ORIGINAL RESEARCH PAPER

Volume-7 | Issue-8 | August-2018 | PRINT ISSN No 2277 - 8179

INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

THE EFFICACY OF TIBIAL NERVE DECOMPRESSION IN PATIENTS WITH DIABETIC FOOT DISEASE: A CLINICAL STUDY

Plastic Surgery

R Venkat Narayanan Plastic & Reconstructive Surgeon, Armed Forces Medical College, Pune

SK Singh* Consultant Plastic Surgeon and Commandant Artificial Limb Center, Pune *Corresponding Author

RD Bavdekar Plastic & Reconstructive Surgeon, Command Hospital (AF), Bangalore

KEYWORDS

Introduction

The prevalence of diabetes is rapidly rising all over the globe at an alarming rate[1]. This metabolic disorder has undergone a major paradigm shift in the past 3 decades with the focus shifting from elderly patients to young or middle aged non-obese individuals who are now more affected by the end organ damage caused by diabetes. It is important to note that the rise in prevalence is seen across the world. In India, diabetes is fast gaining the status of a potential epidemic with more than 62 million diabetic individuals currently diagnosed with the disease.[2] According to Wild et al. the prevalence of diabetes is predicted to double globally from 171 million in 2000 to 366 million in 2030 with a maximum increase in India.[3]

Foot ulcer is one of the most common and dreaded complication of diabetes mellitus, affecting approximately 15% of diabetic patients during their lifetime.[4] This is also a frequent cause of hospitalisation and disability. Given the cultural and social importance given to the foot in our society, most of the patients with diabetic foot ulcers present to healthcare facilities fairly late with advanced foot ulcers and established deformities. Diabetic foot ulcers were estimated to be present in 4.54% newly diagnosed diabetes mellitus patients. Neuropathic type of foot ulcer is found to be more common than purely ischaemic type.[5] Neuro-ischaemic type of foot ulcer, with combination of both neuropathy and vascular occlusive disease is seen to occur in an intermediate frequency.

Foot ulceration is preventable in 80% of patients with adequate foot care and timely interventions. This helps prevent major amputations and other complications.[6] Even when frank ulceration is not evident, patients with neuropathy experience pain in the foot region which may not be localised and is very difficult to treat with routine analgesics.

Tibial nerve compression at the tarsal tunnel is known to cause significant paresthesia and foot pain in diabetic patients and decompression of nerve has been found to be beneficial in diabetic neuropathy. Improvement of sensory symptoms also has a role in increased healing of foot ulcers and decreased recurrence of ulcers.

Aims & Objectives

The aim of this study is to assess the effect of surgical decompression of tibial nerve at the tarsal tunnel in diabetic patients with neuropathic foot pain and chronic ulceration.

Objectives:

1. To assess time taken for chronic ulceration to heal after the tibial nerve decompression
2. To assess the effect of tibial nerve decompression on relief of neuropathic foot pain
3. To observe progression of foot deformity or appearance of new ulcers in other areas of the foot after tibial nerve decompression.

Materials & Methods

The study was conducted from Feb 2013 to Aug 2015 at a tertiary care armed forces referral hospital. All patients with diabetes and foot ulceration were assessed for suitability to undergo decompression of the tarsal tunnel. Eighty nine patients with foot disease and diabetes were examined and 21 patients who fulfilled the inclusion criteria were recruited in the study. After due Institutional ethical committee clearance, all diabetic patients were included after a detailed written informed consent if they had foot ulceration, paresthesia or neuropathic pain in the foot which not subsided by optimal does of NSAIDs and pregabalin or other drugs, deformity in the form of rocker bottom foot, hammer toes, loss of the foot arches, history of foot ulceration or foot surgery, which has subsequently healed.

Patients with active inflammation in the region of ankle, ulcer with soft tissue infection, clinical evidence of foot or leg ischemia, those with no ulceration or deformity and those with other co-morbidities making them unfit for surgery were excluded.

The neuropathic foot pain was scored on a numerical scale of 1 to 10, pre-operatively, at 3 weeks and 3 months post op. A score of 1 was given if pain was mild and not requiring any specific treatment. A score of 5 was given to pain which required the patient to be on a regular medication and 9 was given to severe constant pain relieved only by 2 or more medications. Ulcer healing was observed 3 weeks and 3 months postoperatively. Static 2-point discrimination was assessed pre-operatively in the areas supplied by the medial plantar, lateral plantar and calcaneal nerves in each foot and an average was obtained for each patient. This was recorded on a standard proforma individually and repeated post operatively after 3 months.

Ulceration is graded as per Wagner's classification. The nature and presence of foot deformities like bunions, callosities, collapse of arches, claw foot deformity is noted. Pre-operative X-ray of the foot was done in patients with chronic ulcers overlying bony prominences to exclude underlying chronic osteomyelitis.

Twenty one patients were taken up for decompression of the tibial nerve at tarsal tunnel over study period. Of these, there were 13 males and 8 females. The mean age of these patients was 60.09 yrs with a range from 31 to 78 years. The patients in this study had pre-existing diabetes for an average duration of 9.76 yrs (range 3- 15 years). All patients were on medications (Oral hypoglycemic agents/ Insulin analogues) for diabetes and had varying degrees of glycemic control.

All patients underwent a detailed pre-anaesthesia assessment and were operated under spinal anaesthesia, adequate lighting and loupe magnification (4x). Adequate exposure to identify the medial calcaneal nerve, medial plantar nerve, lateral plantar nerve after the bifurcation of the tibial nerve in the foot was obtained through careful placement of the incision just posterior to the posterior tibial artery pulsation in the ankle and over the calcaneum. The incision was marked starting from just proximal to the medial malleolus and extending to the talo-navicular joint or in the middle of the abductor hallucis muscle in the medial arch of the foot (Fig 1). A pneumatic tourniquet was inflated in the thigh to 150 mm Hg above the systolic pressure, after limb elevation for 5 minutes.

Fig 1: Marking of incision for Tarsal tunnel release (M= Medial malleolus)

The skin was incised and carried down to the fascia, safeguarding the branches of the saphenous nerve. The fascia was opened and posterior tibial pedicle visualised. The tibial nerve was then taped and followed deep to the flexor retinaculum. The retinaculum was incised along the course of the nerve and all branches are preserved carefully. It is important to follow all the nerve branches; the medial plantar nerve passes distally round the medial malleolus and can be followed to the abductor hallucis muscle where it enters once again in a fibroid tunnel at the level of the talonavicular joint. Meticulous tissue handling and avoidance of any injury to the nerve is the most important surgical technique while decompressing peripheral nerves. At the level of the medial malleolus the nerve could be covered by a venous plexus. The lateral plantar nerve was evaluated beneath the abductor hallucis muscle by spreading using a blunt tipped scissors. By identifying the dorsal part of the lateral plantar nerve, the calcaneal branch could be exposed (which is in 80% of the cases not divided and in 20% splits into multiple branches).

No attempt was made to dissect the perineural connective tissue or the epineurium of the tibial nerve during the decompression. Specific attention was paid to carefully preserve all extrinsic and intrinsic blood supply to the tibial nerve. After careful hemostasis using a bipolar cautery, the incision was closed in a single layer using monofilament absorbable suture. A padded compression dressing was given and tourniquet deflated. Patient was advised bed rest on the day of surgery and allowed partial weight bearing ambulation from the first post operative day onwards. Ambulation was gradually increased as tolerated by the patient, over the next 2-3 weeks. Dressing change was done on post operative day 2 and then as required. Patients were monitored for improvement in pain and ulcer healing over the next 3 months.

Results

A total of 21 patients underwent tarsal tunnel decompression over the study period (Feb 2013 to Aug 2015).There was male predominance with 13 males (62%) and 8 females (8%) [Chart 1] The mean age of the study group was 60.09 years with the youngest patient being 31 years old and the oldest being 78 years old. The patients in our study had been diagnosed to have diabetes for a mean period of 9.76 years with a range of 3 to 15 years. [Chart 3] All patients were on medication with either oral agents or a combination of oral agents and insulin analogues. Even though long term glycemic control was not assessed as a part of the study protocol, adequate control of blood sugar levels was obtained prior to surgical therapy.

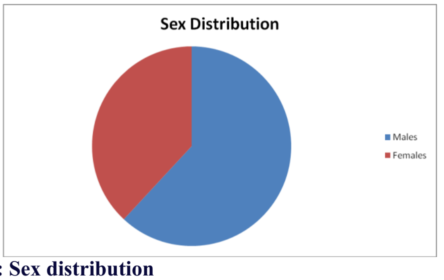

Chart 1: Sex distribution

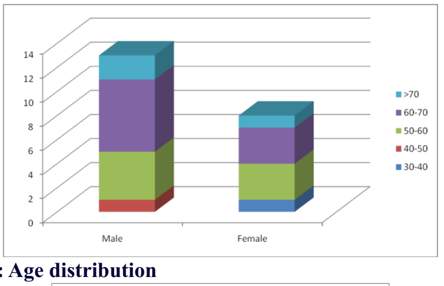

Chart 2: Age distribution

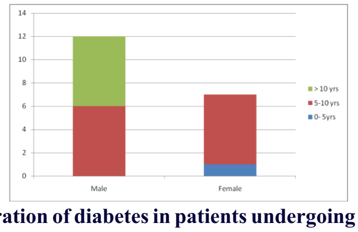

Chart 3: Duration of diabetes in patients undergoing tarsal tunnel decompression

All patients complained of varying degrees of pain in the plantar aspect of the foot either at rest or on prolonged standing associated with a burning sensation over the plantar aspect of the foot. This abnormal sensation in the foot had been present in most of the patients for an average of 20.76 months and a median period of 12-18 months, prior to the procedure (Range 10- 36 months). [Chart 4] All patients had been on oral medications for pain relief with various combinations of pregabalin, NSAIDs and other medications for different periods of time and had no relief from the medications.

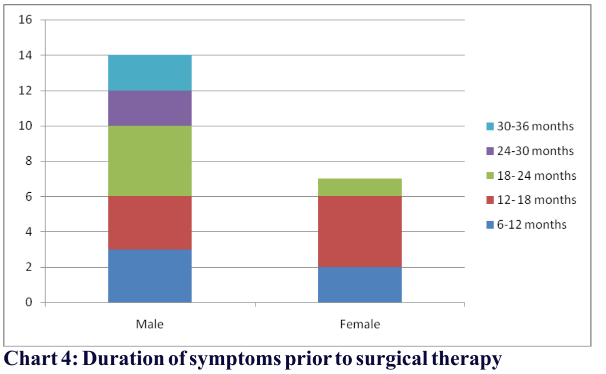

Chart 4: Duration of symptoms prior to surgical therapy

Sixteen patients had pain in the region of the forefoot radiating to the toes, predominantly in the planter aspect. The pain was worse on weight bearing and relieved partially on planter flexion at the ankle. 02 patients had pain the heel region paresthesia in the fore foot. Three patients had pain and paresthesia over the plantar aspect of the entire foot, without any specific localisation. [Chart 5]

Nineteen patients had a non healing ulcer in the foot when they reported for the study. These ulcers were of Wagner's class 1 or 2, without the evidence of any soft tissue infection or osteomyelitis 2 patients had no active ulceration but had deformities and paraesthesia in the foot. These patients had been treated with local dressings, wound care and advised off-loading for varying periods of time, prior to this study.

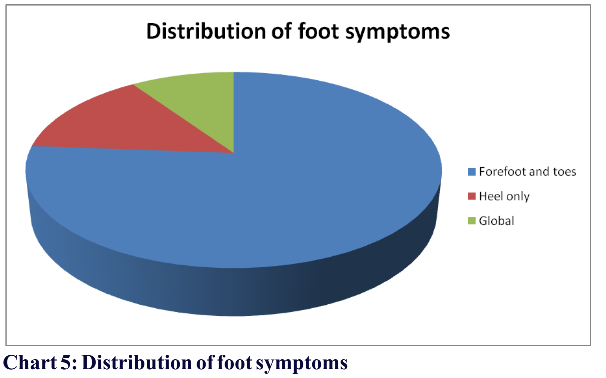

Chart 5: Distribution of foot symptoms

After tarsal tunnel decompression, the mean hospital stay was 19.38 days with a range of 5 to 28 days. Patients were discharged from hospital when they felt confident of managing their surgical site at home or when they returned to full weight bearing.

All patients were assessed for presence of paresthesia, foot pain and Static 2 point discrimination at 3 weeks and 3 months after the procedure. Student's t-test was applied for comparison of the results for pain scores and static two point discrimination. All patients had significant reduction in pain scores after 3 weeks of surgery and 3 months after surgery ($p<0.0001$). The persistent relief of symptoms at 3 months showed that the effect of nerve decompression is long lasting and not due to a temporary relief of compression. The static two point discrimination showed improvement in the post operative period but was not statistically significant in the 3 week follow up ($p=0.08$). The improvement was significant at 3 months post op ($p=0.005$).

Although the assessment of progression of foot deformity was a part of our study, in the 3 month follow up period, none of the patients showed any change in foot architecture. A longer follow up period is required to assess the same.

All patients who presented with foot ulceration had complete ulcer healing after tarsal tunnel decompression. Ulcer healing may occur due to effects of tibial nerve decompression or also has a result of immobilisation and foot elevation post-operatively. During the course of hospitalisation, they had progressively improving paraesthesia and foot pain after the procedure.

One patient in our study had ulcer recurrence during follow up due to unrecognised osteomyelilits of the underlying phalanx, which was managed by excision of the infected bone following which the ulcer healed. However, no patient had persistent symptoms of tarsal tunnel compression after the initial release and none required re- do decompression.

The most commonly encountered complication in our study was delayed wound healing. Since the incision is on the distal leg and foot, which is in itself a poorly perfused area, and because the final suture line is at right angles to the resting lines of skin tension, the wound healing tends to be delayed. The maximum time taken for wound healing was 28 days in a patient who had undergone tarsal tunnel decompression bilaterally. Other complications included wound hematoma, which was managed with repeated needle aspirations in 4 patients, pain in the proximal part of the scar seen in one patient probably due to entrapment of saphenous nerve in the suture.

Discussion

Foot disease affects 15% of all diabetic patients in their lifetime and 4.5% of newly detected diabetics have risk of developing foot ulceration.[6,7] Neuropathy is the most common feature of foot disease in diabetes and various theories have been propounded for explaining the pathogenesis of the same. Altered glucose metabolism and abnormal accumulation of polyols in the nerve sheath has been promulgated as one of the causes of neuropathy, but not proven.[7] Current research centers on the role of reactive oxygen species generated by mitochondria in the hyperglycemic dorsal root neuron thus causing cellular membrane damage and altered nerve function.[8] Addition of α-lipoic-acid, an anti-oxidant, improved nerve conduction velocity in some animal trials[9] In addition, advanced glycation end products cause altered function of matrix metalloproteases and changes in nerve conduction[13]

The end result of these metabolic and oxidative mechanisms is increased permeability of axonal cell membrane and resultant nerve edema. When these changes occur in a nerve that is confined fibro-osseous tunnel it results in increased vascular permeability, chronic oedema, myelin damage, and axonal degeneration.[10]

The tarsal tunnel is a fibro-osseous tunnel beneath the flexor retinaculum, behind and inferior to the medial malleolus. The floor is formed by the medial wall of the talus, calcaneum and the medial wall of the distal tibia. The flexor retinaculum forms the superior and inferior margins as well as the roof. The tibial nerve along with other structures course through this tunnel and innervate the plantar foot through the branches of medial plantar, lateral plantar and calcaneal nerves.[11] All three nerves, as well as the tibial nerve are susceptible to compression at the tarsal tunnel.[12] Symptoms of tibial nerve compression at the tarsal tunnel are often vague. Patients complain of pain and paresthesia in the forefoot, heel or rarely in the toes, which is worsened by dependency of foot.

The diagnosis of tarsal tunnel compression neuropathy is clinical, based on a detailed history and clinical examination. Electrophysiological studies are an adjunct to establish diagnosis. In our study, an objective assessment of tibial nerve function was done by measuring the static 2 point discrimination. Electrophysiological studies were not included as a part of the protocol to minimise the requirement of investigations in an otherwise clinically evident condition. Also, we did not include one point pressure testing with Semmes-Weinstein filament or pedo-barometry for assessment of sensations in the foot as static two point discrimination is both an objective and a sensitive marker for sensory nerve function, especially of the large diameter nerve fibres.

Surgical decompression of the tibial nerve by releasing the tarsal tunnel is appropriate when conservative treatment for neuropathic pain has failed. Key points in surgical technique are the following:

- Identification of tibial nerve under tourniquet control and magnification
- Safe release of the nerves, including medial plantar and the origin of the lateral plantar nerves
- Careful release of the medial border of the abductor hallucis and the adjacent plantar fascia[24,]
- Careful closure of the wound after proper approximation of the edges.

Reported success rates after tarsal tunnel decompression have varied in the literature from 44% to 96%.[27,14] This wide variation may be due to different criteria of patient selection and assessment. In our series, all patients had significant relief of neuropathic symptoms after tibial nerve decompression. This relief form neuropathic pain persisted after 3 months of follow up also indicating that the effect of decompression is immediate and long lasting. There was also significant improvement of static two point discrimination on the distribution of the medial plantar, lateral plantar and calcaneal nerves of these patients. This improvement was not seen significantly in the immediate post op period but after at least 3 months. It correlates with the normal nerve physiology where return of nerve function after prolonged compression is not predictable.

Complications of tarsal tunnel decompression include damage to the tibial nerve and subsequent loss of function in the foot, scarring around the nerve and resultant increase in foot symptoms and residual fibrous bands requiring re-do surgery. Damage to a branch of the saphenous nerve in the proximal part of the incision and resultant painful neuroma post-operatively has been reported.[15] The most common complication seen after tarsal tunnel decompression is delayed wound healing and wound hematoma. Since the incision for the release is at right angles to the resting lines of skin tension, closure becomes difficult after the decompression. Layered closure with absorbable monofilament suture in the depth and stainless steel staples on the skin has been found to give optimal results.[16] We encountered this complication in 9 of our patients but none required secondary procedures.

Conclusion

Patients with diabetes are prone to distressing neuropathic pain in the foot. This is due to nerve edema and compression of the tibial nerve in the narrow confines of the tarsal tunnel. In our study, we selected patients with paresthesia, pain and chronic ulceration in the foot to undergo decompression of the tibial nerve at the ankle and foot. All patients reported good relief of sensory symptoms and had ulcer healing following tarsal tunnel decompression. The commonest complication of tarsal tunnel decompression was delayed wound healing requiring increased duration of hospital stay. Indirectly, increased stay in hospital benefited these patients as it aided in ulcer healing by enforced bed rest.

We recommend that tarsal tunnel decompression to decompress the tibial nerve, be offered to patients of diabetes who have significant sensory symptoms in foot and chronic foot ulceration. Further data from a larger patient series and long term follow ups is required to assess the long term benefit of this procedure.

References

1. V Mohan, S Sandeep, R Deepa, B Shah, C Varghese: Epidemiology of type 2 diabetes: Indian scenario. Indian J Med Res 125, March 2007, pp 217-230
2. Joshi SR, Parikh RM. India - diabetes capital of the world: now heading towards hypertension. J Assoc Physicians India. 2007;55:323–4.
3. Wild S, Roglic G, Green A, Sicree R, King H. Global prevalence of diabetes-estimates for the year 2000 and projections for 2030. Diabetes Care. 2004;27(3):1047–53.
4. Shankhdhar KLK, Shankhdhar U, Shankhdhar S. Diabetic foot problems in India: An overview and potential simple approaches in a developing country. Current Diabetes Reports 2008; 8: 452-457.
5. Sinharay K, Paul UK, Bhattacharya AK, Pal SK: Prevalence of diabetic foot ulcers in newly diagnosed diabetes mellitus patients. J Indian Med Assoc. 2012 Sep;110(9):608-11.
6. Shailesh K. Shahi, Ashok Kumar, et al: Prevalence of Diabetic Foot Ulcer and Associated Risk Factors in Diabetic Patients From North India. The Journal of Diabetic Foot Complications, 2012; Volume 4, Issue 3, No. 4, Pages 83-91.
7. Llewelyn JG et al. (2005) Diabetic neuropathies. In Peripheral Neuropathy, vol 2, 1951-1991 (Eds Dyck PJ and Thomas PK) Philadelphia: Elsevier Saunders
8. King RH (2001) The role of glycation in the pathogenesis of diabetic polyneuropathy. Mol Pathol 54: 400-408.
9. Ziegler D et al. (2006) Oral treatment with alpha-lipoic acid improves symptomatic diabetic polyneuropathy: the SYDNEY 2 trial. Diabetes Care 29: 2365-2370
10. L.F Llanos, J Vila, M Nonez-Samper: Clinical symptoms and treatment of the foot and ankle nerve ntrapment syndromes. Foot and Ankle Surgery 1999 5:211-218.
11. Park TA, Del Toro DR. The medial calcaneal nerve: anatomy and nerve conduction technique. Muscle Nerve 1995;18:32–8.
12. Keck C. The tarsal tunnel syndrome. J Bone Joint Surg 1962;44A: 180-182.
13. Pfeiffer W, Cracchiolo A. Clinical results after tarsal tunnel decompression. J Bone Joint Surg 1994;76-A(8):1222–30.
14. Kaplan PE, Kernahan Jr WT. Tarsal tunnel syndrome: an electrodiagnostic and surgical correlation. J Bone Joint Surg 1981;63:96–9.
15. Kim J, Dellon AL: Pain at the site of tarsal tunnel incision due to neuroma of the posterior branch of the saphenous nerve. J Am Posiatr Med Assoc 2001 (Mar), 91:3, 09-13.
16. SL Barret: Maximising outcomes in tarsal tunnel surgery. Foot and Podiatr Intl. 20(11) Nov 2007.