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Surgical Decompression in the Management of Lower Extremity Diabetic Peripheral Neuropathy: A Narrative Review

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ABSTRACT

Diabetic peripheral neuropathy commonly develops in patients with diabetes mellitus. Standard medical treatments help a minority of patients with the amelioration of pain, but do not modify the course of the illness. Treatment of diabetic peripheral neuropathy with surgical decompression of lower extremity nerves is controversial. However, the surgery has the potential to ameliorate pain and to improve quality of life in patients suffering with painful neuropathy; it also has the potential to modify the course of the disease, improving protective sensation of the skin of the foot, decreasing the probability of ulcer formation and subsequent amputation. This review will briefly look at the etiology of diabetic peripheral neuropathy and the rationale for nerve decompression surgery as a treatment option. Surgery for upper extremity nerves, and diagnostic criteria will be followed by an examination of the evidence published to date on the validity of nerve decompression surgery in the treatment of diabetic neuropathy.

Introduction

Diabetic peripheral neuropathy (DPN), also called distal symmetric polyneuropathy (DSPN), is part of a spectrum of disorders collectively referred to as diabetic neuropathy (DN). DN includes diverse types of autonomic neuropathy which can affect the cardiovascular, urogenital, and gastrointestinal systems; it also includes mononeuropathies and radiculopathies.[1,2] Treatment of DPN to date in the lower extremity has largely been non-surgical. This review will look at the existing options and alternatives, including the option of lower extremity nerve decompression surgery (NDS). To appreciate the rationale for NDS as a treatment option, a brief review of the etiology of DPN and peripheral nerve enlargement will be followed by an examination of nerve decompression surgery in the upper extremity of DPN patients. Finally, the evidence for NDS as a treatment option for DPN in the lower extremity will be examined.

Risk Factors and the Burden of Diabetic Peripheral Neuropathy

Risk factors for DPN include poor glycemic control, increasing age, dyslipidemia, obesity, and tobacco smoking.[3] Lifetime risk for DPN in type 1 diabetes (T1D) is about 33%, and in type 2 diabetes (T2D) 50%.[4-6] Peripheral neuropathy has been reported to be present in 13% of individuals with impaired glucose tolerance (prediabetes).[7] Increasing age is a risk factor; nevertheless rates of DPN for youth with diabetes have been reported at 8.5% for T1D and 17.7% for T2D.[8]

The economic, societal, and personal burdens of DPN are significant.[9,10] At a societal level, DPN imposes a substantial economic burden via increased cost of healthcare and consumption of both monetary and personnel resources, employment absences, and decreased productivity and lifespan of affected patients.[11,12] The disease also has a profound personal impact upon afflicted patients. This includes issues such as impaired balance and gait disturbances, increasing the risk of falls. DPN also renders patients susceptible to toe/foot ulcerations. These require prolonged treatments, despite which patients sometimes progress to amputation. They also confer a financial strain, both from the cost of accessing care (variable depending upon health jurisdiction) and from increased work absences and limitation of employment potential. The incidence of foot ulceration in the diabetic population is around 2 % per year, yielding in excess of 18.6 million affected people worldwide annually.[9,10] Around 50 to 60 % of these foot ulcers become infected, and 20 % culminate in an amputation.[11] DPN is the most important underlying cause of foot ulceration in patients with DM.[2,11,13] The median survival after below-knee amputation in patients with diabetes mellitus is less than 3 years.[14]

Finally, multiple studies have documented that painful DPN adversely affects patient's quality of life, both in physical and mental quality measures. Painful DPN is associated with anxiety, depression, and sleep disturbances. [15-18]

Etiology of Diabetic Peripheral Neuropathy and the Polyol Pathway

The exact etiology of DPN remains unknown; abnormalities of multiple metabolic pathways have been documented, including hexosamine and protein kinase C, accumulation of advanced glycation end products, oxidative stress through the accumulation of free oxygen and nitrogen reactive species, upregulation of mitogen-activated protein kinases and proinflammatory cytokines, and many more. Most pertinent to this discussion is the polyol pathway of sugar metabolism. Elevated levels of glucose in the peripheral nerve in diabetes mellitus drives the polyol pathway of sugar metabolism. Aldose reductase converts glucose into sorbitol, which accumulates causing osmotic stress to the nerve, leading to intraneural oedema.[19-21]

Enlarged Cross Sectional Area of Peripheral Nerves in Diabetic Peripheral Neuropathy

Multiple ultrasound studies over the last decade and a half have consistently reported enlargement of peripheral nerves in patients with DPN. In 2012 Riazi et al. reported that ultrasound measurements of the posterior tibial nerve 1, 3 and 5 cm above the medial malleolus all showed significantly increased cross-sectional area (CSA) of the nerve in patients with DSPN compared to control subjects. Furthermore, nerve conduction studies in those patients demonstrated an "inverse relationship between CSA and distal tibial compound muscle action potential amplitude and between CSA and DSP severity."[22] This enlargement has been confirmed in multiple subsequent studies.[23-26]

The" Double Crush" Hypothesis

The knowledge of the enlargement of peripheral nerves in the lower extremity in DPN led Dellon in 1988 to suggest that a modification of the "double crush hypothesis" might apply in DPN.[27] The original hypothesis was proposed by Upton and McComas in 1973 to explain the relationship of cervical nerve compression combined with a more distal upper extremity nerve compression (at the cubital or carpal tunnel), resulting in more severe symptoms than either lesion alone.[28] Dellon proposed in DPN that the metabolic insults of DPN are the first "crush"; the compression of the enlarged peripheral nerve in the lower extremity is the second "crush", aggravating the DPN symptoms.[29,30] Reworded, the peripheral nerves of diabetic patients are impaired by the metabolic insult/microangiopathy of the disease, rendering them more susceptible to the effects of compression brought on by the increased cross-sectional area of those nerves.

Non-Surgical Treatments for Diabetic Peripheral Neuropathy, Limitations

It is beyond the scope of this review to look in-depth at the various therapeutic modalities for the prevention of the development of DPN and for its treatment. Prevention is primarily found in intensive glycemic control and is more effective in T1D than T2D.[2]

Treatment of DPN is directed primarily at pain management. The four main classes of medications used are tricyclic antidepressants, serotonin-norepinephrine reuptake inhibitors, gabapentinoids, and sodium channel blockers. [31] Alternative therapies include tapentadol (a mu-opioid receptor agonist/noradrenaline reuptake inhibitor (MOR-NRI)), topical agents (lidocaine, nitrates, capsaicin), exercise, spinal cord stimulation, and cognitive behavioural therapy.[2,32,33] Using one or more of these medications, only one-third of patients achieve more than 50% pain relief, meaning that most patients live with chronic pain despite best efforts.[34] NDS is a potential treatment option for these patients.

To date, there are no USFDA approved disease-modifying therapies for DPN.[33]

Surgical Decompression of the Median Nerve in Diabetic Peripheral Neuropathy

Carpal tunnel syndrome is the commonest peripheral entrapment neuropathy. Carpal tunnel release surgery effectively relieves symptoms of carpal tunnel syndrome in patients with DPN, although some authors have noted less (albeit significant) post-operative symptom improvement in DPN patients. [35,36] The diagnosis of carpal tunnel syndrome in DPN patients is clinical, does not include the necessity of nerve conduction studies.

It is recognized that 10 to 15 percent of patients with clinical symptoms of carpal tunnel syndrome have no electrodiagnostic evidence of median mononeuropathy. [37] Perkins et al. concluded that "nerve conduction study has a clear role in determining the presence and severity of DPN but does not reliably distinguish the presence or the absence of carpal tunnel syndrome in subjects with diabetes. Given the high prevalence of clinical carpal tunnel syndrome in subjects with DPN, it is recommended that therapeutic decisions in patients with clinical criteria for carpal tunnel syndrome should be made independently from nerve conduction study findings". [38]

In 2022 the Michigan Collaborative Hand Initiative for Quality in Surgery reported a multicenter cohort study on the Concordance of Electrodiagnostic Scores and Clinical Severity in Carpal Tunnel Syndrome. They concluded "the importance of avoiding the use of electrodiagnostic studies as a screening tool for carpal tunnel syndrome" [39] In 2024 the American Academy of Orthopaedic Surgeons updated their Evidence-Based Clinical Practice Guideline to state that "strong evidence suggests that CTS-6 (Carpal Tunnel Syndrome 6) can be used to diagnose carpal tunnel syndrome, in lieu of routine use of ultrasonography or NCV/EMG." [40] This was endorsed by the American Society for Surgery of the Hand, and the American Association for Hand Surgery. The CTS-6 includes 2 items of the patient's history and 4 findings on physical examination, including a positive Tinel sign. [41,42] Nerve conduction studies/electromyography and neurologist consultation are only to be sought when alternative diagnoses are under consideration – for example more proximal sites of nerve compression, other lower motor neuron diseases such as mononeuritis multiplex or chronic inflammatory demyelinating polyneuropathy.

Surgical decompression of lower extremity nerves for the treatment of Diabetic Peripheral Neuropathy

There are two fundamental issues that need to be addressed when assessing the question if lower extremity nerve decompression surgery (NDS) is a viable treatment option for DPN, and assessing the published research:

- Lower extremity nerve compression in DPN
- Painful versus painless DPN

Lower extremity nerve compression in Diabetic Peripheral Neuropathy

The first issue to be queried is that of nerve compression. The discussion parallels that of CTS and DPN. DPN can exist with or without peripheral nerve compression in the lower extremity, similar to CTS in the upper extremity. [43-45] In the lower extremity recognized sites of entrapment include the (posterior) tibial nerve at the tarsal tunnel; the common peroneal nerve at the fibular neck; the deep peroneal nerve at the crossing of the extensor hallucis brevis tendon on the dorsum of the foot. [29] The controversial issue is whether or not electrodiagnostic studies (EDS) are required for the diagnosis of compression in the setting of DPN in the lower extremity. The argument that EDS are required is specious at best, as noted in the above discussion on CTS and DPN. [2,46]

Painful Versus Painless Diabetic Peripheral Neuropathy

Criticism of the existing literature is valid in that many studies reported do not clearly differentiate between patients with or without pain. This becomes important in determining the measure of success or failure of DNS. Patients with painless DPN are often not particularly bothered by, or indeed aware of, the decreasing sensation in their feet. Indeed, diabetic foot ulcer prevention strategies concentrate on identifying painless DPN patients - without appropriate screening, education, and prophylaxis, unfortunately for many patients their first knowledge of having the disorder is the development of an ulcer. [47-49]

Hence the evaluation of success or failure of NDS in lower extremity DPN should be examined in 2 separate spheres. First, painful DPN patients should be assessed based on amelioration of pain and improvement in their quality of life. Second, all DPN patients (painful and painless) should be assessed based on improvement in foot sensation after the surgery, and subsequent rates of foot ulcer/amputation.

Current Evidence – Nerve Decompression Surgery for the Amelioration of Pain

There is increasing evidence that DNS is effective for lessening lower extremity pain in patients with painful DPN. Older studies have been dismissed by critics as observational, limited by a lack of randomization and/or blinding. [46] However, three recent RCT's address this issue. In 2024, Rozen et al. reported a double-blind randomized control trial (RCT) in which the patient received sham surgery on their contralateral leg as a blinded control. [50] They reported decreased pain in both

the nerve decompression legs and the sham surgery legs to a comparable level at one year, but significantly decreased pain at 56 months in the nerve decompression leg versus the sham legs. Their study design of performing sham surgery on the contralateral leg as a control at the same time as the DNS was prone to placebo effect.[51] Best et al. in 2019 reported a single-blinded RCT. [52] In that study, controls were randomized study recruits, and in outcome evaluations the observers were blinded as to patient status of intervention or control. They found decreased pain in the decompression group compared to control over 1 year within groups both by the McGill visual analogue scale, and the NeuroQol pain assessment. Patients were 3 times more likely to rate the pain in their decompressed leg as better at study endpoint. In 2014 van Maurik reported a non-blinded RCT, the Lower Extremity Nerve Entrapment Study from the University Medical Center, Utrecht Netherlands. [53] They reported finding significant pain reduction in the operative limbs at 1 year compared to the contralateral nonoperative controls, measured by visual analogue scale. In a follow-up study of those patients at a mean of 4.6 years after surgery, pain relief in the surgical limb compared to the control limb had been maintained. [54]

These 3 RCT's build upon a series of studies reported since 1988 indicating the value of NDS to ameliorate the symptoms of painful DN.

Current evidence – Nerve Decompression Surgery for Improving Foot Skin Sensation and Decreasing Rates of Ulceration/ Amputation

The final issue is whether NDS is effective in improving foot sensation, and in the prevention of foot ulcers and subsequent amputation in DPN patients.

In 1992 Dellon first reported the results of a case series of both upper and lower limb decompressions in 60 patients with DPN, and subsequently 20 patients in a single-blinded non-randomized trial.[55-56] The later study reported that 69% of lower extremity decompressions for DPN in the series had "restoration of sensation" at a mean time of 23.3 months after surgery. In 1995 Wieman and Patel published a series of 24 patients with painful DPN who underwent tarsal tunnel decompression (including distal branches) – reported 22 achieved "relief of neuropathic pain" by 1 month post-surgery. [57] In 2004 Dellon's group reported a retrospective analysis of 50 patients with DPN who underwent lower extremity nerve decompression at a mean of 3.4 years observation. No limbs that underwent decompression experienced an ulcer; 12 of the non-operative limbs developed a foot ulceration, and a further 3 had an amputation.[58]

Multiple studies have been reported since that time, and they have been looked at in two recently published systematic review and meta-analyses. The first by Fadel et al. in 2022 concluded there was evidence for the efficacy of DNS in "reducing symptoms, ulcerations, and amputations related to DPN," but that "high-quality RCTs are required to support the utility of this intervention in this patient population." [59]

Most recently, Louca et al. published a systematic review and meta-analysis of the literature examining diabetic foot ulcer incidence following DNS. They concluded that although the clinical studies report lower incidence of diabetic foot ulcer occurrence after DNS, the studies are of "too low-level evidence and high-risk bias for recommendations." In this paper they calculated that a properly conducted RCT would require a sample size of 836 participants to definitively answer the question. [60]

Therefore, one must conclude that the current evidence for NDS to effectively improve foot sensation, and to prevent foot ulceration and subsequent amputation is encouraging but inconclusive.

Conclusion

The evidence in the literature published to date supports that NDS is effective for the amelioration of pain in painful DN. Clinicians should consider offering NDS to patients with inadequate pain relief, or intolerance of, currently available treatments. The evidence to date that NDS in DPN patients improves foot sensation and subsequently lowers rates of ulceration and foot amputation is encouraging but not conclusive. Further study is required to determine if that benefit is conferred by the surgery to DPN patients, and if beneficial to what extent.

Conflict of Interest

The author has no conflict of interest to declare.

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