MUSCLE WEAKNESS AND OTHER LATE COMPLICATIONS OF DIABETIC POLYNEUROPATHY

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SUMMARY – Diabetic polyneuropathy is a progressive and irreversible disease, which leads to disability, changes in functioning in daily activities, and frequent falls and injuries in diabetes patients. It is one of the major reasons for the occurrence of foot ulceration and amputation of lower extremities. The aim of the study was to assess the relationship between muscle weakness and other complications of diabetic polyneuropathy. The study included 71 patients with electrophysiologically confirmed diabetic polyneuropathy. Through programmed questionnaires, our methodology encompassed examination of demographic, history (duration of diabetes), clinical (neuropathic score and examination) and functional characteristics (muscle strength, foot deformity, joint mobility). Muscle weakness was assessed using a semi-quantitative score. For the purpose of analysis, patients were divided into two groups: MS 1 (muscle strength) – patients with muscle strength score 0 (normal muscle strength) and 1 (moderate muscle strength), and MS 2 – patients with score 2 (severe weakness) and 3 (complete loss of strength). MS 1 group consisted of 44 patients and MS 2 group of 27 patients. Significant differences were found in the duration of diabetes between groups MS 1 and MS 2. The Neuropathy Disability Score was higher in group MS 2 ($p=0.001$). Heel stand testing differed statistically significantly between MS 1 and MS 2 groups ($p<0.001$). High arch was observed in 80% of MS 2 patients. Duration of diabetes of more than 10 years and muscle weakness were found to be the factors influencing the degree of severe diabetic neuropathy.

Key words: Diabetes; Muscle weakness; Ulcer; Diabetic neuropathies; Diabetic foot

Introduction

Diabetic polyneuropathy (DPN) is the most common manifestation of diabetic neuropathies and the most common complication of both type 1 and type 2 diabetes$^{1,2}$. Clinical picture consists of variable degrees of sensory symptoms and signs described above, combined with weakness and foot muscle wasting. Mechanical and traumatic consequences of sensory and/or motor denervation pose a significant risk to neuropathic patients, but are almost entirely preventable by DPN treatment and standard diabetes patient education$^3$. Interaction of the sequels of diabetic polyneuropathy and circulatory impairments in the foot leads to numerous structural and functional alterations that give rise to preconditions for the occurrence of ulcer. Foot ulcers are the main cause of morbidity, mortality and disability of diabetic patients. Estimates show that foot ulceration may occur in up to 15% of diabetic patients during their lifetime$^{4,5}$.

What the literature reveals about DPN complications

Motor neuropathy is common in diabetes. Less dramatic in presentation than sensory neuropathy, the presence of motor deficit secondary to diabetic neu-
Muscle weakness and other late complications of diabetic polyneuropathy usually is not evaluated during examination and subsequently goes unrecognized. A recent electrophysiological study has demonstrated that sensorimotor polyneuropathy was present in 59.8% of 167 diabetic patients. Patients with long-term diabetes have been found to be unable to walk on heels. The relation between inability to walk on heels and muscle strength (MS) has not been established, so there is a clear need for quantitative studies of motor function in type 2 diabetes.

Limited joint mobility is often overlooked because it causes moderate disability. The exact pathogenesis of limited joint mobility is unclear. It is thought to be a manifestation of diffuse collagen abnormality found in patients with DPN. The movements of the ankle joint are of vital importance when it comes to diabetes because any reduced mobility in this joint may cause increased plantar pressure during walking.

Different types of foot deformities cause subsequent stresses on the plantar surface of the foot, resulting in callus formation. One theory states that an imbalance between the extrinsic and intrinsic foot muscle causes clawed toes. This imbalance results in hyperextension of the metatarsal phalangeal joint and distal migration of the metatarsal fat pad.

The aim of this study was to evaluate the relation between muscle weakness and other complications of diabetic polyneuropathy. We hypothesized that muscle weakness and duration of diabetes led to an increase in the number of DPN complications as risk factors for the development of foot ulceration.

Patients and Methods

Seventy-one diabetes patients with distal symmetric polyneuropathy (11 type 1 and 60 type 2 diabetes), aged <70 years, participated in the cross-sectional study in 2009. The study was approved by the local ethics committee. Exclusion criteria for the study were active foot ulcer, acute or chronic musculoskeletal disease, or any other secondary polyneuropathy.

Clinical evaluation and quantitative sensory testing

All patients were evaluated with the Michigan Neuropathy Screening Instrument (MNSI) and Neuropathy Disability Score (NDS). The MNSI questionnaire provides a graded patient response of neuropathic symptoms. In general, a higher score represents more neuropathic symptoms. The NDS quantitative measure of both small- and large fiber dysfunction has been derived from the examination of pain sensation, vibration sensation using a 128 Hz tuning fork, temperature sensation in the foot using warm and cool rods, and the ankle jerk reflex using tendon hammer. The maximum score is 12.

Muscle strength was assessed as the ability of a muscle to produce active movement against the examiner’s resistance. Muscle strength was scored using a semi-quantitative grading system that was based on the scoring system as used in Michigan Diabetic Neuropathy Score. Muscle weakness was scored as follows: 0 – normal muscle strength, 1 – moderate, 2 – severe weakness, and 3 – complete loss of strength.

For the purpose of analysis, patients were divided into two groups: MS 1 group with MS score 0 and 1, and MS 2 group with score 2 and 3.

The ability to stand and walk on heel is used to determine the presence of severe symptomatic diabetic polyneuropathy.

Determination of joint mobility

Joint mobility was measured at the ankle joint and first metatarsophalangeal joint (MTPH 1). Joint mobility examination is simple, inexpensive and fast, with the patient supine and the ankle joint in neutral position. Vertical line was marked on the patient’s skin from the heel to the mid-calf and the maximum range of talus flexion and extension in passive motion was measured with goniometer. Regarding MTPH 1, horizontal line was drawn from the first toe to the heel.

The presence of foot deformity (hammer/claw toes, bunion anomaly, varus deformity, valgus deformity, and high arch) was assessed by clinical examination.

Statistical analysis included descriptive statistics with measurements of central tendency. Between-group differences were calculated using Wilcoxon test, Mann-Whitney U test and chi-square test.

Results

The study included 71 patients. The first aim of the study was to evaluate muscle weakness as a result of DPN complications. The examination allocated 44 patients in MS 1 group and 27 patients in MS 2 group.
Muscle weakness and other late complications of diabetic polyneuropathy

Results obtained by comparison of multiple variables between the groups are shown in Table 1. There was no difference in clinical characteristics and MNSI patient questionnaire score between the two groups. Significant differences were found in the duration of diabetes and NDS score. Diabetes duration was significantly more associated with MS 2 group patients (14.44±4.37; \(p = 0.001\)).

Significant differences were found in NDS score: 6.56±1.10 for MS 1 and 7.51±0.89 for MS 2 (\(p < 0.001\)).

Descriptive statistics presenting the value of ankle joint mobility and MTPH 1 is shown in Table 2. We found a lower degree of ankle joint mobility in MS 2 group. In addition, different types of foot deformities were present in both groups (Table 3). Only 15.3% of MS 2 patients were free from any deformity.

A highly statistically significant between-group difference was recorded in the ability to stand and walk on the heel (\(p < 0.001\)) (Table 4).

**Table 1.** Comparison of demographic and clinical characteristics between MS 1 and MS 2

<table>
<thead>
<tr>
<th></th>
<th>MS 1 N=44</th>
<th>MS 2 N=27</th>
<th>(Z^1)</th>
<th>(P)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>58.77±9.57</td>
<td>64.62±5.65</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
| Diabetes duration    | 10.06±6.40 | 14.44±4.37 | 3.414   | 0.001
| HbA1c                | 7.31±1.32  | 7.32±1.25  | 0.042   | 0.967 |
| Cholesterol          | 5.59±0.90  | 5.70±0.85  | 0.059   | 0.953 |
| Triglycerides        | 2.17±0.97  | 2.20±1.01  | 0.924   | 0.355 |
| MNSI                 | 6.43±1.56  | 6.77±1.45  | 3.551   | 0.000
| NDS                  | 6.56±1.10  | 7.51±0.89  | 3.551   | 0.000

MS 1 = muscle strength graded as normal and moderate; MS 2 = muscle strength graded as severe weakness and complete loss of strength; MNSI = Michigan Neuropathy Screening Instrument; NDS = Neuropathy Disability Score; *mean±SD; \(Z\) Wilcoxon W Mann-Whitney U test; \(p < 0.01\)

**Table 2.** Comparison of joint mobility between MS 1 and MS 2 groups

<table>
<thead>
<tr>
<th></th>
<th>MS 1</th>
<th>MS 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>JM MTPH 1</td>
<td>18</td>
<td>12</td>
</tr>
<tr>
<td>JM ankle</td>
<td>28</td>
<td>11</td>
</tr>
<tr>
<td>Reduced</td>
<td>20</td>
<td>12</td>
</tr>
<tr>
<td>45.5%</td>
<td>48.2%</td>
<td></td>
</tr>
<tr>
<td>36.4%</td>
<td>11</td>
<td></td>
</tr>
<tr>
<td>Limited</td>
<td>6</td>
<td>2</td>
</tr>
<tr>
<td>13.6%</td>
<td>7.4%</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>44</td>
<td>27</td>
</tr>
</tbody>
</table>
| 100.0%               | 100.0%

\(x^2 = 0.652, df=2, P=0.722\)

JM MTPH 1 = mobility of the first metatarsophalangeal joint; JM ANKLE = ankle joint mobility; data are n and %; \(x\)-square test for JM MTPH 1

**Table 3.** Different types of deformities

<table>
<thead>
<tr>
<th></th>
<th>MS 1 (N=44)</th>
<th>MS 2 (N=27)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hammer toe</td>
<td>9</td>
<td>8</td>
</tr>
<tr>
<td>Bunion anomaly</td>
<td>10</td>
<td>4</td>
</tr>
<tr>
<td>Varus deformity</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Valgus deformity</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>High arch</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>No deformity</td>
<td>16</td>
<td>3</td>
</tr>
<tr>
<td>Total</td>
<td>44</td>
<td>27</td>
</tr>
</tbody>
</table>
| 100.0%               | 100.0%

**Discussion**

Study results revealed the group of patients with muscle weakness at the ankle and foot to have suffered from diabetes for more than 10 years, and to have a higher neuropathy examination score and an increased frequency of different types of foot defor-
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The group could be defined as a group with the presence of secondary complications of diabetic polyneuropathy. Some studies report on the percentage of weakness in foot flexor and extensor to be 21% and of weakness of knee flexor and extensor 16% in patients suffering from diabetes for up to 30 years. In this study, duration of diabetes was 15 years. Comparison between the groups with shorter diabetes duration (10 years) and longer diabetes duration (15 years) showed muscle weakness to be present in the latter.

In our study, NDS examination found the group of patients with muscle weakness to have a more severe degree of neuropathy. In numerous studies, the walking on heels test proved to be indicative of more severe forms of neuropathy with the presence of predominantly motor symptoms. Limited joint movement, foot deformities, and proprioception deficiency cannot be neglected. Half of our MS 2 patients (group with weakness) were not able to stand or/and walk on heels. Comparing the two groups, we found that the group of patients with normal and moderate muscle strength had a high percentage of patients with no deformities, while the group of patients with weakness had only 15.2% of patients without deformities. High arch was noticed in 80% of patients in the group with muscle weakness. In the study conducted by Holewski et al., the prevalence of hammer toe correlated with the development of foot ulceration. Hammer toe is a deformity that has been examined in numerous studies, as one of the causes of the increased plantar pressure on the foot. Van Schie et al. found clear connection between motor neuropathy, muscle weakness and foot deformity in patients with a history of ulceration. In our study, high arch was perceived as a deformity present in more severe forms of neuropathy.

Limited ankle joint mobility and MTPH were present in both groups of patients, the percentage being a little higher in the group with weakness, however, the difference did not reach statistical significance.

<table>
<thead>
<tr>
<th>Table 4. Comparison of stand and walk on heel</th>
</tr>
</thead>
<tbody>
<tr>
<td>MS 1 Stand/walk on heel</td>
</tr>
<tr>
<td>-------------------------</td>
</tr>
<tr>
<td>Normal</td>
</tr>
<tr>
<td>Reduced ability</td>
</tr>
<tr>
<td>Total</td>
</tr>
</tbody>
</table>

*χ²=14.116, df=1, P<0.001

Results are presented as n and %; *chi-square test

Conclusion

Duration of diabetes for more than ten years is a factor influencing the degree of severe neuropathy. Muscle weakness increases the frequency of foot deformities and is indicative of the development of other risk factors for foot ulceration.

References

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Sažetak

MIŠIČNA SLABOST I DRUGE KASNE KOMPLIKACIJE DIJABETIČNE POLINEUROPATIJE

V. Bokan

Dijabetična polineuropatija je progresivna i ireverzibilna bolest koja dovodi do onesposobljenosti, promjena u funkcionalnosti u aktivnostima dnevnog života i čestih padova i povreda kod dijabetičara. Jedan je od glavnih uzroka nastanka ulceracije stopala i amputacije na donjim ekstremitetima. Cilj rada je bio ispitati povezanost mišićne slabosti i drugih komplikacija dijabetične polineuropatije, pretpostavljajući da mišićna slabost i trajanje dijabetesa povećavaju broj komplikacija dijabetične polineuropatije i predstavljaju rizične čimbenike za razvoj ulceracije stopala. Prospektivnom studijom obuhvaćena je skupina od 71 bolesnika s elektrofiziološki potvrđenom dijabetičnom polineuropatijom. Metodologija je putem programiranih upitnika obuhvatila ispitivanje demografskih, anamnestičkih (trajanje dijabetesa), kliničkih (neuropatski zbir i ispitivanja) i funkcionalnih obilježja (mišićna snaga stopala, deformiteti stopala, pokretljivost zglobova). Mišićna snaga se ispitivala semikvantitativnim zbirom. Za potrebe ispitivanja bolesnici su podijeljeni u dvije skupine: skupina MS 1 (mišićna snaga) s nalazom MS 0 (normalna mišićna snaga) i 1 (srednja mišićna snaga) i skupina MS 2 s nalazom MS 2 (teža slabost) i 3 (oduzetost). Skupina MS 1 imala je 44 bolesnika, a skupina MS 2 27 bolesnika. Skupine su se značajno razlikovale po trajanju dijabetesa, vrijednostima Neuropathy Disability Score koje su bile značajno više u skupini MS 2 (P<0,001) i testu stajanja na petama (P<0,001). Deformitet stopala tipa kavusa bio je prisutan kod 80% bolesnika u skupini MS 2. Trajanje dijabetesa preko 10 godina i mišićna slabost utvrđeni su kao čimbenici koji utječu na težinu dijabetične polineuropatije.

Ključne riječi: Dijabetes; Mišićna slabost; Ulkus; Dijabetična neurpatija; Dijabetično stopalo