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Original Research Article

NON SURGICAL REHABILITATION OF PAINFUL HEEL

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Abstract

Background: Plantar heel pain affects about 1 in 10 persons at some point in their life, and nearly 2 million people seek treatment for it annually. However, this is typically, attributed to a shotgun treatment approach or progressive and protracted escalation of intervention with non-operative treatments that can draw out over many months/years culminating in significant cost, at times compounded with diagnostic testing that can include MRI, CT, EMG, YS, PSSD and bone scans. In recent years we have seen an expanded causation for "plantar fasciitis" along with a multitude of various treatments that target these etiologies which include; neuropathic pain agents (gabapentin), PRP, EPAT/ESWT, prolotherapy, dry-needling, laser and amniotic/ chorionic membrane allograft injection. **Materials and Methods:** Materials used are a nerve/muscle stimulator (DigiStim III; Neuro Technology Inc, Kerrville, Texas); a lead wire with a gel electrode; a needle electrode (Neuro- line Inoject; Ambu, Ballerup, Denmark), 35 × 0.40 (1.4 inches × 27 gauge), with a 30-cm lead wire; 100 U of BTX A solubilized in 0.9% sterile saline without preservative; and a 3- mL sterile syringe with a draw needle. The patient is placed supine with the low-voltage lead (gel electrode) placed on the posterior lower leg. Lead wires are attached to the DigiStim jacks. The Inoject needle attached to the DigiStim is placed just distal to the plantar medial heel sulcus at the proximal abductor hallucis muscle directed posterior proximal at approximately forty-five degrees to both the transverse and sagittal planes of the foot. **Result:** An otherwise healthy daily athletic 59-year-old male presented with a 4-month history of left plantar medial heel pain. Three months of conservative treatments which included change in shoe gear, NSAID's, stretching, icing and custom orthotics were not satisfactorily effective. Findings were positive for exquisite palpable left plantar medial fascial heel pain with tension and daily post-static dyskinesia. Negative toPhalen test. Ultrasound finding of (5 mm) thickened plantar fascia distal to calcaneal insertion. **Conclusion:** Patients presenting with heel pain are commonly inaccurately diagnosed as having plantar fasciitis (used as a catch all) when in fact the diagnosis is a syndrome of several distinct pathologies that may or may not include plantar fasciitis and are of a neuropathic condition.

INTRODUCTION

Terminology for most plantar heel pain has been inconsistently described in the literature and is typically and erroneously defaulted to "plantar fasciitis". This reflects its multifactorial, poorly understood and often disputed etiology.[1–4] Therefore, the term Plantar Heel Pain Syndrome to describe the condition is preferred. It can be attributed to one or a combination of inflammatory, degenerative, and neuropathic conditions localized at the plantar heel.[4–6] Common associated inflammatory processes are plantar fasciitis from acute micro tears of the fascia, insertional periostitis, neuritis and myositis.[7–9] Degenerative processes of painful fasciosis are attributed to chronic repetitive micro injury and scaring with resultant avascularity.[10] Neuropathic conditions such as Baxter's Neuritis (inferior calcaneal nerve entrapment), Tarsal Tunnel Syndrome, medial plantar nerve entrapment, lateral plantar nerve entrapment and medial calcaneal nerve entrapment can be due to compression/entrapment or sensitization from either an inflammatory or avascular plantar heel condition.[9] Plantar heel pain affects about 1 in 10 persons at some point in their life, and nearly 2 million people seek treatment for it annually.[11] Plantar medial heel pain attributed to plantar fasciitis is the most common condition

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podiatrists see in most practices and it is commonly claimed that over 90 percent of patients are cured with conservative treatment.[12] However, this is typically, attributed to a shotgun treatment approach or progressive and protracted escalation of intervention with non-operative treatments that can draw out over many months/years culminating in significant cost, at times compounded with diagnostic testing that can include MRI, CT, EMG, YS, PSSD and bone scans. In recent years we have seen an expanded causation for "plantar fasciitis" along with a multitude of various treatments that target these etiologies which include; neuropathic pain agents (gabapentin), PRP, EPAT/ESWT, prolotherapy, dry-needling, laser and amniotic/ chorionic membrane allograft injection.[13–16] Upon failure of conservative management or "non-surgical" procedures, various operative procedures can be offered which include coblation, radiofrequency debridement, decompression, and release.[17] However, with surgical intervention there are risks which include infection, delayed healing, and nerve injury or entrapment. Long term complications due to postoperative sequelae can include medial or lateral column collapse, and recurrence of heel pain.[18] Botulinum toxin is a protein produced by the anaerobic bacterium clostridium botulinum. Seven serotypes (A–G) of botulinum neurotoxin exist as well as recombinant species. Type A was the first to be FDA approved and is most frequently used.[19,20] In small amounts botulinum toxin A (BTX A) causes muscle paralysis by blocking presynaptic release of neurotransmitter acetylcholine. Acetylcholine plays a vital role in sending signals from the nerve to the muscle causing
movement. BTX A blocks the synaptic transmission and causes the muscle to which the nerve is attached to become paralyzed. The clinical applications for BTX A have been expanding since its first use in the 1980′s for strabismus, misalignment of the eyes.[21] The scope of treatment in the lower extremity has broadened, suggesting its use not only for spastic foot or ankle seen in cerebral palsy patients but also for spastic toes,[22] plantar hyperhidrosis,[23] hallux abducto valgus,[23–26] and plantar fasciitis.[27–31] BTX A has also proven to have analgesic and anti-inflammatory properties.[31–38] In recent years, BTX A has been used for treating chronic muscular and neuropathic pain, such as migraine, myofascial pain syndrome and piriformis syndrome.[38–42] BTX A has been found to have anti-nociceptive and anti-allodynia effects and acts by modulating pain neurotransmitters including sub- stance P, glutamate and anti-inflammatory reactions.[33] Several studies have shown success in treatment of "plantar fasciitis" with BTX A injection. Most are non-specific in their injection target(s) other than the area of the plantar fascia calcaneal origin, plantar medial heel, area of pain, or in combination with the Flexor Hallucis brevis muscle.[27–31,43–45] Additionally, the subject inclusion diagnosis of the "plantar fasciitis" in these studies may be a catchall term of a syndrome consisting of one or a combination of several conditions that contribute to Plantar Heel Pain Syndrome. However, as a secondary neuropathic condition, one must appreciate the variability of the neuroanatomy at the medial aspect of the heel, the unreliability of EMG, limitations of PSSD, MRI, Tinel's sign or Valleix's Point in determining which nerve/branch is involved. Yet, there is a consistent anatomic approximation of these medial heel nerves to the Abductor Hallucis and/or Quadratus Plantae.[46–49] Surgical decompression of the fascia between these two muscles has been a well-documented treatment for recalcitrant heel pain attributed to Baxter's Neuritis, Tarsal Tunnel Syndrome and "plantar fasciitis".[6,48] Also of interest is the high density of nerve ganglia associated with the origin of the intrinsic flexors; Quadratus Plantae muscle at the calcaneus.[50,51] Thus, paralyzing these two muscles to decompress to an extent these nerves could relieve the neuropathic component of Plantar Heel Pain Syndrome. Additionally, one can appreciate the method of injection under electrical stimulation to be more precise than that of "blind" or even ultrasonic guided injection due to the insertional and approximate variability of the intrinsic muscles with the plantar fascia.[52]

MATERIALS AND METHODS

Materials used are a nerve/muscle stimulator (DigiStim III; Neuro Technology Inc, Kerrville, Texas); a lead wire with a gel electrode; a needle electrode (Neuro- line Inoject; Ambu, Ballerup, Denmark), 35 × 0.40 (1.4 inches × 27 gauge), with a 30-cm lead wire; 100 U of BTX A solubilized in 0.9% sterile saline without preservative; and a 3- mL sterile syringe with a draw needle. The patient is placed supine with the low-voltage lead (gel electrode) placed on the posterior lower leg. Lead wires are attached to the DigiStim jacks. The Inoject needle attached to the DigiStim is placed just distal to the plantar medial heel sulcus at the proximal abductor hallucis muscle directed posterior proximal at approximately forty-five degrees to both the transverse and sagittal planes of the foot. The DigiStim is turned on and the 2-Hz button is depressed, allowing for a frequency of two pulses per second to be introduced into the needle. The needle is gently advanced into the abductor muscle towards its origin. A low frequency is used to confirm the motor response of the abductor hallucis with visible contraction of the muscle and corresponding adduction of the hallux. At this point fifty units of toxin are administered. The needle is then partially retracted and redirected toward the origin of the quadratus plantae muscle at the plantar medial Process of the calcaneus. Care is taken in obtaining the motor response from the correct muscle by avoiding stimulation of the muscles in the surrounding area by using a high frequency. The needle is gently advanced until flexion of the lesser digits at the distal inter-phalangeal joints is observed.

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Fifty units of toxin are then administered. The dose selected was based on the use of BTX A in similar studies treating plantar fasciitis.[27–31,43–45] All four patients were consented for "off label" BTX A injection to treat their heel pain both verbally and in writing. They were selected for having no co-morbidities and normal lower extremity neurological, vascular, dermatological, and musculoskeletal findings except for those relating to their heel pain. Mild to moderate forefoot deformities and foot type variations of Cavo Varus and Planus were not excluded. Inclusion criteria are listed in [Table 1] and exclusion criteria in [Table 2]. All patients presented with plantar medial heel pain for at least 3 months which had not satisfactorily responded to non-operative management with at least 4 of the following 7 treatments: Physical therapy; NSAID's/ice; corticosteroid injection; stretching exercises; night splint; change in shoe gear and orthotics. At the initial baseline/pre-injection visit patients were assessed clinically. Initial function was scored with the Foot and Ankle Ability Measure (FAAM),[53] and Plantar Fasciitis Pain/Disability Scale (PFPS) which includes a visual analog scale (VAS).[54] All patients were instructed to cease any prior non-operative treatments including R.I.C.E., cortisone injection, PRP, prolotherapy, PT, night splint, NSAID's. Continued use of shoe inserts or custom orthotics were permitted as well as any prior shoe modification and lower extremity stretching exercises. Patient were advised to maintain their normal daily and athletic activities as tolerated. Follow up examinations were at; 1 week, 3 weeks, 6 weeks, 12 weeks and 26 weeks post-V.A.S. = Visual Analog Scale. 0–100, 0 being no pain and 100 being worst possible pain. FAAM = Foot and Ankle Ability Measure/Activities of Daily Living Sub Scale. Current level of function during usual activities of daily living from 0 to 100 with 0 being unable to perform any usual daily activities. PFPS = Plantar Fasciitis Pain/Disability Scale. 0–97. 0 being equal to no pain or disability.

Table 1

Inclusion Criteria

Age over 18 years with heel pain for 3 months and a baseline V.A.S. of 4 or more for
heel pain in the last 48 h and one or more of the following:

1. Ultrasound positive for thickened > 4 mm plantar fascia.
2. Inability to abduct the 5th toe.
3. Positive Tinel's sign or Valleix's Point to one or more of the posterior tibial nerve, lateral plantar nerve, medial plantar nerve, infra-calcaneal nerve, medial calcaneal nerve.
4. Post-static dyskinesia.
5. Plantar medial heel pain with Phalen's maneuverer.
6. Plantar medial fascial heel pain with tension.

Table 2

Exclusion Criteria

1. Calcaneal bone cyst.
2. Rearfoot soft tissue masses/bursae, tendon pathology.
3. Plantar heel calcifications.
4. Rearfoot dermal conditions.
5. Prior treatment with BTX in the lower extremity.
6. History of coagulopathies.
7. History of rear-foot or ankle surgery.
8. History of HIV.
9. Aminoglycosides.
10. Myastania Gravis.
11. Pregnancy or lactation.
12. Alcoholism or drug abuse.
13. Narcotic pain medication.
14. History of systemic disorders arthritis/fibromyalgia/spondylolisthesis/
15. radiculopathy/sciatica.
16. Rear foot fracture.
17. Heel fat pad atrophy.
18. Trauma.
19. Other conditions of ambulatory disability.

RESULTS

An otherwise healthy daily athletic 59-year-old male presented with a 4-month history of left plantar medial heel pain. Three months of conservative treatments which included change in shoe gear, NSAID's, stretching, icing and custom orthotics were not satisfactorily effective. Findings were positive for exquisite palpable left plantar medial fascial heel pain with tension and daily post-static dyskinesia. Negative toPhalen test. Ultrasound finding of (5 mm) thickened plantar fascia distal to calcaneal insertion. The rest of the podiatric exam was unremarkable including WB foot radiographs. Patient #1 end point results are noted in [Table 3].

Table 3

48-hour VAS and FAAM at pre-injection and 1, 3, 6, 12, and 26-weeks postinjection and PFPS at pre-injection and 6, 12, and 26-weeks post-injection.

Table 3

Patient	Pre-injection	1-week P.I.	3-week P.I.	6-week P.I.	12-week P.I.	24-week P.I.
V.A.S./100	76	51	31	20	11	11
FAAM	34%	71%	90%	92%	93%	96%
PFPS	89			21.4	14	16

V.A.S. = Visual Analog Scale. 0–100, 0 being no pain and 100 being worst possible pain.
FAAM = Foot and Ankle Ability Measure/Activities of Daily Living Sub Scale.
Current level of function during usual activities of daily living from 0 to 100 with 0 being unable to perform any usual daily activities.

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PFPS = Plantar Fasciitis Pain/Disability Scale. 0−97. 0 being equal to no pain or disability.
[Table 4] 48-hour VAS and FAAM at pre-injection and 1, 3, 6, 12, and 26-weeks postinjection and PFPS at pre-injection and 6, 12, and 26-weeks post-injection.

Table 4

Patient	Pre-injection	1-week P.I.	3-week P.I.	6-week P.I.	12-week P.I.	24-week P.I.
V.A.S./100	81	31	31	26	21	21
FAAM	57%	65%	93%	97%	74%	92%
PFPS	60.6			37	42	22

V.A.S. = Visual Analog Scale. 0–100, 0 being no pain and 100 being worst possible pain.
FAAM = Foot and Ankle Ability Measure/Activities of Daily Living Sub Scale.
Current level of function during usual activities of daily living from 0 to 100 with 0 being unable to perform any usual daily activities.
PFPS = Plantar Fasciitis Pain/Disability Scale. 0−97. 0 being equal to no pain or disability.

A 50-year-old otherwise healthy female with greater than 2 year progressively painful right plantar medial heel. Previous treatments included cortisone injection, custom orthotics, physical therapy, night splint, stretching, icing and NSAID's. Findings included sub fascial pain with palpation to the plantar medial heel with positive Tinel's to both plantar medial arch and Valleix's Point to infra-calcaneal nerve (Baxter's). Positive to Phalen test. Plantar medial heel sensory loss. Ultrasound confirmed insertional thickening of the plantar fascia. Weakness to abduction right 5th toe. Remainder of the podiatric exam was unremarkable. No plantar calcaneal spur on lateral radiograph was present. Patient #2 end point results are noted in [Table 4].

A 70-year-old otherwise healthy decades avid runner with five months of daily left heel pain causing him to cease running. Minimal relief with change in shoes, custom orthotics, three cortisone injections, stretching and massage icing. Positive with Ultrasound for insertional thickening (6 mm) of the plantar fascia. Plantar medial palpable fascial pain with tension and sub fascial pain and with Valleix's Point to the infra-calcaneal nerve. Positive to Phalen test. Plantar calcaneal spur in lateral radiograph. Weakness to abduction of the left 5th toe. Remainder of the podiatric exam was unremarkable. Patient #3 end point results are noted.

A 55-year-old otherwise healthy female with over 3-year intermittent left plantar heel pain now constant and progressive in the last 3 months throughout day and worse with standing. Some relief with heat and massage. Minimal relief with conservative treatments of NSAID's, OTC and custom orthotics, various shoes, cortisone injections and physical therapy. Plantar medial heel pain with and without fascial tension left foot. Negative Phalen test. Previous HAV corrective surgery with distal osteotomy. Remainder of the podiatric exam was unremarkable. Patient #4 end point results are noted.

Statistical analysis: Means and standard deviations were computed for all measures, and the normality of the distributions was confirmed using Shapiro–Wilk tests. Paired t-tests were conducted to determine the statistical significance of improvements in pain levels and functionality from preinjection to 1, 3, 6, 12, and 26-weeks post-injection. Significant improvements from pre-injection were found for all measures at each postinjection time point.

DISCUSSION

Plantar Heel Pain Syndrome and its manifestations may likely be related to a compression of the medial/plantar nerve complex of the heel between the Quadratus Plantae and/or the Abductor Hallucis muscles. Specifically, one, or a combination, of the medial plantar nerve, lateral plantar nerve, first branch of the lateral plantar nerve or variant thereof, and the dense neuro-ganglia within the Quadratus Plantae at its calcaneal origin. In my experience, patients with recalcitrant Plantar Heel Pain Syndrome, regardless of the variable pathology, who have failed conventional conservative treatments obtain resolution with surgical decompression between these intrinsic foot muscles.[49] Therefore, injecting BTX A specifically into these two muscles for temporary paralysis should act as a neurologic decompression (as is achieved surgically), breaking the pain cycle. There may also be some direct neuron-analgesic and musculoskeletal anti-inflammatory benefit from the toxin's diffusion to the plantar medial heel area which could account for some success of indiscriminate injection of BTX A to the plantar medial heel area for plantar fasciitis. Although there has been no reported adverse effect of temporary paralysis of the intrinsic foot muscles, it is something to consider and evaluate in a larger study.[55] This injection paradigm may be a valuable early intervention for Plantar Heel Pain Syndrome before considering costlier and less efficacious prolonged treatments and tests that may only address a singular etiology. Certainly, a large well controlled study will be needed to bear this out or not. The number of patients needed for a comprehensive study based on this preliminary statistical analysis can be determined as follows: The majority of the paired t-test results had medium to large effect sizes. The following power analysis provides a conservative estimate based on a medium effect size. Given a medium effect size (Cohen's d = .5) and a minimum sample size of 34, a two-tailed paired t-test will have 80% power to detect a difference at the .05 level of

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significance. Power analysis results using G*Power 3.1.9.2.

CONCLUSION

Patients presenting with heel pain are commonly inaccurately diagnosed as having plantar fasciitis (used as a catch all) when in fact the diagnosis is a syndrome of several distinct pathologies that may or may not include plantar fasciitis and are of a neuropathic condition. It may include Tarsal Tunnel Syndrome, medial or lateral plantar nerve entrapment, "Baxter's Neuritis", mechanically induced intrinsic muscle inflammation, insertional calcaneal enthesopathy and plantar fasciosis. This small investigation of four diverse case reports may offer insight into a more global efficacy using a specific injection paradigm that considers the multifactorial etiologies of Heel Pain Syndrome as a neuropathic condition. Long term relief and prophylaxis against recurrence may likely require addressing the underlying, mechanical, inflammatory, or degenerative processes of the Syndrome, or if initially effective, possibly additional BTX injection at one to two-year intervals. Clearly a larger and higher-level placebo controlled blinded study with at least 40 (forty) patients is needed to challenge the viability of this treatment in the general population for the Plantar Heel Pain Syndrome condition.

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