Acta Neurochir
DOI 10.1007/s00701-014-2087-8

CLINICAL ARTICLE - FUNCTIONAL

Impact of diabetes mellitus duration on effect of lower extremity nerve decompression in 1,526 diabetic peripheral neuropathy patients

Wenxiang Zhong · Wenchuan Zhang · Min Yang · Guowei Li · Qiufeng Ma · Xiaosheng Yang

Received: 12 January 2014 / Accepted: 26 March 2014

Abstract
Background Diabetic peripheral neuropathy (DPN) is one of the most common complications of diabetes mellitus (DM). The aim of this study was to analyze DM duration in the prognosis of diabetic peripheral neuropathy of the lower extremity.
Methods A total of 1,526 DPN patients who were treated with DELLON surgical nerve decompression were divided into a short DM duration group and long DM duration group using a length of 5 years as the standard duration. Before and 1.5 years after surgery, high-resolution ultrasound, quantitative sensory testing (QST), and nerve conduction velocity (NCV) were performed and compared to the normal control.
Results Postoperative NCV of all the patients in either treatment group significantly increased ($p<0.05$) and post-operative CSA significantly decreased ($p<0.05$) compared with that before surgery. The CPT significantly increased compared with the preoperative value ($p<0.05$). The post-operative WPT was significantly lower than the preoperative value ($p<0.01$), and the postoperative VPT was significantly lower than the preoperative value ($p<0.05$). Overall, the postoperative NCV, CSA, CPT, WPT, and VPT values from the short DM duration group were significantly different from that of the long DM duration group ($p<0.05$).
Conclusions The duration of DM is critical to the prognosis of DPN of the lower extremity, and the data from this study suggest that an early diagnosis and subsequent surgery for DPN have important clinical value.

Keywords Diabetic peripheral neuropathy · Peripheral nerve decompression · Quantitative sensory testing · Nerve conduction velocity

Introduction

Diabetic peripheral neuropathy (DPN) is a common diabetic complication, with an incidence rate of 50–60 % [13, 15]. DPN is one of the main causes of chronic lower limb and foot ulcers, dry gangrene, moist gangrene, and amputation [17]. Diabetic patients are predisposed to peripheral nerve entrapment, which accelerates nerve damage and clinical progression [16]. In 1992, Dellon [4] reported the first successful use of peripheral nerve decompression surgery to change the natural course of DPN, which effectively prevents ulcers and amputation [1]. These results indicated that surgical intervention should be applied at the onset of entrapment to prevent progression of diabetic neuropathy [16]. In this clinical study, we analyzed the severity and effect of microsurgical peripheral nerve decompression for DPN patients with either a short or long duration of diabetes mellitus (DM), using electrophysiological tests, quantitative sensory testing, and high-resolution ultrasonography.

Methods

Study design

This study was approved by the Xinhua Hospital Ethics Review Board.

Patients

A total of 1,526 DPN patients (874 males and 652 females; mean age 57±10.73 years) treated in our department in the last

W. Zhong · W. Zhang (✉) · M. Yang · G. Li · Q. Ma · X. Yang
Department of Neurosurgery, Xinhua Hospital,
Affiliated to Shanghai Jiaotong University School of Medicine,
1665 Kongjiang Road, Shanghai, China
e-mail: zhangwench88@hotmail.com

Published online: 24 April 2014

5 years were screened. All patients had a type II DM history according to 1999 WHO diagnostic criteria. The average duration of DM was 6.3±2.8 years (715 cases <5 years; 811 cases >5 years), and the average DPN time was 2.5±1.1 years. The fasting plasma glucose range was 5.4–15.2 mmol/l (mean, 10.28±3.46 mmol/l). Symmetrical numbness and/or tingling under both knees that was more serious at night happened in all the patients. The control group included 50 diabetic patients in the same age range (48–76 years) without DPN.

The inclusion criteria were: (1) Neurological symptoms caused by DM: patients were not included if they had a history of cervical or lumbar spondylosis. Patients with other neuropathies resulting from defined risk factors such as alcohol, nutrition, or uremia were also excluded. (2) No obvious limb ischemia: the main artery pulse of the affected limb must be reached, thereby precluding ischemia-induced neurological symptoms. (3) Typical symptoms of diabetes peripheral neuropathy with nerve entrapment: asymmetric limb pain, numbness, diminished feeling, retarded ankle reflex, abnormal temperature, and vibration sensation. All cases scored 19 points in Toronto Clinical Scoring System (TCSS). (4) Positive Tinel signs: only patients with a positive Tinel sign over the tibial nerve in the tarsal tunnel were included; i.e., not every patient with diabetic neuropathy had surgery. (5) Decreased two-point discrimination: the great toe two-point discrimination was greater than 9 mm [2].

Nerve decompression surgery

All nerve decompression surgeries were performed with a microscope under continuous epidural anesthesia. A 3-cm-long incision was made below the fibular head and the skin, and the superficial and deep fascia were cut. After fascia above the common peroneal nerve and the peroneus longus muscle tendon were excised, the common peroneal nerve trunk was exposed and decompressed. Following this, the abductor hallucis brevis was cut and spread to exposure and divided the roof of the medial and of the lateral plantar tunnels. The medial calcaneal tunnel was then identified and decompressed.

A 6-cm-long curved incision was made along the medial malleolus and the skin and superficial and deep fascia were cut. After the flexor retinaculum was excised, the posterior tibial artery and veins and the tibial nerve were identified and decompressed. Epineurium decompression was performed if there was evidence of epineurial thickening.

A 2-cm-long incision was made transversely between the first and second metatarsal heads and the skin, and superficial and deep fascia were cut to expose the tendon of the extensor hallucis brevis muscle. This tendon was then excised to decompress the deep peroneal nerve.

During the perioperative period, blood glucose was controlled at 6.2–8.0 mmol/l. After the patients were discharged, they were required to monitor their fasting plasma glucose every week and control their blood glucose levels to <8.0 mmol/l.

In all cases before surgery, and 18 months after surgery, three sets of examinations were conducted as described below.

Nerve conduction velocity

A Denmark Medtronic EMG (DK 1 2740) was used to detect nerve conduction velocity (NCV). The testing was performed with parameters including skin temperature of 30 °C, a room temperature of 25 °C, and a quiet indoor environment. Stimulations and recordings were taken using surface electrodes. Bilateral motor NCVs of tibial nerve and sensory nerve conduction velocities (SNCV) of the common peroneal nerve and superficial peroneal nerve were recorded. NCV of 40 cases in the control group were also recorded in the same manner. If NCV was less than the control mean minus 3 standard deviations, it was considered to be abnormal [9].

Quantitative sensory testing

Quantitative sensory testing (QST) was completed by the same technician who was blind to the NCV and TCSS results. The testing was performed using an Israel Medoc computer-aided sensory analyzer (TSA-II) in a quiet indoor environment with a room temperature of 25 °C and a skin temperature of 30 °C. Cold perception thresholds (CPT), warm perception thresholds (WPT), and vibratory perception thresholds (VPT) were determined using the "limit-test" method. Temperature sensation was measured on the lateral dorsal foot; with the basic temperature of 32 °C, a temperature range of 0–50 °C, and a temperature change rate of 1 °C/s. Temperature sensation was measured two times at each point, with a time interval of 5 s. VPT was measured on the toe palm, with a vibration frequency of 100 Hz, a basis intensity of 0 μm/s and maximum intensity of 25 μm/s, and a change rate of stimulus intensity of 0.2 mic/s. Vibratory sensation was measured four times at each point, with a time interval of 4 s. The average of repeated values was compared to that of the control group to quantify the degree of neuropathy [8].

High-resolution ultrasonography

All high-resolution ultrasound studies were performed using a Sequoia 512 ultrasound device (Siemens) with an 8 to 14-MHz transducer. A multislice longitudinal and transverse scan was made through the common peroneal nerve and the posterior tibial nerve to observe the continuity and echogenicity of the nerves. The anteroposterior diameter (D1) and transverse diameter (D2) of the nerves were measured at the cross section of 1.5 cm below the fibular head and medial malleolus. The CSA was calculated by the formula of $S=\pi(D1\times D2/4)$.

Table 1 NCV before and after operation of DPN patients ($\bar{x} \pm s$)

Nerves	Control (m s)	Preoperative NCV (m s)		Postoperative NCV (m s)	
		Short duration	Long duration	Short duration	Long duration
Tibial nerve	44.6±3.7	35.7±3.6	20.5±4.3	40.8±3.1	31.2±3.2
Common peroneal nerve	45.8±4.2	39.3±2.8	28.1±3.3	43.1±1.7	36.4±2.1
Superficial peroneal nerve	48.1±3.5	37.2±3.6	26.5±4.2	42.5±3.6	35.8±3.7
Sural nerve	50.3±4.7	38.6±5.6	27.4±6.2	42.6±2.8	34.9±3.1

Statistical analysis

Data were expressed as the mean ± standard deviation. Student's t tests or Chi-square tests were used where appropriate. A value of $p<0.05$ was accepted as statistically significant.

Results

Patient disposition and baseline characteristics

In this study, no patients received amputation during the follow-up.

Nerve conduction velocity

The NCVs of all the DPN patients were significantly slower than that of controls ($p<0.01$). Eighteen months after nerve decompression surgery, NCVs of all the patients increased significantly compared to that before surgery ($p<0.05$), although this was still slower than the control. The NCVs of patients with a long duration of diabetes were still slower than that of patients with short duration ($p<0.05$; Table 1).

Quantitative sensory testing

The QST scores indicated that the lower extremity feeling functions of DPN patients to cold, warm, and vibratory stimuli were abnormal ($p<0.01$). Eighteen months after surgery, QST scores improved significantly ($p<0.05$). Cold and warm sensory function was almost restored to normal levels. The QST scores of patients with a long duration of diabetes were still worse than that of patients with short duration ($p<0.05$; Table 2).

High-resolution ultrasonography

In the control group, the common peroneal nerve and the tibial nerve were shown as multiple hypoechoic parallel but discontinuous linear areas separated by hyperechoic bands on longitudinal sections and multiple round hypoechoic areas on a homogeneous hyperechoic background on transverse sections. In the DPN group, fusiform swelling of the nerves, increase of CSA ($p<0.05$), intraneural hypoechoic, and disappearance of intraneural parallel and linear structure were shown in ultrasonography. Eighteen months after surgery, the CSA of all the patients improved significantly ($p<0.05$), and the CSA of patients with a long duration of diabetes were still larger than that of patients with a short duration ($p<0.05$; Table 3).

Positive rate of nerve conduction velocity and quantitative sensory testing

Before decompression surgery, the positive rate of NCV in DPN patients with a long duration of diabetes was 90.3 % and that of QST was 95.3 % ($p>0.05$). The positive rate of NCV in DPN patients with a short duration of diabetes was 71.5 % and that of QST was 93.7 % ($p<0.05$).

Discussion

DPN is one of the most common diabetic complications, with an incidence rate of 60 % [6], which is one of the main causes of chronic lower limb and foot ulcers, dry gangrene, moist gangrene, and amputation [17]. In 1992, Dellon [4] reported the first use of peripheral nerve decompression surgery in the

Table 2 QST before and after operation of DPN patients ($\bar{x} \pm s$)

QST	Control	Preoperative QST		Postoperative QST	
		Short duration	Long duration	Short duration	Long duration
CPT (°C)	28.6±2.4	22.3±0.5	18.7±0.8	27.5±1.2	23.2±1.3
WPT (°C)	40.5±1.6	45.4±1.8	50.7±2.1	40.6±1.8	44.8±2.1
VPT (μm)	2.8±1.3	5.1±2.4	10.2±3.7	3.2±1.5	6.8±2.2

Table 3 CSA before and after operation of DPN patients ($\bar{x} \pm s$)

	Control	Preoperative CSA		Postoperative CSA	
		Short duration	Long duration	Short duration	Long duration
Tibial nerve CSA (mm^2)	15.6±3.4	20.5±3.8	27.4±4.1	15.8±2.9	19.7±3.2
Common peroneal nerve CSA (mm^2)	13.5±2.6	17.3±3.6	25.8±4.0	13.6±2.7	17.4±2.9

treatment of DPN patients, which was ultimately successful in changing the natural course of DPN, effectively preventing ulcers and amputation [1, 5]. Recently, Dellon's concept applied in clinical practice gained general acceptance by both European and American researchers alike. Peripheral nerve decompression can provide pain relief, numbness and weakness, reduce the incidence of ulcers and amputation, and promote sensory function reconstruction, turning diabetic peripheral neuropathy treatment from passive to active [1, 12, 7].

Past research has focused on the diagnosis and treatment of DPN, including methods such as diagnostics and evaluation of clinical effect. Nevertheless, little clinical research has been formed investigating the duration of DM. In the present study, 1,526 diabetic peripheral neuropathy patients were divided into two groups according to their duration of DM (5-year cut-off point). Not only were the clinic symptoms and signs indicated by the TCSS, but also NCV, QST, and high-resolution ultrasonography were applied to assess the severity of DPN.

Eighteen months after decompression surgery by the same neurosurgeon, CSA, NCV, and QST of the patients improved and significant differences were found. However, CSA, NCV, and QST of patients with a long duration of diabetes were still worse than those of patients with a short duration. These results showed that DPN patients with a long duration of diabetes had less improvement even after decompression. The natural history of DPN is progressive and irreversible [1], and when DPN occurs in patients with a long duration of diabetes, the severity of nerve injury is more serious, which is more difficult to reconstruct sensory and motor function. These results point to the importance of an early diagnosis and surgery of DPN.

In our study, the positive rate of QST was significantly higher than that of NCV in patients with a short duration of diabetes, indicating that QST can be applied to the early diagnosis of DPN. Nerve conduction velocity is an objective, quantitative, non-invasive, and reliable test, and regarded as the "gold standard" for evaluating peripheral neuropathy. However, NCV reflects only the conduction of thick myelinated nerve fibers, and is not sensitive to small nerve fiber lesion [3]. On the contrary, the QST is sensitive to small nerve fibers (A-ð type, C type nerve fibers) [10]. Cold sensation is conducted through small myelinated nerve fibers (A-ð class), and warm sensation is conducted through non-myelin-C-type nerve fiber conduction [14, 11, 9]. The combination of NCV and QST is necessary to screen for DPN in the high-risk population, which is valuable to early diagnosis and surgery.

Conclusions

Our findings indicate that the duration of DM is critical to the severity and prognosis of DPN of the lower extremity. The combination of NCV and QST is valuable for effective early diagnosis and surgery of DPN, which has important clinical value for the progression of DM.

Acknowledgments The authors report no declarations of interest. The authors alone are responsible for the content and writing of the paper. This study was supported by a grant from the National Science Foundation of China (NSFC, No. 30800316). The authors are grateful to the Science and Technology Commission for the funding of this study.

Clinical trial registration number was No. 30800316.

Conflicts of interest All the authors have no conflicts.

References

1. Aszmann O, Tassler PL, Dellon AL (2004) Changing the natural history of diabetic neuropathy: incidence of ulcer/amputation in the contralateral limb of patients with a unilateral nerve decompression procedure. Ann Plast Surg 53(6):517–522
2. Bril V, Perkill B (2002) Validation of the Toronto Clinical Scoring System for diabetic polyneumpathy. Diabetes Care 25:2048–2052
3. Chauplannaz G, Vial C (2004) Electrodiagnositic assessment of neuromuscular junction disorders. Rev Med Liege 59:184–189
4. Dellon AL (1992) Treatment of symptomatic diabetic neuropathy by surgical decompression of multiple peripheral nerves. Plast Reconstr Surg 89(4):689–697
5. Dellon AL (2002) Preventing foot ulceration and amputation by decompressing peripheral nerves in patients with diabetic neuropathy. Ostomy/Wound Manag 48(9):36–45
6. Dellon AL, Aszmann OA, Tassler P (2003) Outcome of surgical decompression of nerves in diabetic on incidence of ulceration and amputation. J Reconstr Microsurg 19:350–355
7. Dellon AL (2007) Neurosurgical prevention of ulceration and amputation by decompression of lower extremity peripheral nerves in diabetic neuropathy: update 2006. Acta Neurochir Suppl 100:149–151
8. Kincaid JC, Price KL, Jimenez MC, Skljarevski V (2007) Correlation of vibratory quantitative sensory testing and never conduction studies in patients with diabetes. Muscle Nerve 36(6):821–827

9. Onde ME, Ozge A, Senol MG, Togrol E, Ozdag F, Saracoglu M, Misirli H (2008) The sensitivity of clinical diagnostic methods in the diagnosis of diabetic neuropathy. J Int Med Res 36(1):63–70
10. Orstavik K, Norheim L, Jorum E (2006) Pain and small-fiber neuropathy in patients with hypothyroidism. Neurology 67:786–791
11. Peter STC, Didier PC (2004) Technology lecture review: quantitative sensory testing. Muscle Nerve 29:734–747
12. Rader AJ (2005) Surgical decompression in lower-extremity diabetic peripheral neuropathy. J Am Podiatr Med Assoc 95(5):446–450
13. Reiber GE (1996) The epidemiology of diabetic foot problems. Diabet Med 13(Suppl 1):S6–S11
14. Rutkove SB, Veves A, Mitsa T, Nie R, Fogerson PM, Garmirian LP, Nardin RA (2009) Impaired distal thermoregulation in diabetes and diabetic polyneuropathy. Diabetes Care 32(4):671–676
15. Sangiorgio L, Iemmolo R, Le Moli R, Grasso G, Lunetta M (1997) Diabetic neuropathy: prevalence, concordance between clinical and electrophysiological testing and impact of risk factors. Panminerva Med 39(1):1–5
16. Siemionow M, Zielinski M, Sari A (2006) Comparison of clinical evaluation and neurosensory testing in the early diagnosis of superimposed entrapment neuropathy in diabetic patients. Ann Plast Surg 57(1):41–49
17. Sirmreieh M, Taylor BV, Dyck PJ (2005) Diabetic neuropathies classification, clinical features and pathophysiological basis. Neurologist 11:63–79