Standard of Care: Plantar Fasciitis

Case Type / Diagnosis: (diagnosis specific, impairment/ dysfunction specific)

ICD9 - 728.71 plantar fibromatosis

Plantar fasciitis is an inflammatory condition that occurs as a result of overstressing the plantar fascia. It is the most common cause of inferior heel pain and has been diagnosed in patients from the ages of 8-80. [1-4] Plantar fasciitis affects approximately 10% of the population and is more commonly found in middle-aged women and younger male runners.[2, 3, 5] A majority of the patients diagnosed with this inflammatory condition are over the age of 40.[3] Bilateral symptoms can occur in 20-30% of those diagnosed with plantar fasciitis.[6] However in these cases it is important to rule out other systemic processes such as rheumatoid arthritis, systemic lupus erythematosus, Reiter's disease, gout and ankylosing spondylitis.[3, 6] The primary symptom of plantar fasciitis is pain in the heel when the patient first rises in the morning and when the plantar fascia is palpated over it's origin at the medial calcaneal tuberosity.[2-4, 6, 7]

The plantar fascia (aponeurosis) is a thick fibrous band of connective tissue that originates at the medial and lateral tuberosities of the calcaneus. It runs longitudinally and has 3 portions, medial, central, and lateral. At the midfoot level, the fascia divides into 5 separate bands, which blend with the flexor tendon sheaths and transverse metatarsal ligaments and attach at the base of each proximal phalange. The central portion of the plantar fascia is the largest section. It is the most superficial layer of the plantar fascia, originating at the medial calcaneal tuberosity and inserting on the proximal phalanges of the five toes. The medial band derives slightly more distally and medially to the central portion. The medial portion covers the great toe intrinsic muscles and is continuous with the abductor hallucis muscle. The lateral band comes from the lateral portion of the medial calcaneal tuberosity along with the abductor digiti minimi muscle. This portion of the fascia gets thinner as it goes distally and is uncommonly involved in plantar fasciitis. [2, 3, 7-9]

The function of the plantar fascia is to augment the biomechanics of the foot during the stance phase of gait. At heel strike (initial contact), the plantar fascia is in a slack position. This allows the midfoot to remain flexible so it can conform to uneven surfaces and enhance its ability to absorb any shock it may encounter as the foot flattens. As one moves through the stance phase of gait into toe off (preswing), the ankle, foot and toes move into a dorsiflexed position. As the foot and toes dorsiflex, the midtarsal joints are passively extended causing the plantar fascia to be stretched distally from its origin on the medial calcaneal tubercle. This action approximates the rearfoot and hindfoot, increasing the arch height. Subsequently the midtarsal bones become more stable as a result of the arch heightening. This creates a stiffer lever for more efficient push off by the foot. This action of the plantar fascia is known as the Windlass mechanism.[3, 9-11]

The etiology of plantar fasciitis is multifactorial. The tension placed on the plantar fascia will increase as a result of anatomical factors such as abnormal foot posture or tight/weak posterior calf musculature. In addition, environmental factors such as increased frequency/distance/speed of walking or running, a change in terrain or changes in foot wear will place abnormal stress on this tissue structure. However it appears that the combination of both anatomical and environmental factors eventually lead to dysfunction and overload of the fascia.[12-14]

Typically plantar fasciitis is an inflammatory condition that results from an overload of the plantar fascia at its insertion on the calcaneus. Occasionally one may sustain an overload in the fibers in the middle of the fascia.[1-4, 7, 10, 12, 13, 15] Overloading of the fibers will occur whether caused by excessive activity on a normal foot or stress caused by a foot with abnormal structure, repetitive traction stress that exceeds the plantar fascia's ability to stretch. This overload results in microtears in this soft tissue at or near the interface of the bone and fascia.[3, 7, 8, 10, 13, 15] Initially an inflammatory response develops but with a chronic overload, granulation tissue consistent with tendinosis develops as demonstrated by biopsy.[3, 10, 15] There is increasing evidence that many of the conditions thought to be a result of overuse do not involve inflammation. Histologically, abnormal tendons from patients with chronic tendinopathy demonstrate a loss of collagen continuity, an increase in ground substance and vascularity and an increase in the presence of fibroblasts and myofibroblasts. In patients with chronic tendinopathy, very few to no inflammatory cells are found.[16, 17] This is true with plantar fasciitis as well. Collagen necrosis, angiofibroblastic hyperplasia, chondroid metaplasia and matrix calcification are found histologically.[10]

The most common risk factors associated with plantar fasciitis are:[1-4, 6, 7, 10, 13, 18]

- Tightness or weakness of the posterior calf musculature
- Pes planus or pes cavus foot structures
- Sudden gain in weight or obesity
- Unaccustomed walking or running (i.e. increased speed, distance or uphill)
- Change in walking or running surface
- Occupations involving prolonged weightbearing
- Shoes with poor cushioning

Each of the above factors can predispose an individual to plantar fasciitis due to abnormal biomechanics in the foot.

The posterior calf musculature have an integral part in the functioning of the plantar fascia. Any dysfunction in the posterior calf musculature, either tightness or weakness, will lead to an alteration in the normal biomechanics of the foot.[8, 12, 13] Tightness in the posterior calf will cause the calcaneus to be more everted at heel strike (initial contact) and push off (preswing). This leads to a restriction in the midfoot's ability to supinate and decreases the amount of dorsiflexion achieved during late stance and early push off (preswing). On the other hand, weakness in the posterior calf muscles will decrease the amount of propulsion during push off (preswing). This leads to an increased loading on the intrinsic muscles of the foot and the plantar fascia at its calcaneal attachment. These abnormalities lead to poor biomechanics of the foot that alter its force absorption and production. The resultant abnormal biomechanics increase the tensile strain on the plantar fascia and will predispose an individual to overload at its insertion.[12, 13]

Abnormal foot structure such as pes planus and pes cavus can predispose an individual to plantar fasciitis.[7, 8] The planus foot will place an increased load on the plantar fascia at its insertion on the calcaneus.[7, 8, 12, 13] The pes planus foot tends to demonstrate excessive subtalar joint pronation that leads to excessive calcaneal eversion. This excessive eversion will lead to stretching of the plantar fascia during the foot flat (loading) phase of gait as the medial longitudinal arch will lengthen more than in the normal foot. The pes planus foot structure can also predispose an individual to plantar fasciitis during the propulsive phase of gait. As has been described during the Windlass Mechanism, the plantar fascia is responsible for stabilizing the arch during the propulsive phase of gait. In a foot that has a less stable arch (as is typical in the planus foot), more force is placed on the plantar fascia to stabilize and elevate the arch. This increased force, if combined with other factors could lead to overload of the fascia.[10, 13]

The foot with a more cavus structure has limited calcaneal eversion that results in an elevated arch with limited subtalar joint motion. Typically this structure is rigid and the high arch results in an approximation of the forefoot and hindfoot. This causes a shortening of the plantar fascia. The ability of the pes cavus foot to dissipate weightbearing forces is somewhat limited.[12] Due to the relative immobility of the bony structures in the pes cavus foot, the soft tissues of the foot absorb the weightbearing forces placed on it. The pes cavus foot structure, if subjected to excessive or repetitive forces, will produce increased stress on the insertion of the fascia on the calcaneus and predispose someone to plantar fasciitis.[2, 3, 9, 10, 12] The cavus foot tends to produce more stress on the plantar fascia during the initial stages of stance (heel strike to midstance), while the planus foot stresses the fascia during mid to late stance and toe off.[9]

Obesity is a very common factor in patients with plantar fasciitis.[2, 3] Obesity or any sudden increase in body weight will subsequently cause an increase in the amount of force the plantar fascia receives during the stance phase and will predispose a person to plantar fasciitis.[3]

Other factors that can predispose an individual to plantar fasciitis is any unaccustomed walking or running (i.e. increased speed, distance or uphill), change in walking or running surface, occupations involving prolonged weightbearing or shoes with poor cushioning. These factors will introduce the fascia to an unusual or increased amount of stress that the tissue is unaccustomed to. This abnormal stress can lead to an overloading of the plantar fascia.[2-4, 6]

It is a common thought by many health care professionals that calcaneal osteophytes (bone spurs) are a cause for plantar heel pain however; calcaneal bone spurs tend to be more a result of any abnormal stress placed on the plantar fascia.[3, 9] Any abnormal stress that is placed on the plantar fascia will result in a chronic tractioning at its insertion onto the medial calcaneal tuberosity. This will lead to periosteal failure and subsequent avulsion of the periosteum at the tuberosity. The void created when the periosteum is pulled away is filled in with calcium (exostosis) and a heel spur will develop. Tisdel et al.[1], found 50% of patients with heel pain do not have a bone spur and 15% of adults who have no complaints of heel pain present with a bone spur. Gulick et al.[19], demonstrated no correlation between a patient's pain report on the Visual Analog Scale (VAS) and bone spur size or density. Most authors agree that there is no real correlation between the presence of a calcaneal bone spur and plantar fasciitis/heel pain.[2-4, 7, 8, 20]

Indications for Treatment:

Heel pain
Arch pain
Pain in plantar fascia insertion
Pain with first few steps in the morning or after sitting for an extended period of time

Contraindications / Precautions for Treatment:

See appropriate treatment/modality procedures

Evaluation:

Medical History: Review medical history questionnaire and medical history reported in computer system. Review any diagnostic imaging, tests or work up listed under LMR.

History of Present Illness: Interview patient at the time of examination to review patient's history and any relevant information that would pertain. If the patient is unable to give a full history, then interview the patient's legal guardian or custodian. Determine any past treatments that have taken place. Some examples with plantar fasciitis could be sudden onset of heel pain, pain worse in the AM or upon standing after sitting extended period of time, significant increase in weight-bearing activity. Ask if there has been any significant weight gain.

Social History: Review patient's home, work, recreational and social situation. Areas to focus on would be any weight-bearing activity, excessive walking, running or standing, carrying loads.

Medications: Typically NSAIDS for pain/inflammation control

Examination (Physical / Cognitive / applicable tests and measures / other)
This section is intended to capture the most commonly used assessment tools for this case type/diagnosis. It is not intended to be either inclusive or exclusive of assessment tools.

Pain: as measured on the VAS, activities that increase symptoms, decrease symptoms, location of symptoms.

Palpation: Palpate entire foot/arch. Focus on medial insertion of plantar fascia.

ROM: Ankle Dorsiflexion/Plantarflexion/Inversion/Eversion, Toe Flexion/Extension, Knee Flexion/Extension, And Hip Flexion/Extension/Abduction/Internal Rotation/External Rotation. Focus on gastrocnemius/soleus length.

Strength: Ankle DorsiFlexion/PlantarFlexion/Inversion/Eversion, Toe Flexion/Extension, Knee Flexion/Extension, And Hip Flexion/Extension/Abduction/Internal Rotation/External Rotation

Sensation: If abnormal as found via dermatomal screen or if diabetic, use Semmes-Weinstein assessment.

Posture/alignment: Primary focus on static foot posture. Tend to be at extremes of pes planus/cavus. Secondary exam may include assessment in subtalar neutral.

Gait: Focus on dynamic foot posture with and without footwear. Tend to either over or under pronate during stance and through toe off phases of gait. May also assess running on treadmill if appropriate

Balance: Single leg stance test, Star Excursion Test

Footwear: Assess type and wearing patterns of footwear, use of orthotics.

Differential Diagnosis (if applicable): Stress fracture, posterior tibial tendonitis, sciatica, metetarsalgia, cancer, RA, calcaneal fracture, peripheral neuropathy, infection. Bilateral symptoms can occur in 20-30% of those diagnosed with plantar fasciitis. However in these cases it is important to rule out other systemic processes such as rheumatoid arthritis, systemic lupus erythematosus, Reiter's disease, gout and ankylosing spondylitis.

Assessment:

Establish Diagnosis and Need for Skilled Services

Problem List (Identify Impairment(s) and/ or dysfunction(s))

1. Pain
2. Decreased ROM
3. Decreased Strength
4. Decreased Balance
5. Decreased Function
6. Decreased Foot Biomechanics

Prognosis: Good with patient adherent to stretching program, and use of biomechanical devices. If chronic, may need to resort to other treatment procedures such as injection, extra-corporial shock wave therapy (ECST), plantarfasciotomy if conservative treatment fails. According to the literature, approximately 80-90% of people suffering from plantar fasciitis will have a complete resolution of their symptoms in 6-18 months, with or

without treatment.[1, 2, 7, 15, 21] Conservative treatments include non-steroidal anti-inflammatories (NSAIDs), orthotics, heel cups/cushions, night splints, Achilles tendon stretching and physical therapy treatment (including exercise and modalities such as ultrasound, phonophoresis, iontophoresis and friction massage). All of these interventions have demonstrated some positive effect in the outcome of plantar fasciitis, [1-8, 13-15, 19-24] however there is no consensus as to which modality or combination of modalities is the most effective.[15, 21] In their systematic review of literature for the Cochrane Collaborative, Crawford et al.[21] concluded there was limited evidence that any of the conservative treatments were any more effective than no treatment at all. The authors determined the principle reason for this finding was a lack of good studies. Another thought that several authors had was in the multifactorial etiology of plantar fasciitis. Many factors, both environmental and anatomical, may predispose a person to plantar fasciitis but determining which factors are present may be the key to deciding the best form of treatment.[12, 13] Gross et al.[14], in their review of the literature, concluded that it was difficult to evaluate the effectiveness of any specific intervention because of the multiple interventions used in and between studies.

Goals (Measurable parameters and specific timelines to be included on eval form)

1. Decrease Pain
2. Increase ROM
3. Increase Strength
4. Increase Balance
5. Increase Function
6. Correct Foot Biomechanics

Treatment Planning / Interventions

Established Pathway	___ Yes, see attached.	_X_ No
Established Protocol	___ Yes, see attached.	_X_ No

Interventions most commonly used for this case type/diagnosis.

This section is intended to capture the most commonly used interventions for this case type/diagnosis. It is not intended to be either inclusive or exclusive of appropriate interventions.

1. Stretching
2. Strengthening
3. Foot Orthotics
4. Ultrasound
5. Phonophoresis
6. Iontophoresis
7. Night Splints
8. Ice

Frequency & Duration: Typically patients are seen 1-2x/wk for 4-6 weeks

Patient / family education:

1. Instruction in home exercise program
2. Instruction in pain control and ways to minimize inflammation
3. Instruction in activity level modification

Recommendations and referrals to other providers.

1. Orthotist
2. Podiatrist
3. Orthopod

Re-evaluation / assessment

Standard Time Frame- 30 days or less if appropriate

Other Possible Triggers- A significant change in signs and symptoms, new orthotics may trigger a gait assessment, change in medication for iontophoresis.

Discharge Planning

Commonly expected outcomes at discharge:

Although it could take upwards of 6 months, patients should return to previous level of activity without significant complaints of pain. Patients should be independent with a HEP and have a good knowledge of how to control their symptoms if they return. They will also demonstrate a good knowledge of the etiology of plantar fasciitis and ways to help prevent the symptoms from returning.

Transfer of Care (if applicable)

Referral back to referring physician if symptoms do not change

Patient's discharge instructions:

Continue home exercise program. If symptoms return, call clinic or MD

Authors:

Joel Fallano, PT
Kari Lawson, PT
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Reviewed by:

Adele Gagne, SLP
Barb Odaka, PT
Reginald Wilcox, PT

Revised:

Joel Fallano, PT
4/07

REFERENCES

1. Tisdel CL, Donley BG, Sferra JJ. Diagnosing and treating plantar fasciitis: A conservative approach to plantar heel pain. *Cleve Clin J Med*. 1999;66:231-235.

2. Singh D, Angel J, Bentley G, Trevino SG. Fortnightly review. plantar fasciitis. *BMJ*. 1997;315:172-175.

3. DeMaio M, Paine R, Mangine RE, Drez D,Jr. Plantar fasciitis. *Orthopedics*. 1993;16:1153-1163.

4. Barrett SJ, O'Malley R. Plantar fasciitis and other causes of heel pain. *Am Fam Physician*. 1999;59:2200-2206.

5. Pyasta RT, Panush RS. Common painful foot syndromes. *Bull Rheum Dis*. 1999;48:1-4.

6. Charles LM. Plantar fasciitis. *Lippincotts Prim Care Pract*. 1999;3:404-407.

7. Young CC, Rutherford DS, Niedfeldt MW. Treatment of plantar fasciitis. *Am Fam Physician*. 2001;63:467-74, 477-8.

8. Schepsis AA, Leach RE, Gorzyca J. Plantar fasciitis. etiology, treatment, surgical results, and review of the literature. *Clin Orthop*. 1991;(266):185-196.

9. Saidoff D, McDonough AL. Medial calcaneal heel pain upon weight bearing in an intrinsic foot deformity. In: *Critical Pathways in Therapeutic Interventions - Extremities and Spines.* 1st ed. St. Louis, MO: Mosby; 2002:319-338.

10. Fredericson M. Common injuries in runners. diagnosis, rehabilitation and prevention. *Sports Med*. 1996;21:49-72.

11. Michaud T. *Foot Orthoses and Other Forms of Conservative Foot Care.* 1st ed. Newton, MA: Thomas C Michaud; 1997.

12. Bedi HS, Love BR. Differences in impulse distribution in patients with plantar fasciitis. *Foot Ankle Int*. 1998;19:153-156.

13. Kibler WB, Goldberg C, Chandler TJ. Functional biomechanical deficits in running athletes with plantar fasciitis. *Am J Sports Med*. 1991;19:66-71.

14. Gross MT, Byers JM, Krafft JL, Lackey EJ, Melton KM. The impact of custom semirigid foot orthotics on pain and disability for individuals with plantar fasciitis. *J Orthop Sports Phys Ther*. 2002;32:149-157.

15. Buchbinder R. Clinical practice. plantar fasciitis. *N Engl J Med*. 2004;350:2159-2166.

16. Khan KM, Cook JL, Taunton JE, Bonar F. Overuse tendinosis, not tendinitis. part 1: A new paradigm for a difficult clinical problem. *Physician Sportsmed*. 2000;28:38-43, 47-8.

17. Sharma P, Maffulli N. Tendon injury and tendinopathy: Healing and repair. *J Bone Joint Surg Am*. 2005;87:187-202.

18. Rolf C. Overuse injuries of the lower extremity in runners. *Scand J Med Sci Sports*. 1995;5:181-190.

19. Gulick DT, Bouton K, Detering K, Racioppi E, Shafferman M. Effects of acetic acid iontophoresis on heel spur reabsorption. *Phys Ther Case Rep*. 2000;3:64-70.

20. Davis PF, Severud E, Baxter DE. Painful heel syndrome: Results of nonoperative treatment. *Foot Ankle Int*. 1994;15:531-535.

21. Crawford F, Thomson C. Interventions for treating plantar heel pain. *Cochrane Database of Systematic Reviews*. 2005;3.

22. Barry LD, Barry AN, Chen Y. A retrospective study of standing gastrocnemius-soleus stretching versus night splinting in the treatment of plantar fasciitis. *J Foot Ankle Surg*. 2002;41:221-227.

23. Gudeman SD, Eisele SA, Heidt RS,Jr, Colosimo AJ, Stroupe AL. Treatment of plantar fasciitis by iontophoresis of 0.4% dexamethasone. A randomized, double-blind, placebo-controlled study. *Am J Sports Med*. 1997;25:312-316.

24. Japour CJ, Vohra R, Vohra PK, Garfunkel L, Chin N. Management of heel pain syndrome with acetic acid iontophoresis. *J Am Podiatr Med Assoc*. 1999;89:251-257.