot muscles - a follow-up study of long-term diabetic polyneuropathy using magnetic resonance imaging (MRI). Diabetologia. 2009 Jun; 52(6): 1182-1191.
34. Balducci S, Sacchetti M, Orlando G, Salvi L, Pugliese L, Salerno G, et al. The study on the assessment of determinants of muscle and bone strength abnormalities in diabetes (SAMBA). Nutr Metab Cardiovasc Dis. 2013; 1-9.
35. Sacco IC, Hamamoto AN, Gomes AA, Onodera AN, Hirata RP, Hennig EM Role of ankle mobility in foot rollover during gait in individuals with diabetic neuropathy. Clin Biomech. 2009; 24(8): 687-692.
36. Lázaro-Martínez JL, Aragón-Sánchez FJ, Beneit-Montesinos JV, González-Jurado MA, Morales EG, Hernández DM. Foot Biomechanics in Patients with Diabetes Mellitus. Journal of the American Podiatric Medical Association. 2011; 101(3): 208-214.
37. Hajrasouliha AR, Tavakoli S, Esteki A, Nafisi S, Noorolahi-Moghaddam H. Abnormal viscoelastic behaviour of passive ankle joint movement in diabetic patients: an early or a late complication?. Diabetologia. 2005; 48(6): 1225-1228.
38. Wrobel JS, Najafi B. Diabetic Foot Biomechanics and Gait Dysfunction. Journal of Diabetes Science and Technology. 2010; 4(4): 833-845.
39. Sartor CD, Hasue RH, Cacciari LP, Butugan MK, Watari R, Pássaro AC, et al. Effects of strengthening, stretching and functional training on foot function in patients with diabetic neuropathy: results of a randomized controlled trial. Musculoskeletal Disorders. 2014, 15: 137.
40. Allet L, Armand S, de Bie RA, Golay A, Monnin D, Aminian K, Staal JB, de Bruin ED. The gait and balance of patients with diabetes can be improved: a randomised controlled trial. Diabetologia. 2010; 53(3): 458-466.