Plantar Fasciitis: Are Pain and Fascial Thickness Associated With Arch Shape and Loading?
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Background and Purpose
Although plantar fascial thickening is a sonographic criterion for the diagnosis of plantar fasciitis, the effect of local loading and structural factors on fascial morphology are unknown. The purposes of this study were to compare sonographic measures of fascial thickness and radiographic measures of arch shape and regional loading of the foot during gait in individuals with and without unilateral plantar fasciitis and to investigate potential relationships between these loading and structural factors and the morphology of the plantar fascia in individuals with and without heel pain.

Subjects
The participants were 10 subjects with unilateral plantar fasciitis and 10 matched asymptomatic controls.

Methods
Heel pain on weight bearing was measured by a visual analog scale. Fascial thickness and static arch angle were determined from bilateral sagittal sonograms and weight-bearing lateral foot roentgenograms. Regional plantar loading was estimated from a pressure plate.

Results
On average, the plantar fascia of the symptomatic limb was thicker than the plantar fascia of the asymptomatic limb (6.1 ± 1.4 mm versus 4.2 ± 0.5 mm), which, in turn, was thicker than the fascia of the matched control limbs (3.4 ± 0.5 mm and 3.5 ± 0.6 mm). Pain was correlated with fascial thickness, arch angle, and midfoot loading in the symptomatic foot. Fascial thickness, in turn, was positively correlated with arch angle in symptomatic and asymptomatic feet and with peak regional loading of the midfoot in the symptomatic limb.

Discussion and Conclusion
The findings indicate that fascial thickness and pain in plantar fasciitis are associated with the regional loading and static shape of the arch.
Since the pioneering work of Hicks,1,2 in which tensile forces within the plantar fascia of cadaveric limbs were related to foot structure, the aspect ratio of the medial longitudinal arch (ie, the height-to-length ratio) has commonly been implicated in the development of plantar fasciitis. Low-arched foot structures and foot pronation, in particular, have been suggested to increase tensile load within the plantar fascia, thereby increasing the risk of microdamage.3,4 However, evidence for the role of aberrant arch mechanics in plantar fasciitis is equivocal. Although there is some evidence from radiographic studies that a lower static arch shape is more frequent in individuals with plantar fasciitis than in those without plantar fasciitis,5,6 studies using motion analysis techniques typically have shown negligible differences in foot motion or arch dynamics between subjects with symptoms and pain-free controls.7–9 As a consequence, we have previously questioned the role of arch mechanics in the etiology of plantar fasciitis.7,10 However, the majority of research conducted to date has failed to confirm the clinical diagnosis of plantar fasciitis via diagnostic imaging modalities, despite a well-documented lack of specificity of clinical signs and symptoms in diagnosing plantar fasciitis.11

Although no single imaging technique is comprehensive, sonography provides an inexpensive method for quantifying pathology of the plantar fascia. In particular, thickening of the plantar fascia has become a well-established sonographic criterion for the diagnosis of plantar fasciitis, and a reduction in sagittal thickness has commonly been reported with the resolution of heel pain.12–14 However, recent research involving individuals with diabetes has indicated that the morphology of the plantar fascia also may be related to the regional loading of the foot. In a series of experiments, D’Ambrogi and colleagues15–17 demonstrated that, although individuals with diabetic neuropathy had a thicker plantar fascia, similar to that seen in plantar fasciitis, fascial dimensions were positively correlated with the vertical force beneath the forefoot during walking. The authors speculated that the thickened fascia effectively increased the stiffness of the arch, resulting in greater plantar pressures during gait. Although there also is evidence that plantar fasciitis is associated with altered regional loading of the foot during gait,18,19 the relationship between fascial thickness and plantar loading was evident only in individuals with diabetes and not in control subjects without diabetes. Whether the effect represents a systemic change associated with diabetes, a local change associated with mechanical factors, or their combination is unclear.16

Moreover, it is unknown to what extent, if any, local mechanical factors are related to the morphology of the plantar fascia in individuals with plantar fasciitis. It is particularly important to establish the effect of local mechanical factors on the morphology of the plantar fascia, given that fascial dimensions often are used to monitor the progression of plantar fasciitis.20–22 The aims of the current investigation, therefore, were to compare sonographic measures of fascial thickness and radiographic measures of arch shape and regional loading of the foot during gait in individuals with and without unilateral plantar fasciitis and to investigate potential relationships between these loading and structural factors and the morphology of the plantar fascia in individuals with and without heel pain.

**Materials and Methods**

**Subjects**

Ten subjects (3 male and 7 female) with unilateral plantar heel pain (\(\bar{X} \pm SD\) age=48±12 years, height=1.67±0.09 m, weight=79.3±10.2 kg) and 10 asymptomatic control subjects individually matched for age, sex, and body weight (\(\bar{X} \pm SD\) age=47±12 years, height=1.68±0.11 m, weight=81.6±10.6 kg) participated in the study. Subjects with heel pain had tenderness, localized to the calcaneal insertion of the plantar fascia, which was exacerbated with weight bearing following periods of rest. Subjects were excluded if they had diffuse or bilateral pain, evidence of inflammatory arthropathy,23 or a history of trauma or foot surgery. The mean (\(\bar{X} \pm SD\)) duration of heel pain was 9±6 months. Subjects gave written informed consent prior to participation in the study, in accordance with university research ethics policy.

**Protocol**

Prior to testing, the magnitude of heel pain on return to weight bearing following rest was measured with a 10-cm visual analog pain scale* anchored by the terms “no pain” and “worst pain ever.” Non-weight-bearing sagittal sonograms of the fascial insertion of each foot subsequently were acquired with a variable-frequency 12-5 MHz linear array transducer (HDI 5000†) and coupling gel. Subjects were positioned prone with their ankle in neutral (0° of dorsiflexion and plantar flexion). The sagittal thickness of the proximal insertion of the plantar fascia was measured, to the nearest tenth of a millimeter, at a standard reference point 5 mm from the insertion, at the anterior aspect of the inferior border of the calcaneus (Fig. 1). The bias and limits of agreement for repeated measurements of fascial thickness using this technique are 0.01±0.06 cm.7

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Weight-bearing lateral radiographic projections of both feet were obtained during quiet bipedal stance.\textsuperscript{24} Radiographic images were saved to a personal computer in DICOM (Digital Imaging and Communications in Medicine) format and post-processed using MATLAB software.\textsuperscript{2} As depicted in Fig. 2, landmarks on the inferior surface of the calcaneus and the dorsum of the first metatarsal were manually digitized, and the calcaneal inclination and metatarsal declination angles were derived,\textsuperscript{25} relative to the horizontal, using a calibration grid positioned within the field of view.\textsuperscript{26} The calcaneal-first metatarsal (CMT1) angle, the angle subtended by the calcaneal inclination and metatarsal declination angles,\textsuperscript{25} subsequently was calculated using Euclidean geometry, in which the remaining angle of a triangle (CMT1) is calculated from the 2 known angles.\textsuperscript{7} The root mean square error in determining the CMT1 angle via this method is 0.2 degree, with the limits of agreement for repeated measures of $\pm 0.5$ degree.\textsuperscript{26}

Following a familiarization period, a 23- $\times$ 44-cm EMED-SF pressure platform\textsuperscript{3} with a spatial resolution of 4 sensors per square centimeter was used to collect pressure data at a sampling rate of 50 Hz. The pressure platform provided an opportunity to estimate site-specific or regional forces within the foot.\textsuperscript{27} Subjects completed 3 walking trials for each limb at their preferred pace. Consistency between trials was ensured by monitoring the stance phase duration, which differed by less than 5\% between limbs. Trials were repeated if footsteps did not fall entirely within the boundaries of the pressure platform or if we observed gait adjustments secondary to visual targeting of the platform. Novel software\textsuperscript{4} was used to calculate the peak regional vertical force beneath the rear foot, midfoot, forefoot, and digits using a standardized masking procedure in which the length of the footprint, excluding the toes, was divided into equal thirds.\textsuperscript{19} Peak regional forces have been shown to be more sensitive to gait anomalies associated with plantar fasciitis than those derived from conventional foot-ground reaction force curves.\textsuperscript{19} Peak regional ground reaction forces were normalized to body weight and averaged over the 3 walking trials.

**Data Analysis**

The Statistical Package for the Social Sciences (version 12)\textsuperscript{5} was used for all statistical procedures. Kolmogorov-Smirnov tests were used to evaluate data for underlying assumptions of normality. Because all outcome variables were normally distributed, means and standard deviations were used as summary statistics. Differences in each of the dependent variables of interest (arch shape, fascial thickness, and peak vertical force beneath the rear foot,

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\textsuperscript{2} The MathWorks Inc, 3 Apple Hill Dr, Natick, MA 01760-2048.

\textsuperscript{3} Novel GmbH, Munich, Germany.

\textsuperscript{4} SPSS Inc, 233 S Wacker Dr, Chicago, IL 60606

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**Figure 1.**

The thickness of the proximal insertion of the plantar fascia ($d$) was measured from sagittal sonograms at a standard reference point 5 mm from the anterior, inferior border of the fascial insertion into the calcaneus (C).

**Figure 2.**

Illustration of the calcaneal-first metatarsal (CMT1) angle. The 4 points of reference (+) represent the anterior, inferior aspect of the calcaneal tubercle, the anterior, inferior aspect of the calcaneocuboid joint, and the proximal and distal thirds of the dorsal aspect of the shaft of the first metatarsal.
midfoot, forefoot, and digits) were compared between groups and limbs using a 2-factor analysis of variance. The limbs of the control subjects were individually matched to the symptomatic and asymptomatic limbs of the subjects with plantar fasciitis, giving rise to nominally termed symptomatic (Control S) and asymptomatic (Control A) control limbs. In each case, group (heel pain and control) and limb (symptomatic and asymptomatic) were treated as within-subject factors, with the standard error adjusted for paired observations as outlined previously. Significant group-limb interactions were investigated using paired t tests. Relationships among the magnitude of pain, the sagittal thickness of the plantar fascia, static arch shape, and the average peak regional loading of the foot were investigated using scatter plots and Pearson product-moment correlations. An alpha level of .05 was used for all univariate tests of significance.

Results

There was a significant group × limb interaction in the sagittal thickness of the plantar fascia (F=43.8; df=1,9; P<.05). The plantar fascia of the symptomatic limb (6.1±1.4 mm) was 48% thicker than that of its asymptomatic counterpart (4.2±0.5 mm) and 75% to 79% thicker than the fascia of the matched control limbs (3.4±0.5 mm and 3.5±0.6 mm). Similarly, the plantar fascia of the asymptomatic limb was significantly thicker than that of control limbs.

As demonstrated in Figure 3, there was no significant group × limb interaction in the peak regional loading of the foot. Similarly, there was no significant group × limb interaction in the mean CMT1 angle between the symptomatic limbs (150°±7°) and asymptomatic limbs (126°±7°) of the subjects with heel pain and the matched control limbs (128°±10° and 128°±8°, respectively).

Table 1 demonstrates the relationship between perceived pain on weight bearing and the sagittal thickness of the plantar fascia, arch shape, and regional loading of the symptomatic foot. Significant correlations were noted between the magnitude of pain and fascial thickness (r=.68, P<.05), the magnitude of pain and the CMT1 angle (r=.76, P<.05), and the magnitude of pain and midfoot loading (r=.76, P<.05).

As shown in Table 2, the sagittal thickness of the plantar fascia was positively correlated with the CMT1 angle in the symptomatic feet (r=.89, P<.05) and asymptomatic feet (r=.64, P<.05) of the subjects with heel pain. Fascial thickness in the symptomatic foot also was positively related to the maximum force beneath the midfoot of the symptomatic limb (r=.79, P<.05). No significant correlations were found between the sagittal thickness of the plantar fascia and the arch shape and peak regional loading in the control limbs (Tab. 2).

Peak midfoot force was positively correlated with CMT1 in both the symptomatic (r=.93, P<.001) and asymptomatic (r=.64, P<.048) limbs of the subjects with heel pain but was not correlated in the matched control limbs (r=.281 and .47, respectively).

Discussion

Although sagittal thickening of the plantar fascia has been widely documented in people with plantar fasci-
It is possible that muscular weakness, particularly of the intrinsic foot muscles, may result in a relatively greater internal loading of the plantar fascia and adaptive thickening. Reduced strength of the ankle and digital plantar flexors has been documented in individuals with plantar fasciitis, suggesting that the plantar fascia may play a more pronounced role in arch maintenance. Although such a mechanism would explain the fascial thickening noted in people with diabetic neuropathy, in which intrinsic foot muscle atrophy is common, the potential role of reflex inhibition of musculature secondary to heel pain cannot be discounted.

Similarly, it is equally plausible that plantar fasciitis may be characterized by a systemic or degenerative fascial thickening, comparable to that observed in tendon, which results in a reduced capacity of the plantar fascia to tolerate normal tensile load. In support of this hypothesis, reduced mechanical properties of tendon with degenerative change has been noted in animal models. As such, abnormal shape and movement of the arch would not necessarily be associated with plantar fasciitis, a finding consistent with the majority of research conducted to date. Moreover, degenerative thickening has been hypothesized to proceed asymptatically in humans and would account for the increased fascial dimensions observed in the asymptomatic limb of individuals with heel pain. Prospective studies have indicated that as many as 45% of thickened Achilles tendons progress to develop clinical symptoms within 12 months and that 40% of individuals with unilateral Achilles tendinopathy develop symptoms in the contralateral limb. Although there is anecdotal evidence that plantar fasciitis may progress in a similar manner, the clinical course of plantar fasciitis remains undocumented.

Previous investigators studying the effect of diabetes on the morphology of the plantar fascia have speculated
that thickening of the fascia increased the stiffness of the foot and, as a consequence, resulted in greater load beneath the forefoot during gait. The findings of the current study, however, do not support such a conclusion in plantar fasciitis. Rather, in the symptomatic limb, the sagittal thickness of the plantar fascia was found to be correlated with peak midfoot loading. Although it is possible that greater midfoot loading increases the internal compressive stress at the calcaneal attachment during mid-stance, resulting in pain and adaptive thickening of the plantar fascia, it may equally represent an antalgic gait response in which individuals with heel pain make gait adjustments that specifically avoid dynamic loading of the painful area, as has been reported previously.

Collectively, the findings of the current investigation may suggest that, although abnormal arch shape is not characteristic of plantar fasciitis, arch shape may influence midfoot loading and modify the internal pressure and level of pain at the fascial insertion. However, it is unknown whether the pain associated with plantar fasciitis is influenced primarily by external midfoot load, resulting in localized pressure near the fascial enthesis, or by the tensile stress borne directly by the plantar fascia as a consequence of arch shape.

Similarly, how the greater fascial dimensions may relate to clinical symptoms of heel pain is unclear. In tendinopathy, tendon dimensions have been shown to be positively correlated with both the severity of extracellular matrix disruption and the level of tendon blood flow. Although the role of collagen disruption in tendon pain has been questioned, recent research has shown that pain levels associated with plantar fasciitis are positively correlated with hyperaemia, as determined by power Doppler ultrasonography. Although suggestive that pain may be associated with neovascular ingrowth, as proposed in tendon, positive color flow and hypoechogenicity are neither specific to nor consistent findings in plantar fasciitis and often are reported in asymptomatic limbs. It is likely, therefore, that neovascularization is not the primary cause of pain in people with plantar fasciitis. Although alternative biochemical hypotheses involving neurotransmitters, such as glutamate and substance P, have been implicated in tendinopathy and fascia, the significance of these factors in plantar fasciitis remains unknown.

As with all research, this study had a number of limitations. Chiefly, it should be remembered that this study evaluated the relationship among pain, fascial thickness, arch shape, and regional loading of the foot at the univariate level and, as such, cannot account for potential collinearity among variables. Given the positive correlations found in the current study among arch shape, midfoot loading, and fascial thickness, it is unknown which, if any, of these variables is independently associated with heel pain. Moreover, in light of the cross-sectional nature of the current study, conclusions regarding cause and effect cannot be made. Thus, it is unknown whether arch shape and midfoot loading contribute to the development of plantar fasciitis or whether heel pain influences the shape and loading of the foot during gait. Although the former offers a therapeutic window for mechanical interventions, such as insoles, taping, and arch supports, the latter would imply inherent limitations to such an approach. We recommend, therefore, that future studies use a prospective study design in which a multivariate modeling approach is used to estimate the respective roles of fascial thickness, arch shape, and regional loading in the development of heel pain.

Summary

The findings of the current investigation suggest that the severity of pain and fascial thickness associated with plantar fasciitis are related to both the regional loading and static shape of the arch of the foot. Although the effect is absent in individuals without plantar fasciitis, it is unknown whether these physical characteristics contribute to the development of plantar fasciitis or occur as a result of gait adaptations secondary to heel pain.

All authors provided concept/idea/research design. Dr Wearing, Dr Smeathers, and Dr Urry provided writing. Dr Wearing, Dr Smeathers, and Mr Sullivan provided data collection and analysis. Mr Yates and Dr Urry also provided data collection. Dr Wearing provided project management and subjects. Mr Dubois provided facilities/equipment and institutional liaisons. Dr Smeathers, Mr Sullivan, Mr Yates, Dr Urry, and Mr Dubois provided consultation (including review of manuscript before submission).

Ethical clearance for the project was obtained from the Queensland University of Technology Human Research Ethics Committee (QUT Ref No. 2335H).

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References

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