Evaluation of the Clinical Efficacy of Multiple Lower Extremity Nerve Decompression in Diabetic Peripheral Neuropathy

Wenchuan Zhang [1] Shiting Li [1] Xuesheng Zheng [1]

[1] Department of Neurosurgery, XinHua Hospital, Affiliated to Shanghai JiaoTong University School of Medicine, Shanghai, China

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Address for correspondence and reprint requests Dr. Xuesheng Zheng, Department of Neurosurgery, XinHua Hospital, Affiliated to Shanghai JiaoTong University School of Medicine, Kongjiang 16, Shanghai, China 200092 (e-mail: xueshengzheng@gmail.com).

Abstract

Background In 1992, Dellon reported the first use of peripheral nerve decompression surgery in the treatment of limb diabetic peripheral neuropathy (DPN). However, the efficacy of this procedure is still under debate. In this clinic study, we analyzed the therapeutic effect of microsurgical peripheral nerve decompression for DPN patients, using both clinical evaluation and electrophysiological testing.

Patients and Methods In 560 patients with DPN , the surgical nerve decompression as described by Dellon was performed. Before and 18 months after surgery, Toronto Clinical Scoring System, quantitative sensory testing, and nerve conduction velocity tests were evaluated in all cases. The control group included 40 diabetic patients in the same age range but without DPN.

Results The scores of nerve conduction velocity, quantitative sensory testing, and Toronto Clinical Scoring System improved significantly after microsurgical decompression of the entrapped nerves, although they were still worse than in the control group. According to the Wagner classification, 208 cases (37.1%) were rated as 1 (surface ulcer, no clinical infection) before surgery, but all were rated as 0 (no surface ulcer) 18 months after surgery.

Conclusion Microsurgical decompression of entrapped peripheral nerves for DPN helped improve nerve conduction, restore lower limb feeling and motor function, and cure ulcers.

Keywords

- diabetic peripheral neuropathy
- nerve decompression
- Toronto Clinical Scoring System
- quantitative sensory testing
- nerve conduction velocity

Introduction

Diabetic peripheral neuropathy (DPN) is a common diabetic complication, with the incidence rate as high as 50 to 60%.[1,2] DPN is one of the main causes of chronic lower limb and foot ulcers, dry gangrene, moist gangrene, and amputation.[3] Distal symmetric polyneuropathy (DSPN) is the most common type of diabetic neuropathy and does not require surgical intervention.[4] Diabetic patients are predisposed to peripheral nerve entrapment, which is superimposed to preexisting DSPN and accelerates nerve damage and clinical progression, expediting development of diabetic foot syndrome.[4] Therefore, surgical intervention should be applied at onset of the entrapment to prevent progression of diabetic neuropathy.[4] In 1992, Dellon[5] was the first who reported the use of peripheral nerve decompression surgery in the treatment of limb DPN, which turned out to be successful. Nerve decompression changed the natural course of DPN and effectively prevents ulcers and amputation.[6]

In this clinic study, we analyzed the therapeutic effect of microsurgical peripheral nerve decompression for DPN patients, using both clinical evaluation and electrophysiological testing.

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Patients and Methods

This study was approved by the Xinhua Hospital Ethics Review Board. A total of 560 DPN patients (260 males and 300 females; mean age, 58 ± 11.32 years) treated in our department between January 2005 and June 2008 were screened. All patients had a type 2 diabetes mellitus (DM) history according to the 1999 World Health Organization diagnostic criteria. The average DM duration was 7.6 ± 2.5 years, and the average DPN duration was 2.5 ± 1.1 years. The fasting plasma glucose at the first visit to our department was 5.2 to 17.1 mmol/L, with an average of 10.48 ± 3.37 mmol/L. According to the Wagner classification,[22] 208 cases (37.1%) were rated as 1 (surface ulcer, no clinical infection) before surgery. The control group included 40 diabetic patients in the same age range (48 to 76 years) but without DPN.

Inclusion and Exclusion Criteria

Inclusion and exclusion criteria included the following. The neurological symptoms must be caused by DM. No history of cervical or lumbar spondylosis. No other neuropathy resulting from defined risk factors such as alcohol, nutrition, or uremia. No obvious limb ischemia. The main artery pulse of the affected limb must be palpable, thereby precluding ischemia-induced neurological symptoms. Typical symptoms of DPN with nerve entrapment, including asymmetric limb pain, numbness, diminished feeling, retarded ankle reflex, abnormal temperature, and vibration sensation. All cases scored 19 points on the Toronto Clinical Scoring System (TCSS).[23] Positive Tinel sign over the tibial nerve in the tarsal tunnel (i.e., not every patient with diabetic neuropathy had surgery). Decreased two-point discrimination: the great toe two-point discrimination was greater than 9 mm.[7]

Nerve Decompression Surgery

Targets included posterior tibial nerve and its branches near the ankle, common peroneal nerve, the dorsal foot branch of deep peroneal nerve, the internal and external branches of the plantar nerve, and the lateral calf cutaneous nerve. The connective tissue and fascia at the anatomic narrow sites compressing the nerve were incised. Decompression of the four medial ankle tunnels was done as described by Dellon,[5] and included neurolysis of the medial and lateral plantar nerves and calcaneal nerve in their own separate tunnels and removal of the septum between the medial and lateral plantar tunnels. Epineurium decompression was done if there was epineurial thickening. Microsurgical intraneural neurolysis was performed if there was intraneural fibrosis.

The tibial nerve in the tarsal tunnel is approached through an incision that is posterior to the medial malleolus and midway to the Achilles tendon.[8] The tibial nerve and its branches are often compressed at the medial ankle. There are four tunnels to decompress: the tibial nerve in the tarsal tunnel, the medial plantar nerve in the medial plantar tunnel, the lateral plantar nerve in the lateral plantar tunnel, and the calcaneal nerve in one or more calcaneal tunnels. The tibial nerve must be separated from the artery and vein and inspected. The epineurium is opened, and a clear division of the medial and lateral plantar nerves within the tarsal must be observed.

The common peroneal nerve is decompressed at the fibular neck.[8] An incision of approximately 3 cm is made across the fibular neck 3 cm below the capitulum fibulae. The deep fascia is lifted and then incised to expose the common peroneal nerve, which is usually entrapped by the fascial band of the peroneus longus muscle. The fascial band is lifted carefully and divided.

The incision for neurolysis of the superficial peroneal nerve is typically parallel to the fibula.[8] The incision is made into the subcutaneous plane. A small elevation from the fascia accompanied by a small blood vessel and some fat often marks the spot of the entrapment as the superficial peroneal nerve travels from deep to the fascia to enter the subcutaneous plane. The incised fascial edges are cauterized.

The deep peroneal nerve becomes entrapped beneath the extensor hallucis brevis tendon and the underlying bones, at the juncture of the first and second metatarsals and the cuneiform.[8] The incision is made obliquely. The extensor hallucis brevis tendon is identified and resected.

In the perioperative period, the blood glucose was controlled at 6.2 to 8.0 mmol/L. After the patients were discharged, they were required to monitor the fasting plasma glucose every week and control the blood glucose to less than 8.0 mmol/L.

Examinations

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

TCSS[7] grades the degree of neuropathy on the basis of symptom, reflex, and sensory scores. Symptom scores include foot pain, numbness, tingling, weakness, ataxia, and upper limb symptoms. Each symptom scored 1 point, with a total of 6 points. Deep tendon reflex scores include bilateral knee reflex and ankle reflex. No reflex is recorded as 2 points, weakened reflex as 1 point, and normal reflex as 0 points (total 8 points). Sensory scores include pinprick, temperature sensation, light touch, vibration, and joint position sense. No sensation is recorded as 1 point; otherwise, 0 points are assigned (total 5 points). Therefore, the maximum TCSS score is 19. A score of 0 to 6 means no neuropathy.

Denmark Medtronic EMG (DK 1 2740; Medtronic, Minneapolis, MN, USA) was used to detect nerve conduction velocity (NCV). Parameters are skin temperature of 30°C, room temperature of 25°C, and quiet indoor environment. Stimulation and recording were performed using surface electrodes. Bilateral motor NCVs of the tibial nerve and sensory NCVs of the common peroneal nerve and the superficial peroneal nerve were recorded. NCVs of 40 patients in the control group were also recorded in the same way. If NCV was less than the control mean minus three standard deviations, it was considered as abnormal.[9]

Quantitative sensory testing (QST) was completed by a technician who was blind to the NCV and TCSS results. Parameters included the following: performed in a quiet indoor environment, room temperature of 25°C, skin temperature of 30°C, and using the Medoc (Ramat Yishai, Israel)

computer-aided sensory analyzer (TSA-II). Cold perception threshold (CPT), warm perception threshold (WPT), and vibratory perception threshold (VPT) were determined using the "limit-test" method. Temperature sensation was measured on the lateral dorsal foot, with a basic temperature of 32°C, a temperature range of 0°C to 50°C, and a temperature change rate of 1°C/s. Temperature sensation was measured four times at each point, with a time interval of 5 seconds. VPT was measured on toe palm, with a vibration frequency of 100 Hz, a basis intensity of 0 μm and maximum intensity of 25 μm, and a change rate of stimulus intensity of 0.2 μm/s. Vibratory sensation was measured four times at each point, with time interval of 4 seconds. The average of repeated values was compared with that of the control group to quantify the degree of neuropathy.[10]

Statistical Analysis

Data were expressed as the mean ± standard deviation. Student t tests or χ^2 tests were used where appropriate. $p < 0.05$ was accepted as statistically significant.

Results

Ulceration and Amputation Rates

In this study, 208 DPN cases (37%) had surface ulcers without infection before surgery (Wagner classification 1). During the 18-month follow-up, all the ulcers were cured after surgery (Wagner classification 0), and no new ulcers emerged. The rest of the patients (n = 352) did not have lower limb ulcers in this period. Thus, there was no amputation indication for any patient in this study during the follow-up.

TCSS

All 560 patients in this study were typical DPN patients, with a TCSS score of 19 points. After surgery, symptoms and signs such as lower extremity pain, numbness, weakness, walking imbalance, deep tendon reflex, and toe feeling improved significantly. Eighteen months after surgery, the TCSS score of 493 patients (88%) was 15 points or lower (►**Table 1**). The average postoperative TCSS score was 12.5 ± 3.7 ($p < 0.05$).

Table 1 Toronto Clinical Scoring System (TCSS) Scores of 560 Diabetic Peripheral Neuropathy (DPN) Patients before and 18 Months after Nerve Decompression Surgery

TCSS Scores	Preoperative TCSS of DPN Patients Number (%)	Postoperative TCSS of DPN Patients Number (%)
7–9	0 (0%)	73 (13%)
10–12	0 (0%)	224 (40%)
13–15	0 (0%)	196 (35%)
16–19	560 (100%)	67 (12%)

Note: The maximum TCSS score is 19; and a score of 0–6 means no neuropathy. TCSS scores of 493 DPN patients (88%) improved to 15 points or even better 18 months after nerve decompression surgery. The average postoperative TCSS score was 12.5 ± 3.7 ($p < 0.05$).

NCV

The NCVs of the DPN patients were much slower than the controls. Eighteen months after the nerve decompression surgery, tibial NCV accelerated from 28.2 ± 7.20 m/s to 36.31 ± 3.33 m/s ($p < 0.05$), common peroneal NCV accelerated from 33.3 ± 5.31 m/s to 42.21 ± 1.41 m/s ($p < 0.05$), and superficial peroneal NCV accelerated from 35.2 ± 8.11 m/s to 41.56 ± 4.54 m/s ($p < 0.05$), although the NCVs were still slower than those in controls (►**Table 2**).

QST

According to the QST scores, the lower extremity feeling functions of DPN patients to cold, warm, and vibratory sensations were abnormal. Eighteen months after surgery, QST scores improved significantly. CPT improved from 21.11 ± 0.6°C to 26.33 ± 1.1°C ($p < 0.05$). WPT improved from 46.34 ± 2.3°C to 39.76 ± 2.3°C ($p < 0.01$). VPT improved from 9.18 ± 2.2 μm to 6.23 ± 3.6 μm ($p < 0.05$). Cold and warm sensory functions were almost restored to the normal level (►**Table 3**).

Two-Point Discrimination

Eighteen months after surgery, the average great toe two-point discrimination was 6.72 ± 1.62 mm ($p < 0.05$).

Follow-Up

A longer telephone follow-up was done in May 2010. In total, 47 patients were lost to follow-up. Among the rest of the patients (n = 513), the longest postoperative time was 5 years, and the shortest was approximately 2 years. Only three patients have suffered from lower limb superficial

Table 2 Nerve Conduction Velocity (NCV) Tests of 560 Diabetic Peripheral Neuropathy (DPN) Patients before and 18 Months after Nerve Decompression Surgery Compared with Tests of Controls (mean ± standard deviation)

Nerve	Preoperative NCV of DPN Patients (m/s)	Postoperative NCV of DPN Patients (m/s)	NCV of Controls (m/s)
Tibial nerve	28.2 ± 7.20	36.31 ± 3.33	43.8 ± 3.90
Common peroneal nerve	33.3 ± 5.31	42.21 ± 1.41	48.1 ± 6.42
Superficial peroneal nerve	35.2 ± 8.11	41.56 ± 4.54	46.3 ± 9.03

Note: Compared with the controls, the lower limb NCVs of the DPN patients were much slower. Eighteen months after nerve decompression surgery, the tibial nerve NCV ($p < 0.05$), common peroneal nerve NCV ($p < 0.05$), and superficial peroneal nerve NCV ($p < 0.05$) recovered significantly. However, the postoperative NCVs were still slower than those in controls (tibial nerve postoperative NCV versus control NCV, $p < 0.05$; common peroneal nerve postoperative NCV versus control NCV, $p < 0.05$; and superficial peroneal nerve postoperative NCV versus control NCV, $p < 0.05$).

Table 3 Quantitative Sensory Testing (QST) Results of 560 Diabetic Peripheral Neuropathy (DPN) Patients before and 18 Months after Nerve Decompression Surgery Compared with Results of Controls (mean ± standard deviation)

QST Item	Preoperative QST of the DPN Patients	Postoperative QST of the DPN Patients	QST of the Controls
CPT, °C	21.11 ± 0.6	26.33 ± 1.1	28.8 ± 2.5
WPT, °C	46.34 ± 2.3	39.76 ± 2.3	40.1 ± 1.3
VPT, μm	9.18 ± 2.2	6.23 ± 3.6	3.04 ± 2.8

Note: Compared with controls, the QST results of DPN patients are abnormal ($p < 0.01$). Eighteen months after nerve decompression surgery, lower limb QST results improved. CPT (high number normal) was significantly higher than that before surgery (improved; $p < 0.05$). WPT (low number normal) was significantly lower than that before surgery (improved; $p < 0.01$). VPT (low number normal) was significantly lower than that before surgery (improved; $p < 0.05$). CPT and WPT recovered to an extent similar to the control QST ($p > 0.05$).

ulcers after their 18-month follow-up. No patient needed surgical amputation during this period.

Discussion

The pathogenesis of DPN is closely related to such factors as nerve ischemia due to nerve and vascular compression, increased polyol pathway activity, nerve demyelination, lipid metabolic disorder and vitamin deficiency. In particular, peripheral nerve entrapment is a very important factor.[11] The peripheral nerves of diabetic patients are susceptible to compression because intranerve water content increases, retrograde axonal transport changes, and the nourishing blood vessels of nerve axons are affected by microangiopathy.[12]

In the past, surgeons could only help with infection, necrosis, ulcer repair, or amputation. Dellon established a double-crush model. The first crush is that DM causes deficient axoplasmic flow and increased endoneurial water content, making the nerve more susceptible to entrapment. The second crush is that the edematous nerve is entrapped at anatomically narrow sites.[8] Some authors believed that nerve decompression microsurgery can effectively relieve such symptoms as pain, numbness, and fatigue; rebuild physical feeling; and reduce the incidence of ulcer and amputation, turning DPN treatment from passive to active.[6,13,14] However, a recent meta-analysis failed to identify a single randomized controlled trial or any other well-designed prospective study controlling for nonoperated limb that showed improvements in predefined end points after decompressive surgery.[15] Given the current evidence available, this treatment alternative should be considered unproven.[16]

In this study, 560 DPN patients underwent peripheral nerve decompression microsurgery. Not only were the clinical symptoms and signs relieved, as indicated by TCSS, but also the NCV and QST scores improved. Although NCVs only improved moderately, QST indicates that cold and warm sensory function was almost restored to normal. In addition, the lower limb superficial ulcers were cured after surgery. The positive features of this study are the high patient number, strict inclusion criteria (all patients scored 19 points in TCSS), and multiple quantitative scoring systems for multiple purposes. Thus, our findings support that patients with DPN, especially those with asymmetric symptoms, might get benefits from the decompression procedure, which was described by Dellon. In this study, the control group consisted of patients with DM but without DPN; thus, the therapeutic effect of decompression surgery was not compared with that of nonsurgical therapies. In fact, the purpose of the control group was just to provide a reference for the used indices (TCSS, NCV, and QST). Thus, the control group was much smaller than the surgical group.

Small nerve fibers are involved in the early stage of DPN. With progression of the disease, middle and thick fibers are also involved gradually.[17] NCV is an objective, quantitative, noninvasive, and reliable test and is 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 lesions.[18] However, QST is sensitive to small nerve fibers (A-ð type, C-type nerve fibers[19]). The cold sensation is conducted through small myelinated nerve fibers (A-ð class), and the warm sensation is conducted through nonmyelin C-type nerve fibers.[20,21] Therefore, our findings suggest that nerve decompression surgery improves the function of both thick myelinated nerve fibers and small fibers, but is more effective for small fibers.

The TCSS is a scoring system based on clinical data and is considered as one of the best clinical examinations for peripheral neuropathy.[3] Our study found that TCSS results are consistent with QST and NCV results. Because TCSS is simple and comprehensive, it can be used as an early screening test in outpatient clinic.

Among the 560 patients who received lower limb nerve decompression surgery, two patients suffered from wound dehiscence. After daily dressing change, antibiotics administration, and strict blood glucose control, the wounds healed within 2 months. We thought the cause was ambulation initiated too early. Thus, the patients were not encouraged to ambulate until 72 hours after surgery. Another patient had postoperative extensive lower leg subcutaneous hemorrhage, which was cured by surgical incision and drainage; however, the function of his common peroneal nerve was impaired moderately. We thought the tourniquet prevented us from finding the early signs of subcutaneous hemorrhage. Since then, we abandoned using tourniquets. We think strict hemostasis using bipolar coagulation is key to prevent such complication.

In summary, in this series, peripheral nerve decompression microsurgery for DPN patients helped relieve suffering, improve nerve conduction, and rebuild the lower limb

sensory and motor function. A combination of several evaluation systems helps the surgeon to get an objective and comprehensive impression.

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Conflict of Interest
None

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