CHAPTER 25

SUBCALCANEAL PAIN

Jeffrey S. Boberg, D.P.M.
Scott A. Melamed, D.P.M.

INTRODUCTION

The list of differential diagnoses for plantar heel pain is extensive. The etiology of heel pain can be divided into three basic categories: systemic, osseous and soft tissue. This paper will briefly review the more common causes of heel pain and propose a new etiology and treatment plan for individuals with heel pain.

SYSTEMIC ETIOLOGY

Heel pain may be the result of a multitude of systemic disorders. The clinical presentation is usually that of generalized, bilateral heel pain, often with edema. The heels are diffusely tender and should not typically be confused with mechanical heel pain.

The systemic disorders can be subdivided into three major categories: immunologic, infectious and metabolic. Gout is probably the most common metabolic disorder and can be diagnosed by radiographic findings or serology. Immune disorders can be further divided into the seropositive and seronegative arthropathies, but this does not change the clinical picture.

Infectious causes of heel pain can be classified as either active or reactive. Active infectious processes are fortunately rare and failure to diagnose could lead to disastrous consequences. Group B strep, gonorrhea, salmonella and tuberculosis have all been associated with hematogenous calcaneal osteomyelitis. Reactive infectious arthridities are non-purulent joint inflammations that develop after an infection has occurred elsewhere in the body. The primary infection is believed to produce an antigen similar to surface antigens on joint surfaces, triggering an autoimmune response. This has been documented in patients with HIV, Reiter's syndrome and rheumatoid arthritis, and may be a cause of plantar heel pain. The author has had several patients with bilateral heel pain who were non-responsive to conventional care and had dramatic improvement in symptoms when antibiotics were initiated for non-podiatric causes. A review of the literature clearly demonstrates that patients with bilateral heel pain do not respond to surgical treatment as well as patients with unilateral pain.[1-4] Perhaps the etiology in these patients is not mechanical, and a course of antibiotics might be appropriate.

The use of antibiotics in the treatment of joint pain remains controversial. The available literature does show limited success with the use of macrolides and tetracylines. Macrolides are believed to possess anti-inflammatory properties through decreased leukocyte activity, diminished levels of cytokines and PGE2. The tetracyclines have been shown to inhibit collagenase and metalloproteases, enzymes present in the breakdown of cartilage. They also reduce the rate of phagocytosis, leukotaxis and T-cell activity. These drugs also possess anti-oxidant effects.

There have been successful outcomes in three double-blind studies in which antibiotics have been used to treat rheumatoid arthritis. In one study of patients with active rheumatoid arthritis of less than one year's duration, 44% of patients were in or near remission at 3.3 years after initiating antibiotic therapy of minocycline (100 mg BID). In another study, (a 48-week open trial involving 18 patients with active rheumatoid arthritis), all patients were on 200 mg of minocycline daily. Statistically significant improvement was noted on almost all variables of disease activity, however, antibiotics such as ciprofloxin, rifampin, metronidazole and griseofulvin have not been shown to be effective.

OSSEOUS ETIOLOGY

The inferior calcaneal step has been identified as a source of heel pain. The prominence of the calcaneal tuber has been implicated as a source of unremitting pain by Malay,[5] but has not been confirmed by others. In a review of 195 painful heels, Malay found 8.3% to have prominent calcaneal tuberosities and half of these were non-responsive to conservative

care and required surgical intervention.[5]

The inferior calcaneal spur has long been associated with heel pain. The spur has been clearly shown to lie within the intrinsic musculature and not in the plantar fascia.[6,7] The first branch of the lateral plantar nerve lies within close proximity to this calcaneal prominence. Population studies have shown the spur to be present in 8% to 18% of the general population.[8,9] However, once the age of 50 is reached, the incidence climbs to over 50%.[10] In symptomatic individuals, a spur was present 75% of the time. It was, however, also present in two-thirds of the asymptomatic feet.[11] Overall, surgical results from resection of the calcaneal spur have been good and may even show better long-term benefits versus fascial resection alone.[12,13] Problems associated with heel spur resection include sensory loss and delayed wound healing following a longitudinal medial incision.

SOFT TISSUE ETIOLOGY

Neurologic Etiology

Non-osseous causes of heel pain may primarily involve either the nerves, fascia or muscles.

Przylucki and Jones,[14] were the first to recognize entrapment of the muscular branch to the abductor digiti minimi as a source of heel pain. Release of this nerve was later popularized by Baxter. Surgical decompression of the nerve has been shown to be uniformly successful. A review of several studies involving a total of 158 patients revealed over 90% of the patients had good to excellent results.[15-18] Unfortunately, there are no clear diagnostic tests or physical findings to confirm this diagnosis. Electrodiagnostic testing has not proven helpful and need not be ordered.[16] Results of the physical exam are equivocal. Many of the reported clinical findings are non-diagnostic for nerve entrapment and commonly found with other etiologies of heel pain.

Fascial Etiology

Plantar fasciitis is currently believed to be the most common cause of heel pain. Excessive pull of the plantar fascia, most commonly seen in a pronated foot, will irritate the calcaneus resulting in painful ambulation. Fascitis as a cause of heel pain is further supported by surgical reports of excellent relief of pain by simply releasing the plantar fascia. Long-term studies have shown good to excellent results in over 85% of patients whether the release was performed by open, percutaneous or endoscopic techniques.[4,19-24]

It is now well-documented that transection of the plantar fascia may clinically destabilize the foot and yield a myriad of pain syndromes. Digital instability, metatarsalgia and lateral column pain are some of the more commonly reported complications following fasciotomy.[25-28] There are, however, multiple inconsistencies with the fascial etiology theory. Despite all surgical reports of fascial resection, either open or endoscopic, there is little mention of fascial inflammation. One would surmise that chronically inflamed fascia requiring surgical intervention would yield some degree of clinically visible inflammation. Occasional biopsies of fascia have been performed in conjunction with heel surgery; these have failed to demonstrate evidence of inflammation. Of over 40 articles reviewed, only two noted some evidence of pathologic changes.[29,30] One of the papers described a myxoid degeneration more consistent with age than acute inflammatory changes.[31] Several authors now recommend sectioning only one-half to one-third of the fascia to reduce the chance of developing mechanical aberrations. If tension on the fascia were responsible for heel pain, one would anticipate even greater pain if half the fascia were cut, because all the pulling forces would now be transmitted to the remaining fibers. Additionally, the fascia is non-contractile. Pain in the morning and after rest, even very brief periods, is not consistent with an inelastic, non-contractile ligament.

The physical exam also reveals inconsistencies. Most patients with heel pain have little pain at the actual insertion of the fascia, into the proximal more central part of the heel. Typically, heel pain patients have pain along the medial heel extending distally into the medial arch. After prolonged standing or high levels of activity, the heel can become swollen and a Tinel's sign of the medial plantar nerve can often be elicited. One consistent finding is the similar success rate for all types of surgical approaches. Plantar fasciotomy, heel spur resection and nerve decompression all seem to be equally effective. The author believes the common denominator in all these procedures is fascial decompression.

Muscular Etiology

The author, therefore, proposes the primary etiology of heel pain is overuse of the intrinsic musculature similar to what is seen in chronic exertional compartment syndrome. The intrinsic muscles are supinators of the midtarsal and subtalar joints, hence their overuse in the pronated foot, typically seen in patients with heel pain. Physical findings are consistent as well. Tenderness on exam is usually most intense over the abductor muscle belly along the medial heel and proximal arch. Since the posterior tibial nerve penetrates this muscle, a Tinel's sign and other neurologic signs and symptoms can be readily explained. Post-static dysgenesis can be more readily appreciated by contracture of an inflammed muscle.

Radiographic findings are also consistent. Amis noted an increase in subfascial thickness (the tissues superior to the fascia).[32] Calcaneal spurs, as previously noted, are located within the intrinsic musculature, not the plantar fascia. Bone scans which typically show an increased uptake in the calcaneus also show increased tracer in the medial arch in the blood flow and blood pool phases. This would be expected with tracer uptake in the muscles, not the relatively avascular fascia. In the author's experience an MRI will also show edema within the muscles and no significant fascial aberrations.

Non-surgical treatment is designed to allow relaxation of the plantar musculature and prevent their overuse. Arch stabilization through orthotics, strappings, casting, shoe modifications, etc. are common and acceptable methods of treating heel pain. Oral as well as parenteral anti-inflammatory medications are used frequently and anecdotally useful. There are no controlled studies demonstrating the long-term usefulness of these anti-inflammatory medications.[33-37] The author has used injections of botulinum toxin type A in several patients with unremitting heel pain. By causing a paralysis of the intrinsic muscles, this will hopefully allow for relaxation and ultimate healing. In three patients the author has been unsuccessful in obtaining a paralytic muscle. The use of this toxin is very safe and no serious side effects have been reported, but at $400 for a 100 unit injection the treatment can be cost-prohibitive.

The author's current surgical approach is a fascial decompression procedure. The actual surgical technique is similar to that of neurolysis as described by Baxter. A vertical incision is placed along the medial heel, angling slightly distal as it approaches the plantar skin. The incision is deepened and the fascia over the abductor muscle (medial band of plantar fascia) is incised. The abductor is retracted and the thick underlying fascial band is incised. This will decompress the muscles in the plantar central and plantar medial compartments. The plantar fascia is left intact and mechanical stability of the foot is preserved. The patient is placed in a weight-bearing cast for one month.

The author wishes to emphasize these are theoretical precepts in the treatment of heel pain. While the author has taken this approach recently, sufficient data has not been collected to make any definitive recommendations.

REFERENCES

1. Wolgin M, Cook C, Graham C, Mauldin D: Conservative treatment of plantar fasciitis: long-term follow-up. *Foot Ankle* 15(3): 97-102, 1994.
2. Powell M, Post WR, et al: Effective treatment of chronic plantar fasciitis with dorsiflexion night splints: a crossover prospective randomized outcome study. *Foot Ankle Intl* 19(1):10-17, 1998.
3. Mizel MS, Marymont JV, Trepman E: Treatment of plantar fasciitis with a night splint and shoe modification consisting of a steel shank and anterior rocker bottom. *Foot Ankle Intl* 17(12): 732-735, 1996.
4. O'Malley, MJ, Page: Endoscopic Plantar Fasciotomy for Chronic Heel Pain. *Foot Ankle* 21: 505-510, 2000.
5. Malay DS: Plantar fasciitis and heel spur syndrome: a retrospective analysis. *Update '96- Reconstructive Surgery of the Foot and Leg*, Podiatry Institute Publishing, Tucker, GA 133-135, 1996.
6. McCarthy DJ, Gorecki GE: The anatomical basis of inferior calcaneal lesions. *J Am Podiatry Assoc* 69: 527, 1979.
7. Foreman MW, Green MA: The role of intrinsic musculature in the formation of Inferior calcaneal exostoses. *Clin Pod Med Surg* 7(2):217-223, 1990.
8. Shama SS, Kominsky SJ, Lamont H: Prevalence of non-painful heel spur and its relation to postural foot position. *J Am Podiatry Assoc* 73(3):122-123, 1983.
9. Prichasuk S, Subhadrabandhu T: The relationship of pes planus and calcaneal spur to plantar heel pain. *Clin Ortho Rel Res* 306:192-196, 1994.
10. Banadda BM, Gona O, et al:Calcaneal spurs in a black African population. *Foot and Ankle* 13(6): 352-354, 1992.
11. Williams PL, Smibert JG, et al: Imaging study of the painful heel syndrome. *Foot and Ankle* 7(6): 345-349, 1987.
12. Gormley J, Kuwada GT: Retrospective analysis of calcaneal spur removal and complete fascial release for the treatment of chronic heel pain. *J Foot Ankle Surg* 31(2):166-169, 1992.
13. Brekke, MK, Green, DR,: Retrospective Analysis of Minimal Incision, Endoscopic and Open Procedures for Heel spur Syndrome. Update '97, *J Am Podiatry Med Assoc* 88:64-72,1998
14. Przylucki H, Jones CL: Entrapment neuropathy of muscle branch of the lateral plantar nerve. *J Am Podiatry Assoc* 71(3):119-124, 1981.
15. Sammarco GJ, Helfrey RB: Surgical treatment of recalcitrant plantar fasciitis. *Foot & Ankle Intl* 17(9):520-526, 1996.
16. Baxter DE, Pfeffer GB: Treatment of chronic heel pain by the surgical release of the first branch of the lateral plantar nerve. *Clin Ortho Rel Res* 279:229-236, 1992
17. Goecker RM, Banks, AS,: Analysis of Release of the First Branch of the Lateral Plantar Nerve. *J Am Podiatry Med Assoc* 90:281-286, 2000
18. Baxter DE, Thigpen CM: Heel pain—operative results. *Foot and Ankle* 5(1):16-25, 1984.

19. Benton-Weil W, Borrelli AH, Weil LS Jr, Weil LS Sr: Percutaneous plantar fasciotomy: a minimally invasive procedure for recalcitrant plantar fasciitis. *J Foot & Ankle Surg* 37(4):269-272, 1998.
20. Perelman GK, Figura MA, Sandberg NS: The medial instep plantar fasciotomy. *J Foot Ankle Surg* 34(5):447-457, 1995.
21. Graves RH, et al: Fluoroscopy-assisted plantar fasciotomy and calcaneal exostectomy: a retrospective study and comparison of surgical techniques. *J Foot Ankle Surg* 33(5):475-481, 1994.
22. Lundeen RO, Aziz S, Burks JB: Endoscopic Plantar Fasciotomy: A Retrospective Analysis of Results in 53 Patients, *J Foot Ankle Surg* 39:208-217, 2000
23. Stone PA, McClure LP: Retrospective Review of Endoscopic Plantar Fasciotomy: 1994 through 1997. *J Am Podiatric Med Assoc* 89(2):89-93, 1999.
24. Woelffer, KE, Figura, MA, Sandberg, NS, Five-Year Follow-up of Instep Plantar Fasciotomy for Chronic Heel Pain, *J Foot Ankle Surg* 39:218-233, 2000
25. Sharkey NA, Ferris L, Donahue SW: Biomechanical consequences of plantar fascial release or rupture during gait: part I — disruptions in longitudinal arch conformation. *Foot & Ankle Intl* 19(12):812-819, 1998.
26. Murphy GA, Pneumaticos SG, et al: Biomechanical consequences of sequential plantar fascia release. *Foot & Ankle Intl* 19(3):149-152, 1998.
27. Thordarson DB, Kumar PJ, et al: Effect of partial versus complete plantar fasciotomy on the windlass mechanism. *Foot & Ankle Intl* 18(1):16-19, 1997.
28. Kitaoka HB, Luo ZP, An KN: Mechanical behavior of the foot and ankle after Plantar Fascia Release in the Unstable foot. *Foot Ankle* 18:8-15, 1997
29. LeMelle DP, Kisilewicz P, Janis LR: Chronic plantar fascial inflammation and fibrosis. *Clin Pod Med and Surg* 7(2):385-389, 1990.
30. Leach RE, Seavey MS, Salter DK: Results of surgery in athletes with plantar fasciitis. *Foot and Ankle* 7(3):156-161, 1986.
31. Tountas AA, Fornasier VL: Operative treatment of subcalcaneal pain. *Clin Ortho Rel Res* 332:170-178, 1996.
32. Amis J, Jennings L, Graham D, Graham CE: Painful heel syndrome: radiographic and treatment assessment. *Foot and Ankle* 9(2):91-95, 1988.
33. O'Brien D, Martin WJ: A retrospective analysis of heel pain. *J Am Podiatric Med Assoc* 75(8): 416-418, 1985.
34. Miller RA, Torres J, McGuire M: Efficacy of first-time steroid injection for heel syndrome. *Foot & Ankle Intl* 16(10): 610-612, 1995.
35. Scherer PR, et al: Heel pain syndrome: pathomechanics and nonsurgical treatment. *J Am Podiatric Med Assoc* 81(2): 68-72, 1991.
36. Gill LH, Kiebzak GM: Outcome of nonsurgical treatment of plantar fasciitis. *Foot & Ankle Intl* 17(9): 527-532, 1996.
37. Lynch DM, Goforth WP, et al: Conservative treatment of plantar fasciitis: a prospective study. *J Am Podiatric Med Assoc* 88(8):375-380, 1998.