



Workplace Safety and Insurance
Appeals Tribunal

Tribunal d'appel de la sécurité professionnelle
et de l'assurance contre les accidents du travail

Plantar Fasciitis (Heel Pain)

Discussion paper prepared for

The Workplace Safety and Insurance Appeals Tribunal

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Prepared by:

Timothy Daniels, MD, FRCSC

Division of Orthopaedic Surgery, St. Michael's Hospital, Toronto,
Ontario Assistant Professor, Department of Surgery
University of Toronto

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Addendum added by:

Johnny T. Lau, MD, MSc, FRCSC

Medical Director, Foot and Ankle Specialty Program, Altum Health,
(University Health Network, Toronto Western Hospital)
Assistant Professor, Department of Surgery
University of Toronto

Senior Editor, Marvin Tile CM, MD, BSc (Med), FRCSC

Dr. Timothy R. Daniels graduated from the University of Saskatchewan in 1986. He did post-graduate training in orthopaedics at the Royal University Hospital in Saskatoon from 1987 to 1991. He became a Fellow in orthopaedics in 1991. He went on to do a foot and ankle fellowship at the Emory University School of Medicine in Atlanta, Georgia, from 1992 to 1993 and a research fellowship at Milwaukee County Medical Complex, Wisconsin, in 1993. He was an Associate Professor at Emory University School of Medicine from 1992 to 1993 and joined the faculty at the University of Toronto in 1993. He currently holds the rank of Associate Professor in the Department of Surgery. He is a Reviewer for the Journal of Rheumatology, the

Canadian Journal of Surgery and the Canadian Medical Association Journal (CMAJ) and an Associate Editor for Foot & Ankle International. His clinical and research interests are in orthopaedics and foot/ankle conditions. He has published extensively on these subjects.

Dr. Johnny Lau: After graduating in 1994 in medicine from the University of Toronto, Dr. Johnny Lau completed his orthopaedic training in Toronto and during this time completed a Masters Science Degree with an interest Foot and Ankle Biomechanics. From there, Dr. Lau went to Baltimore, Maryland for a one-year Clinical Fellowship in Reconstructive Foot and Ankle Surgery at Union Memorial Hospital.

Dr. Lau, cross-appointed to the University of Toronto as Assistant Professor in orthopaedic surgery, sub specializes in reconstructive foot and ankle surgery, sports and arthroscopic foot and ankle surgery, foot and ankle arthritis surgery (including ankle replacements and joint transplants), rheumatoid foot and ankle reconstruction and diabetic foot infection and ulcers.

Dr. Lau's research interest is in the treatment of 1st great toe and ankle arthritis. He has developed a total ankle replacement.

Dr. Lau is a past President of the Canadian Orthopaedic Foot and Ankle Society, and actively participates in committees for the American Orthopaedic Foot and Ankle Society. Dr. Lau was Chair of the Ankle Arthritis Working Group for the American Academy of Orthopaedic Surgery, and led an international group of surgeons to develop guidelines for treating ankle arthritis.

Dr. Lau has participated in training medical students, residents, and 45 national and international fellows. He is Program Director of the University of Toronto Foot and Ankle Fellowship program, Divisional Fellowship Director for all University of Toronto Orthopaedic Fellowships, and the co-chair of the University of Toronto Biennial Foot and Ankle Symposium in Toronto.

Dr. Lau is the Orthopaedic Foot and Ankle Consultant for the Toronto Football Club, Toronto Blue Jays, Toronto Raptors, Toronto Maple Leafs, National Hockey League Player's Association, and the David L. MacIntosh Sports Medicine Clinic and Varsity Blues Intercollegiate Athletics Program University of Toronto.

Dr. Marvin Tile graduated from the University of Toronto Medical School in 1957. He did post-graduate training in Orthopaedic Surgery at the University of Toronto from 1958 to 1963, and was awarded the Royal College Fellowship in Surgery (Orthopaedics) in 1963. He was granted the Detweiler Fellowship in 1963 and travelled extensively in Europe, visiting leading orthopaedic centres. He joined the faculty at the University of Toronto in 1966 and holds the rank of Professor (Emeritus) in the Department of Surgery (Orthopaedics).

His clinical and research interests have been in orthopaedic trauma care, and also in the management of arthritis, including hip and knee arthroplasty. He also has a major interest in low back pain.

He has published widely, especially in orthopaedic trauma. He has authored two texts: *Fractures of Pelvis and Acetabulum*, AO Trauma and Thieme, Fourth Edition, 2015 and *Rationale of Operative Fracture Care* with Dr. Joseph Schatzker, Springer-Verlag, 3rd Edition, 2005, now in 6 languages. Since 1966, he has been on the Active Staff in orthopaedic surgery at Sunnybrook Health Sciences Centre, a University of Toronto, fully affiliated hospital. He was Chief of Orthopaedic Surgery at that institution from 1971 to 1985 and Chief Surgeon from 1985 to 1996. He has been elected to many prestigious positions. He was the founding president of the Ontario Orthopaedic Association (1978-1980), Past President of the International Society for the Study of Lumbar Spine (1986- 1987), Past President of the Canadian Orthopaedic Association (1991-1992), and in 1992-1994, Past President of the AO Foundation, Switzerland (devoted to research and education in fracture care, world wide). As well, he was Chair of the Sunnybrook Foundation (1996-2001). An endowed Chair in Orthopaedic surgery has been established in his name at Sunnybrook HSC and the University of Toronto. Dr. Marvin Tile has been a medical counselor in orthopaedics for the Tribunal since 2004. He is a Member of the Order of Canada.

This medical discussion paper will be useful to those seeking general information about the medical issue involved. It is intended to provide a broad and general overview of a medical topic that is frequently considered in Tribunal appeals.

Each medical discussion paper is written by a recognized expert in the field, who has been recommended by the Tribunal's medical counsellors. Each author is asked to present a balanced view of the current medical knowledge on the topic. Discussion papers are not peer reviewed. They are written to be understood by lay individuals.

Discussion papers do not necessarily represent the views of the Tribunal. A vice-chair or panel may consider and rely on the medical information provided in the discussion paper, but the Tribunal is not bound by an opinion expressed in a discussion paper in any particular case. Every Tribunal decision must be based on the facts of the particular appeal. Tribunal adjudicators recognize that it is always open to the parties to an appeal to rely on or to distinguish a medical discussion paper, and to challenge it with alternative evidence: see *Kamara v. Ontario (Workplace Safety and Insurance Appeals Tribunal)* [2009] O.J. No. 2080 (Ont Div Court).

PLANTAR FASCIITIS (HEEL PAIN)

Mechanics: The plantar fascia is a multilayered inelastic fibrous sheet (aponeurosis) composed primarily of Type I collagen. It originates from a bony prominence on the bottom of the heel bone (the medial calcaneal tuberosity) and inserts through several fibrous bands into the plantar plate of the metatarsophalangeal joints, flexor tendon sheaths and the base of the proximal phalanges of the toes. The distal insertion site of the Achilles tendon (heel cord) and the origin of the plantar fascia are close in proximity and the outer layer of the two structures is continuous. Since the plantar fascia originates proximally and inserts distally to the subtalar and midtarsal joints, any functional change in its length can alter the position of the said joints. The plantar fascia is the most important static soft tissue supporter of the medial arch; transecting it in loaded Cadaveric specimens results in a 21% loss of the height of the medial arch [1]. The plantar fascia has an important functional role in the normal gait cycle. In the latter part of the stance phase, the toes dorsiflex. This functionally shortens the plantar fascia causing the calcaneus to rotate inwards (into varus) and the medial arch to elevate (windlass mechanism). The internal rotation of the calcaneus causes a divergence in the joint axis of two of the mid-foot joints (the talonavicular and calcaneocuboid joints), thereby locking the joint complex. The now stable midfoot allows for the smooth transition of forces to occur from the hindfoot to the forefoot. In this manner, the plantar fascia is an important soft tissue stabilizer of the foot with an essential functional role during gait.

Pathophysiology: The bottom surface of the heel (calcaneal fat pad) is made up of elastic adipose-tissue-supported spiral fibrous bands (septa) and is the primary structure that cushions the heel from compressive forces at heel strike [2]. The plantar fascia acts as a tie-rod in the foot truss. The primary forces it is subjected to are tensile. The tension is (greatest) at the origin of the plantar fascia near its insertion at the medial calcaneal tuberosity [3]. This is also the location where the plantar fascia commonly becomes swollen and inflamed. It is theorized that the repetitive tensile forces created by walking or standing cause micro-tears in the fascia, leading to acute and eventual chronic inflammation. If the inflammation affects the entire plantar fascia, it is referred to as a plantar fasciitis; however, if it is isolated to the insertion site of the heel, it is called a heel pain (the more common of the two.) MRI studies have demonstrated an increased thickness of the plantar fascia from 3 mm in a normal heel to 7.4mm in patients with a chronic heel pain [4]. Biopsies of the inflamed area have revealed collagen necrosis (death of some of the fibrous tissue), angiofibroblastic hyperplasia (overgrowth of fibrous tissue and tiny blood vessels), chondroid metaplasia (formation of cartilage tissue) and matrix calcification – all microscopic changes typically seen in areas with chronic inflammation and deficient blood flow. On physical examination, the patient often has tightness of the Achilles tendon – this is consistent with the close

anatomical relationship of the heel cord with the plantar fascia [5]. It is unclear whether a tight heel cord is a precipitating factor or simply associated with the heel pain [6-9].

In a small percentage of patients, the heel pain is the result of both inflammation of the plantar fascia and entrapment of the nerves in the distal tarsal tunnel. The most common nerve involved in this process is the first branch of the lateral plantar nerve sometimes referred to as Baxter's nerve. This is a motor sensory branch which supplies sensory nerves to the periosteum of the calcaneus and motor nerves to the abductor digiti minimi muscle.

Etiology: Heel pain may be caused by plantar fascia rupture, fat pad atrophy, stress fractures of the calcaneus, proximal plantar fasciitis, distal plantar fasciitis, plantar fibromatosis, tendonitis of the flexor hallucis longus, tumor of the calcaneus and nerve entrapment, or injury [5]. The most common cause is inflammation of the proximal portion of the plantar fascia. There is no consensus regarding etiology. Snook and Christman wrote, "it is reasonably certain that a condition which has so many different theories of etiology and treatment does not have valid proof of any one cause" [10]. Factors associated with or aggravating this condition are better defined: age, sex, obesity, seronegative inflammatory disorders and activity levels. The average age is 45 years. Prevalence is twice in females vs. males [5]. There may be a history of elevated stress to the foot as a result of increased activities, prolonged standing or weight gain. Physical activity certainly aggravates and can sometimes precipitate the heel pain [11-14]. A retrospective review of injuries among runners cited plantar fasciitis as one of the five most common injuries [14].

Numerous studies have assessed whether or not abnormal foot mechanics predispose an individual to heel pain [5, 7, 9, 12, 13, 15]. Pes planus [16] and pes cavus were the most common foot deformities assessed. A predominance of either deformity has not been identified. Theoretically, both deformities can cause abnormal stresses on the plantar fascia [3, 16-21] and clinical correlations have been suggested [12]. However, a consistent correlation has not been evidenced by retrospective clinical reviews.

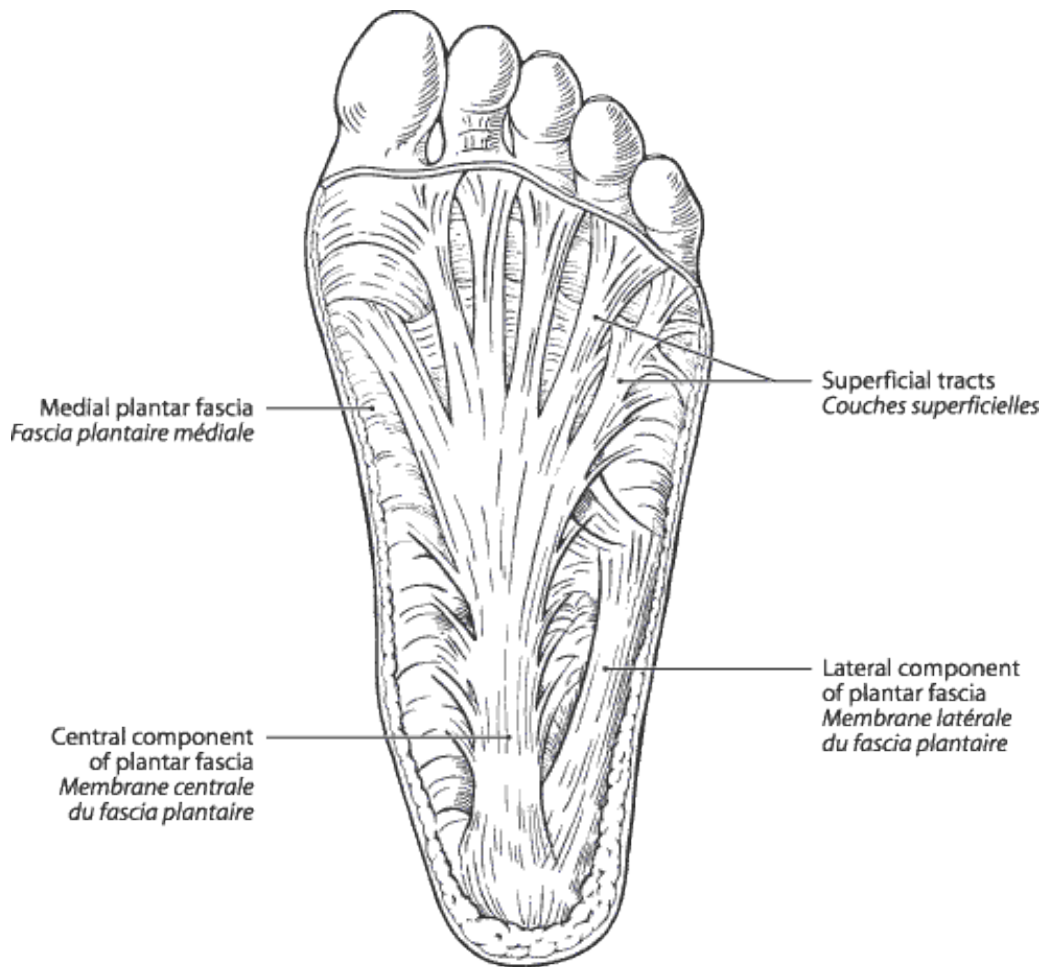
Several studies have implicated body weight as a causative factor [8, 10, 22,23]. Furey [22] identified an increased prevalence of heel pain among obese patients, and Snook and Chrisman [10] noted half of their patients with heel pain were overweight. This makes intuitive sense given that heel pain is considered an over-use syndrome associated with advancing in age. Increased body mass elevates the forces on the aging soft tissues that are less capable

of tolerating the stresses. Weight loss in such patients is often impossible since the painful heel limits their activities.

Factors such as acute injury, the presence of a heel spur, the type of footwear, the walking surface and employment or chronic repetitive activity (other than athletics) have been proposed, but they have not been established as causative [24]. A correlation between prolonged standing or walking has been suggested [8, 9, 25, 26], but direct causation is unsubstantiated since heel pain can occur in all types of patients with varying levels of recreational activities and job requirements. The consistency of the surface on which one walks has not been established as a causative or aggravating factor. This is likely because tension, rather than compression, is the primary force which precipitates injury and inflammation of the plantar fascia. The magnitude of the tensile forces placed through the plantar fascia is related more to the activity level, types of activities, body weight and stability of the medial arch [3, 17-21] rather than the consistency of the walking surface. Although many patients are concerned with the presence of a heel spur, it is not recognized as an etiologic factor. Approximately 50% of patients with a heel pain and 16% without have a heel spur [27]. Anatomically, the heel spur is located at the origin of the flexor digitorum brevis muscle, not at the origin of the plantar fascia. Presumably, the same process occurring at the plantar fascia can occur at the origin of the intrinsic muscles of the foot leading to periostitis and new bone formation. Lapidus et al have demonstrated that successful treatment of heel pain is not contingent with excision of the heel spur [8].

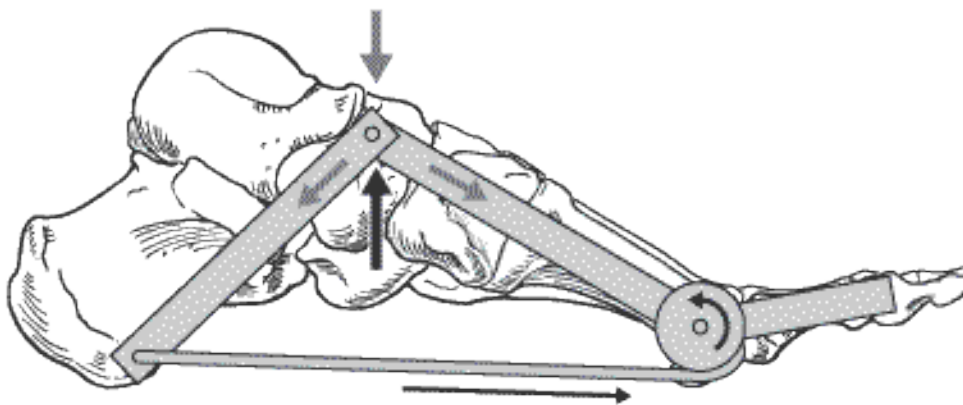
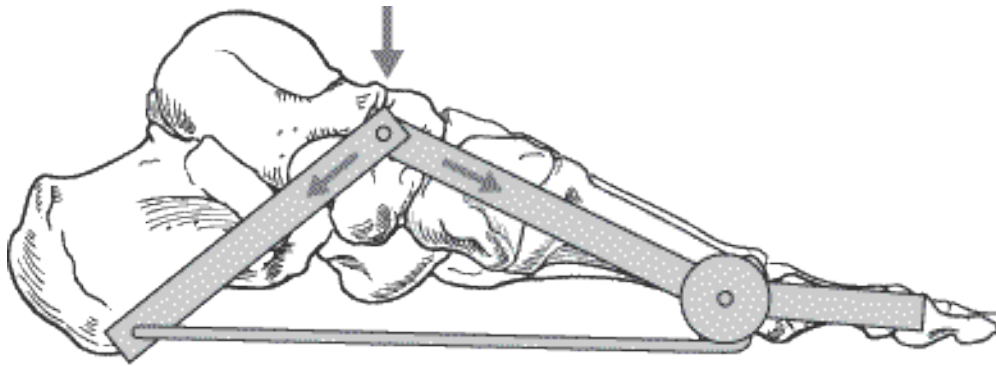
Pain at the sites of tendon or ligament insertion (enthesopathy) is often observed in patients with seronegative arthropathies [5]. Clinical syndromes such as ankylosing spondylitis, psoriatic arthritis, Reiter's syndrome, inflammatory bowel disease and Behcet's syndrome must be ruled out. Associated complaints such as skin lesions, conjunctivitis, arthritis, back pain or abdominal complaints should prompt a referral to a rheumatologist. Screening tests include antigen-B27 (HLA-B27) and sedimentation rate (ESR) can aid in the diagnosis.

Conclusion: The plantar fascia is an inelastic structure that plays an important role in maintaining the stability of the medial arch and midtarsal joints. The primary force placed through the plantar fascia is tension, not compression. It functions as a tie-rod in the foot truss and, during the gait cycle, is under maximal tension in the mid-portion and terminal portions of the stance phase. It is generally accepted that the primary etiology of heel pain is the result of repetitive tensile forces placed through aging tissue that is no longer capable of tolerating the stresses. While associated factors for this condition have been identified, no one activity is known to be the cause of heel pain.



The plantar fascia is a multilayered fibrous aponeurosis
Le fascia plantaire est une aponévrose formée de plusieurs épaisseurs de membrane fibreuse

Figure 1-The plantar fascia is a multilayered fibrous aponeurosis



The windlass mechanism puts tension on the plantar fascia and raises the arch passively.
Le mécanisme de treuil applique une tension sur le fascia plantaire et soulève la voûte plantaire de façon passive.

Figure 2-The windlass mechanism puts tension on the plantar fascia and raises the arch passively

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Timothy Daniels, MD, FRCSC

PLANTAR FASCIITIS (HEEL PAIN) ADDENDUM:

by Johnny T. Lau, MD, MSc, FRCSC
Senior Editor, Marvin Tile CM, MD, BSc (Med), FRCSC

Introduction

The 2003 discussion paper prepared by Dr. Tim Daniels for the Workplace Safety and Insurance Appeals Tribunal (“Tribunal”) provides a thorough overview of the mechanics of the plantar fascia and pathophysiology of plantar fasciitis, as well as a review of the etiology of this condition. This addendum provides a focused overview of the etiologic causes and risk factors associated with the condition based on more recently published literature and is intended to be read in conjunction with Dr. Daniels’ 2003 paper.

Etiology

Multiple studies have failed to identify a consistent causative factor leading to plantar fasciitis. As such, it is thought that the etiology of the condition may be multifactorial [5]. However, it is important to recognize that the majority of the best evidence on the causes of plantar fasciitis are based on case-control studies. These studies are limited in showing causation, and at best, they can only show an association between predisposing factor(s) and plantar fasciitis. Randomized control trials (RCTs) are the gold standard to show causation, but to the best of our knowledge, no RCTs related to identifying etiologic causes of plantar fasciitis have been published. The lack of high level RCTs may explain why numerous risk factors for plantar fasciitis have been identified.

Risk Factors

Patient factors

Older patients have been shown to be more likely to develop plantar fasciitis. In the study by Rano et al. the mean age of patients in the heel pain group was significantly higher than those in the control group (47.5 ± 1.4 years versus 38.4 ± 2.2 years) [7]. Similarly, a study of physically-active military personnel found that those older than 40 years of age had an almost three times increased incidence of plantar fasciitis compared to those aged 20-24 years old [13].

Females have also been shown to be more likely to have plantar heel pain compared to their male counterparts [7]. Reb et al. reported that of the 3,315 patients with plantar fasciitis, 61.2% were female[8]. Similarly, Scher et al. found that incidence of plantar fasciitis in female military personnel was almost twice that of males [13]. However, the study of automobile assembly plant workers

did not show an association between female sex and incidence of plantar fasciitis [14].

Obesity has been shown to be a significant risk factor for plantar fasciitis in multiple studies. In the studies by Irving et al. and by Riddle et al., patients with plantar fasciitis/chronic plantar heel pain were age and sex-matched to control asymptomatic patients [2, 10]. Statistical analysis and comparison of the two groups showed that increased body mass index (BMI) was associated with an increased risk for plantar fasciitis. Other studies have also found that those with plantar fasciitis had higher BMIs than those who did not [6-8]. While these studies have shown that BMI is an increased risk factor for plantar fasciitis, the 2004 study by Riddle et al. also showed that BMI is a predictor of the degree of functional loss and disability reported by patients with plantar fasciitis [11]. It is important to recognize that in these previously reported studies associating BMI and plantar fasciitis, the patient population was predominately sedentary. In a study of athletic individuals by Rome et al., the authors found no associated risk between BMI and plantar heel pain [12].

Foot mechanics

Some studies have tried to determine if abnormal foot mechanics and alignment could predispose individuals to plantar fasciitis. In the study by Irving et al., patients with chronic calcaneal heel pain were compared to age and sex-matched asymptomatic control patients [2]. The authors found that patients with plantar fasciitis were more likely to have a pronated foot compared to control patients. Similarly, a study of automotive assembly line workers also found that those with forefoot pronation were associated with a fivefold increased risk of presenting with plantar fasciitis [14]. However, a study of regular runners found no association between foot pronation and plantar heel pain [12]. Ribeiro et al. also found that there were no differences in hindfoot (rearfoot) alignment in runners with symptoms of or previous history of plantar fasciitis compared to asymptomatic control runners [9]. Interestingly, this study did find runners with plantar fasciitis had elevated medial foot arches compared to control runners.

With regards to ankle motion, the study by Riddle et al. reported that patients with less passive ankle dorsiflexion had higher risk of plantar fasciitis [10]. However, other studies failed to show that decreased ankle range of motion increased the likelihood of plantar fasciitis [2, 12].

Activity or work-related factors

The question of whether prolonged activity or work could be related to the occurrence of plantar fasciitis has been examined by numerous studies. Riddle et al. published two articles in 2003 and 2004, [10, 11]. The first, a case-control study showed “that the risk of plantar fasciitis increases as the range of dorsiflexion decreases. Individuals who spend the majority of their workday

on their feet and those whose body-mass index is $<30 \text{ kg. /m}^2$ are also at an increased risk for the development of plantar fasciitis.” As noted previously in the section on etiology, it is important to recognize that the majority of the best evidence on the causes of plantar fasciitis are based on case-control studies. These studies are limited in showing causation, and at best, they can only show an association between predisposing factor(s) and plantar fasciitis.

The 2004 paper [11] refers to functional disability not causality and concludes: “we identified only one variable that is associated with the extent of disability in patients with plantar fasciitis. A patient’s BMI (obesity) appears not only to increase the risk of plantar fasciitis but also to the extent of the patient’s disability.”

The study by Werner et al. of automotive assembly line workers was able to better associate the degree of activity to the risk of plantar fasciitis [14]. Specifically, the authors found that workers who spent an additional 10% of time standing or walking had a 52% increased risk of presenting with plantar fasciitis. It was further shown that in workers who spent an additional 10% on hard surfaces (such as concrete, asphalt, or linoleum tile on concrete), there was a 30% increase of plantar fasciitis. Finally, when truck drivers and fork lift operators were studied, they found that for each additional 10 times exiting their vehicle, there was a 50% increased incidence of plantar fasciitis.

Radiographic factors

A number of studies have tried to determine the relationship between heel spurs and plantar fasciitis. Kuyucu et al. found that (calcaneal) heel spur length was significantly correlated with perceived pain and disability by patients [4]. The study by Zhou et al. also found that the location of the heel spur was important to associated pain. In particular, they found that calcaneal spurs superior to the plantar fascia heel origin resulted in significantly less pain scores compared to patients with heel spurs that stretched forward from the plantar fascia origin to extend distally within the plantar fascia [15]. However, the study by Ahmad et al. found that neither heel spur shape or size significantly correlated with pain and functional limitations [1].

Symptom duration

In the study by Klein et al., which reviewed 182 patients diagnosed with plantar fasciitis, the authors compared patients with symptoms less than 6 months (acute plantar fasciitis) to those with symptoms greater than 6 months (chronic plantar fasciitis) [3]. The study found that pain intensity and functional limitations did not differ between the two groups. As well, no specific risk factors were identified in developing chronic symptoms. The authors ultimately concluded that chronic plantar fasciitis (i.e. extending beyond 6 months) was not associated with worsening symptoms or disability.

Biological Factors

In the 2003 discussion Paper Dr. Daniels discusses the possible role of biologic factors in the etiology of heel pain/plantar fasciitis. Pain at the insertion of tendon or fascia into bone is common and is called Enthesopathy. It occurs commonly at the shoulder (rotator cuff), the elbow (tennis elbow), the Achilles Tendon (Achilles's Tendonitis) and in many other locations. Heel pain is commonly associated with seronegative arthropathies, a family of inflammatory arthritic diseases including ankylosing spondylitis, Reiter's Disease, Psoriatic Arthropathy and Arthritis associated with inflammatory bowel disease.

A recent study by Hoffman, Nazarian and Smith, included ultrasound and anatomic dissection of the plantar fascia [16].

In the study they conclude that Enthesopathy is an important cause of pain, specifically at the base of the fifth metatarsal. Other studies also show this as an important cause at the insertion of the fascia to the os calcis and relate this to the thickness of the plantar fat pad [17].

Consensus Statement

Schneider, Baca, et al, representing the American College of Foot and Ankle Surgeons recently published a Consensus Statement: Diagnosis and Treatment of Adult Acquired Infracalcaneal Heel Pain in the Journal of Foot and Ankle Surgery [18]. The six member panel used all the best available evidence and their vast clinical experience.

In attempting to reach consensus, the authors state that the information is not evidence based, but largely case control or anecdotal. Therefore, they could not say whether the observations were *causative* or *rather the result* of plantar fasciitis. They found the two distinct patient populations, (1) runners/athletes) and (2) sedentary individuals with a high BMI had different biomechanical factors and by inference, different biologic factors as well.

Summary

More recent studies have confirmed the clinical importance of Heel Pain/ Plantar fasciitis in clinical practice. The etiology of plantar fasciitis is likely to be multifactorial, as both intrinsic and extrinsic risk factors have been reported. To-date numerous studies have examined various intrinsic and extrinsic risk factors implicated in the etiology of plantar fasciitis; in general the factors can be divided into Biological (Enthesopathy, seronegative arthritis) or biomechanical (high BMI, foot pronation, short heel cord, activity level (runners) and many more). Clearly, the interaction of these many factors is different in each individual; and as always in medicine, each case must be assessed and judged individually [19].

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