

Long-Term Prognosis of Plantar Fasciitis

A 5- to 15-Year Follow-up Study of 174 Patients With Ultrasound Examination

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Background: Plantar fasciitis (PF) affects 7% to 10% of the population. The long-term prognosis is unknown.

Purpose: Our study had 4 aims: (1) to assess the long-term prognosis of PF, (2) to evaluate whether baseline characteristics (sex, body mass index, age, smoking status, physical work, exercise-induced symptoms, bilateral heel pain, fascia thickness, and presence of a heel spur) could predict long-term outcomes, (3) to assess the long-term ultrasound (US) development in the fascia, and (4) to assess whether US-guided corticosteroid injections induce atrophy of the heel fat pad.

Study Design: Cohort study; Level of evidence, 3.

Methods: From 2001 to 2011 (baseline), 269 patients were diagnosed with PF based on symptoms and US. At follow-up (2016), all patients were invited to an interview regarding their medical history and for clinical and US re-examinations. Kaplan-Meier survival estimates were used to estimate the long-term prognosis, and a multiple Cox regression analysis was used for the prediction model.

Results: In all, 174 patients (91 women, 83 men) participated in the study. All were interviewed, and 137 underwent a US examination. The mean follow-up was 9.7 years from the onset of symptoms and 8.9 years from baseline. At follow-up, 54% of patients were asymptomatic (mean duration of symptoms, 725 days), and 46% still had symptoms. The risk of having PF was 80.5% after 1 year, 50.0% after 5 years, 45.6% after 10 years, and 44.0% after 15 years from the onset of symptoms. The risk was significantly greater for women ($P < .01$) and patients with bilateral pain ($P < .01$). Fascia thickness decreased significantly in both the asymptomatic and symptomatic groups ($P < .01$) from 6.9 mm and 6.7 mm, respectively, to 4.3 mm in both groups. Fascia thickness ($P = .49$) and presence of a heel spur ($P = .88$) at baseline had no impact on prognosis. At follow-up, fascia thickness and echogenicity had normalized in only 24% of the asymptomatic group. The mean fat pad thickness was 9.0 mm in patients who had received a US-guided corticosteroid injection and 9.4 mm in those who had not been given an injection ($P = .66$).

Conclusion: The risk of having PF in this study was 45.6% at a mean 10 years after the onset of symptoms. The asymptomatic patients had PF for a mean 725 days. The prognosis was significantly worse for women and patients with bilateral pain. Fascia thickness decreased over time regardless of symptoms and had no impact on prognosis, and neither did the presence of a heel spur. Only 24% of asymptomatic patients had a normal fascia on US at long-term follow-up. A US-guided corticosteroid injection did not cause atrophy of the heel fat pad. Our observational study did not allow us to determine the efficacy of different treatment strategies.

Keywords: long-term prognosis; prognosis; plantar fasciitis; heel spur; human; plantar heel pain; ultrasound; heel fat pad; corticosteroid

The plantar fascia is a thickened, fibrous sheet of connective tissue that originates from the medial tubercle of the calcaneus and fans out, attaching to the plantar plates of the metatarsophalangeal joints and caput of the fifth metatarsal bones. It provides support for the longitudinal arch

and serves as a dynamic shock absorber.^{17,28} Plantar fasciitis (PF) is one of the most frequent causes of heel pain.^{14,17} It occurs in 7% to 10% of the population over a lifetime and has a significantly negative impact on foot-specific and general health-related quality of life.^{6,15} PF is often a syndrome resulting from repeated trauma.²⁸ The risk factors are thought to be high body mass index (BMI) or sudden weight gain, excessive running, prolonged standing/walking occupations, work-related weightbearing activities,

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limited ankle dorsiflexion, cavus (high-arched) foot, and pes planus (excessive foot pronation). The scientific evidence for these theories is slight.^{2,12} The diagnosis is primarily based on symptoms (heel pain during walking) and clinical findings (pressure pain over the anteromedial aspect of the inferior heel).^{2,6,12,33} Ultrasound (US) is often used to confirm the diagnosis of PF and to rule out other causes of heel pain.²⁹ US is a noninvasive, widely available, and inexpensive imaging technique for assessing fascia abnormalities.^{1,13,16,25}

Many treatments are available for PF,³¹ but the results are not convincing. Recommendations include reduced activity, stretching, insoles, slowly increased rehabilitation (including strength training) within pain limits, avoidance of barefoot walking, nonsteroidal anti-inflammatory drugs (NSAIDs), shockwave therapy, and injections with a corticosteroid or platelet-rich plasma. Only rehabilitation (eccentric training and slow heavy-strength training) and corticosteroid injections (in the short term) have documented effects.^{26,31} Shockwave therapy and platelet-rich plasma seem in some studies to be effective,²¹ although documentation is modest.³ Earlier studies have shown that up to 49% of the patients with PF are symptomatic 1.5 to 5 years after the onset of symptoms.^{4,5,22,27,35} Despite the frequent occurrence of the condition both inside and outside the sports environment, the long-term prognosis has only been sparsely investigated.

Our study had 4 aims: (1) to assess the long-term prognosis of PF, (2) to evaluate whether baseline characteristics (sex, BMI, age, smoking status, physical work, exercise-induced symptoms, bilateral heel pain, fascia thickness, and presence of a heel spur) could predict long-term outcomes, (3) to assess the long-term US development in the fascia, and (4) to assess whether US-guided corticosteroid injections induce atrophy of the heel fat pad.

METHODS

Study Design and Participants

In the period from April 1, 2001, to December 28, 2011 (“baseline”), 269 patients were diagnosed and treated for PF by the same 2 examiners (U.F. and L.B.) at Stadium Clinic in Aarhus, Denmark. All patients were instructed in rehabilitation according to the website www.sportnetdoc.com and furthermore underwent a variety of treatments as recommended by their physician. The 269 patients were contacted in 2016 (“follow-up”) and invited to participate in this study, and 174 patients were included. All patients gave informed consent before inclusion in the study. They were interviewed and underwent a new clinical evaluation and US examination of the plantar fascia of both

feet at Stadium Clinic or Regional Hospital Silkeborg. An investigator (L.H.) conducted the follow-up examinations and interviews.

Inclusion criteria consisted of documented PF based on symptoms and clinical and US examination findings at baseline. Exclusion criteria were (1) a chronic inflammatory condition such as psoriasis, psoriatic arthritis, spondyloarthropathy, rheumatoid arthritis, or inflammatory bowel disease, among others; and (2) age younger than 18 years at the onset of symptoms.

The Scientific Ethics Committee of Region Midtjylland, Denmark, and the Danish Data Protection Agency approved clearance for the study, which was performed in accordance with the Declaration of Helsinki II and the Oviedo Convention. This article was structured based on the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) reporting guideline.³⁴

Interview

As the first step of the evaluation, a telephone interview was conducted regarding characteristics at the onset of symptoms and at follow-up. The cause and presence of unilateral or bilateral heel pain at the onset of symptoms (as noted on the baseline report) were recorded. At follow-up, the date of the disappearance of symptoms, relapse, and applied treatments and medications were recorded. At both baseline and follow up, information was collected regarding weight, height, physical work (standing >50% of the time), smoking status, and grading of pain on the plantar fascia of both feet at rest, during walking and running, and on pressure on a 0-to-10 numerical rating scale (NRS; higher scores indicate worse outcomes). The patients were classified into 2 groups at follow-up: an asymptomatic group that scored 0 in all the categories of the NRS and a symptomatic group that scored >0 on the NRS in either rest, during walking, during running, or on pressure. If the patient had bilateral heel pain at baseline, the most painful foot at baseline was the one included in the study.

US Evaluation

The transducer was placed over the plantar aspect of the hindfoot to examine the plantar fascia at its origin on the calcaneus (Figure 1A) and then in the arch. Long-axis sonograms were obtained medial to the midline of the plantar surface of the foot (Figure 1B), where the plantar fascia was most well defined. A Toshiba US scanner with a 7.5-Hz probe with a built-in water pillow was used for the US scans in the period from 2001 to 2002, a Hitachi 8500 scanner with a 12- to 14-Hz linear probe with Doppler was used in

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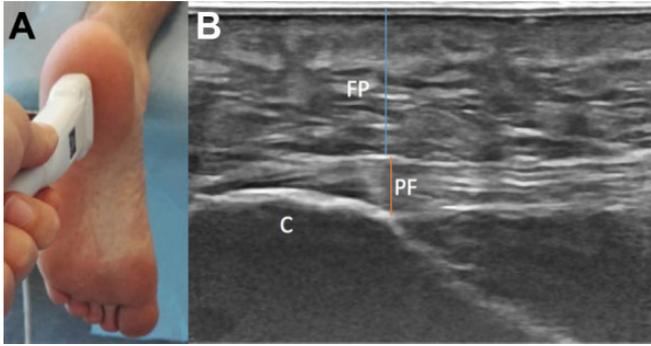


Figure 1. (A) The transducer was placed over the plantar aspect of the hindfoot. (B) Long-axis sonogram of the plantar fascia. C, calcaneus; FP, fat pad (blue line shows thickness); PF, plantar fascia (red line shows thickness).

the period from 2003 to 2011, and a Hitachi Preirus scanner with an 18-Hz linear probe with Doppler and elastography was used starting in 2011. Two examiners (U.F. [rheumatologist] and L.B. [radiologist]), both with more than 20 years of US experience, performed the US at baseline. Another investigator (L.H.) performed the follow-up examinations, 80 (58.4%) of which were re-evaluated in blinded fashion by one of the first examiners (U.F.). An interobserver and intraobserver investigation similar to the study by Rathleff et al²⁵ was performed. The difference in fascia thickness between the US scans (performed by L.H.) was <0.2 mm.

The following were recorded from the US examination at baseline and follow-up: (1) plantar fascia thickness, (2) echogenicity, (3) bony erosions, (4) heel spurs, (5) ossification, and (6) signs of (prior) fascia ruptures. The following were recorded only at follow-up: (7) thickness of the heel fat pad, (8) elastography (grading scale of 1-5), and (9) color Doppler activity (grading scale of 0-4).¹⁹

The plantar fascia thickness was measured longitudinally at the thickest point, either at the origin of the fascia or in the arch of the foot (Figure 2). The heel fat pad was measured vertically at the shortest distance from the superficial border of the fascia to the skin above the calcaneus (Figure 2). At least 3 measurements on 3 different still pictures were used for each foot, and the average of the measurements for fascia thickness and thickness of the heel fat pad was registered. Echogenicity was subdivided into 3 groups: group 1, normal fascia (isoechoic) with a regular fibrillary appearance and fascia thickness <4 mm (Figure 2A)^{25,30}; group 2, doubtful pathological echogenicity (Figure 2B); and group 3, obviously diseased fascia that was hypoechoic and thickened (>4 mm) and with a irregular or missing fibrillary appearance (Figure 2C).^{17,23}

Elastography (Figure 3) of the plantar fascia was performed by compressing the transducer with force applied on the screen (green square in Figure 3) and was displayed as a color-coded image in which the color represents the relative stiffness in the fascia (red square in Figure 3). Stiffness was categorized from 1 (stiffest) to 5: 1 is blue, 1.5 is >50% blue and <50% green, 2 is 50% blue and 50%

green, 2.5 is <50% blue and >50% green, 3 is green, 3.5 is >50% green and <50% yellow/red, 4 is 50% green and 50% yellow/red, 4.5 is <50% green and >50% yellow/red, and 5 is yellow/red.¹⁸ Color Doppler activity was graded using a 0-to-4 scale (grade 0: negative Doppler activity; grade 1: a single vessel; grade 2: Doppler activity in <25% of the fascia; grade 3: 25%-50% Doppler activity; and grade 4: Doppler activity in >50% of the fascia).¹⁹ Grading was estimated in a 0.5-cm longitudinal part of the fascia with maximal Doppler activity (region of interest), as described by Krogh et al.¹⁹ Doppler settings were with a gain setting just below the noise level and the V-scale set to 350.³²

In the Cox regression analysis, we wanted to evaluate whether baseline characteristics could predict the prognosis. An empirical cutoff value of 6.5 mm was used to see if the thickness of the plantar fascia had a prognostic value (ie, whether patients with a thickness >6.5 mm had a worse prognosis than those with a thickness between 4 mm [our definition of a thickened fascia] and 6.5 mm).

Statistical Analysis

STATA software version 14 (StataCorp) was used for statistical analyses. A Kaplan-Meier survival analysis was used to estimate the long-term prognosis of PF. A multiple Cox regression analysis was used for the prediction model on the baseline characteristics. A mixed-model analysis was used to estimate the development of fascia thickness in the affected feet from baseline to follow-up and for the calculation of heel fat pad thickness and the distribution of elastography between the affected and contralateral feet at follow-up. A Z test was used for a comparison of mixed-model analyses. Multinomial logistic regression was used for the calculation of echogenicity. A *P* value of <.05 was considered statistically significant.

RESULTS

Of the 269 patients, 258 were invited from April 1 to September 1, 2016, to participate in this study (Figure 4). Eleven could not be located, 75 did not respond to our letter, and 3 did not want to participate because of a busy schedule or poor health. In all, 180 patients (69.7%) agreed to participate in this study, and 6 of them were excluded because of age below 18 years at the onset of symptoms or other chronic inflammatory conditions. In all, 174 (67.4%) were enrolled in the study, and a telephone interview was carried out in 172. Of the remaining participants, 1 answered by mail, and 1 only had time for a short interview (including information on medical history and the date on which symptoms disappeared). Table 1 shows the participants' characteristics. The patients varied in age from 20 to 79 years at the onset of symptoms and from 26 to 88 years at follow-up, and BMI ranged from 17.8 to 43.3 kg/m² at both the onset of symptoms and follow-up. The mean follow-up time was 9.7 years (range, 4.7-27.3 years) from the onset of symptoms and 8.9 years from baseline. There were 91 (52%) female and 83 (48%) male patients.

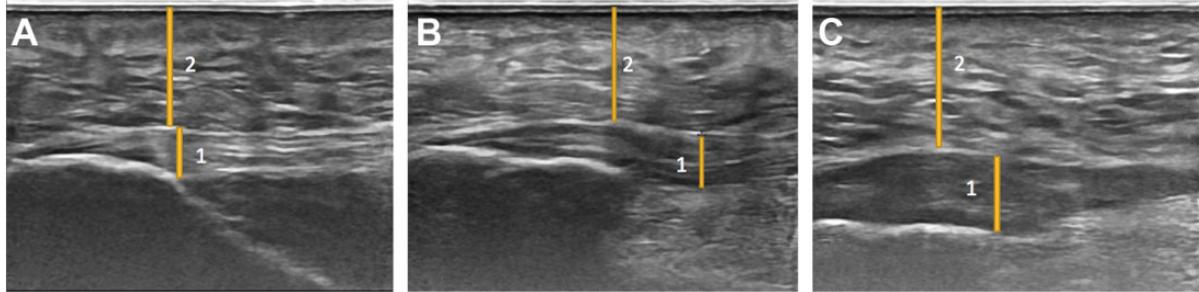


Figure 2. (1) Plantar fascia and (2) heel fat pad measurements on ultrasound. (A) Normal fascia with a regular fibrillar appearance. (B) Doubtful pathological echogenicity. (C) Diseased fascia.

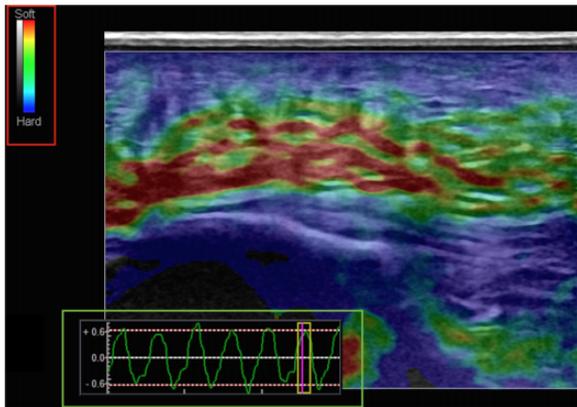


Figure 3. Elastography of the plantar fascia.

On average, the patients had tried 3.8 different treatments (range, 1-9). Overall, 93% of the patients tried US-guided corticosteroid injections, 79% insoles, 66% rehabilitation, 46% NSAIDs, 25% shockwave therapy, 21% acupuncture, 10% laser therapy, 9% US treatment, 2% surgery, and 34% tried other treatments such as massage, fish oil ingestion, and injections with platelet-rich plasma (1%). In the asymptomatic group, 31.9% had at least 1 relapse before they were permanently asymptomatic. During follow-up, 77.5% in the symptomatic group stated they had experienced an asymptomatic period (Table 1).

The follow-up results from the NRS showed that in the symptomatic group, the patients scored, on average, 0.7 (95% CI, -0.7 to 2.0) during rest, 1.8 (95% CI, -0.2 to 4.0) during walking, 2.8 (95% CI, 0.2 to 5.4) during running, and 2.1 (95% CI, -0.1 to 4.2) on pressure.

Long-Term Duration of Symptoms

At follow-up, there were 94 (54%) patients in the asymptomatic group and 80 (46%) in the symptomatic group. In the asymptomatic group, the symptoms of PF had lasted a mean of 725 days (range, 41-4018 days) before symptoms disappeared. The Kaplan-Meier survival function (Figure 5) indicated that the risk of having PF was 80.5% (95% CI, 73.5-85.6) after 1 year, 50.0% (95% CI, 42.4-57.1)

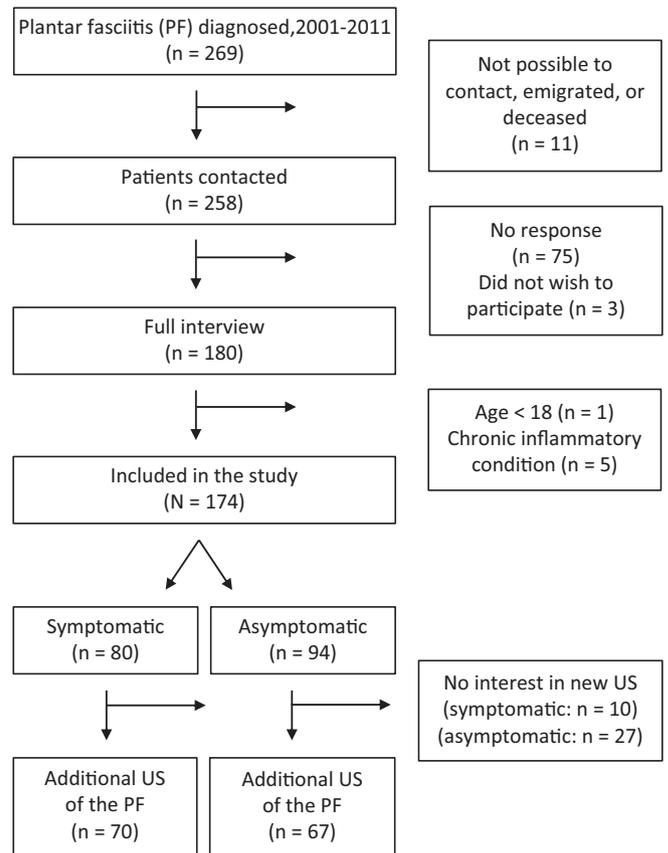


Figure 4. Study flowchart. US, ultrasound.

after 5 years, 45.6% (95% CI, 37.9-53.0) after 10 years, and 44.0% (95% CI, 35.9-51.8) after 15 years.

Cox Regression Analysis for the Prediction of Outcomes Based on Baseline Characteristics

The multiple Cox regression analysis (Table 2) revealed that the hazard rate ratio (HRR; the chance of being asymptomatic per year) for the characteristics at baseline was 1 for men and 0.49 (95% CI, 0.30-0.80; $P < .01$) for women (ie, for every 100 men cured per year, only 49 women were cured per year). The HRR for unilateral heel pain was 1,

TABLE 1
Participant Characteristics^a

	Asymptomatic Group	Symptomatic Group	All
Participants, n (%)	94 (54)	80 (46)	174 (100)
Age, y			
At onset of symptoms	48.5 (21.8-79.4)	44.5 (20.7-67.8)	46.7 (20.7-79.4)
At follow-up	57.7 (34.9-88.8)	54.2 (26.2-76.8)	56.1 (26.2-88.8)
Follow-up time (onset of symptoms to follow-up), y	9.8 (4.9-18.2)	9.6 (4.7-27.3)	9.7 (4.7-27.3)
Female sex, n (%)	42 (44.7)	49 (61.3)	91 (52.3)
Body mass index, kg/m ²			
At onset of symptoms	24.8 (19.2-43.3)	25.0 (17.8-40.5)	24.9 (17.8-43.3)
At follow-up	25.4 (19.4-43.3)	25.5 (17.8-36.3)	25.5 (17.8-43.3)
Physical work, n (%)			
At onset of symptoms	37 (39.4)	43 (53.8)	80 (46.0)
At follow-up	32 (34.0)	29 (36.3)	61 (35.1)
Smoker, n (%)			
At onset of symptoms	23 (24.5)	16 (20.0)	39 (22.4)
At follow-up	0 (0.0)	2 (2.5)	2 (1.1)
Exercise-induced symptoms, n (%)	76 (80.9)	56 (70.0)	132 (75.9)
Time from onset of symptoms to baseline, d	256.5 (4-1932)	360.7 (5-5478)	304.4 (4-5478)
Duration of symptoms, d	724.5 (41-4018)	3623.8 (2212-5035)	—
No. of treatments tried	3.4 (1-8)	4.3 (1-9)	3.8 (1-9)
Relapse, n (%)	30 (31.9)	62 (77.5)	92 (52.9)

^aData are shown as mean (range) unless otherwise specified.

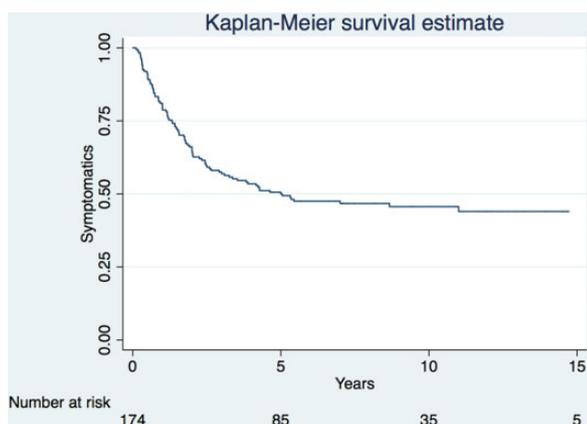


Figure 5. Kaplan-Meier survival function. X-axis: Time in years since the onset of symptoms (year 0). Y-axis: Patients who are symptomatic (1.00 = 100% is symptomatic).

and it was 0.33 (95% CI, 0.15-0.72; *P* < .01) for bilateral pain. The remaining baseline characteristics of BMI, age, smoking status, physical work, time from onset of symptoms to baseline, fascia thickness, and presence of a heel spur were not significant (all *P* values > .05).

Follow-up US Evaluation

At follow-up, a new US examination was performed in 137 patients. Only 108 contralateral feet were used for a comparison, while the remaining 29 were excluded from the comparison because they were symptomatic.

The fascia thickness at baseline was, on average, 6.9 mm (range, 3.8-11.0 mm) in the asymptomatic group and 6.7 mm

TABLE 2
Cox Regression Analysis for Prediction of Outcomes Based on Baseline Characteristics

	Hazard Rate Ratio (95% CI)	<i>P</i> Value
Sex		<.01
Male	1	
Female	0.49 (0.30-0.80)	
Body mass index		.09
≤ 25 kg/m ²	1	
> 25 kg/m ²	0.65 (0.40-1.06)	
Age		.05
≤ 40 y	1	
> 40 y	1.93 (0.99-3.73)	
Smoking status		.64
Nonsmoker	1	
Smoker	0.88 (0.51-1.52)	
Physical work		.24
No	1	
Yes	0.75 (0.46-1.20)	
Heel pain		<.01
Unilateral	1	
Bilateral	0.33 (0.15-0.72)	
Time to ultrasound		.44
≤ 0.5 y	1	
> 0.5 y	1.22 (0.74-2.00)	
Fascia thickness		.49
≤ 6.5 mm	1	
> 6.5 mm	1.20 (0.72-1.98)	
Heel spur		.88
No	1	
Yes	0.96 (0.56-1.63)	

(range, 4.3-12.5 mm) in the symptomatic group (Figure 6). At follow-up, the fascia thickness was reduced to 4.3 mm (95% CI, 3.5-5.1 mm) in the asymptomatic group and

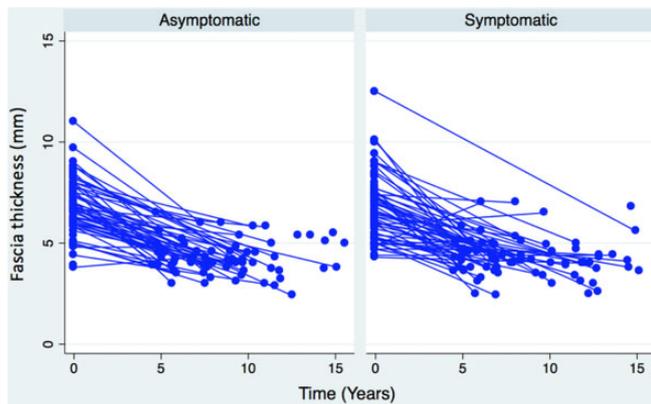


Figure 6. Development of fascia thickness in the affected foot from baseline (time 0) to follow-up in the asymptomatic and symptomatic groups.

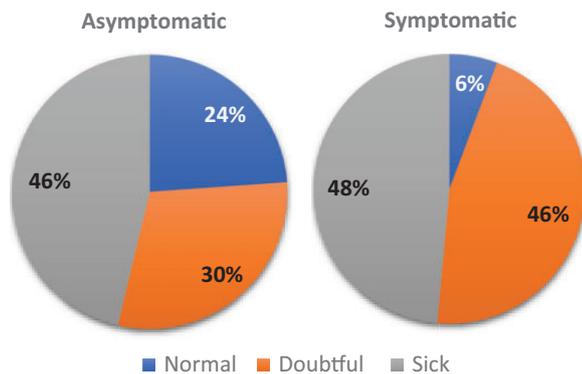


Figure 7. Echogenicity of the plantar fascia in asymptomatic and symptomatic patients at follow-up.

4.3 mm (95% CI, 3.3-5.3 mm) in the symptomatic group (both groups, $P < .01$). There was no statistically significant difference between the 2 groups ($P = .66$). The survival analysis ($P = .36$) and multiple Cox regression analysis ($P = .88$) (Table 2) showed that fascia thickness (≤ 6.5 mm and > 6.5 mm) had no impact on the prognosis. At baseline, 100% had US evidence of fascia abnormalities (see Figure 2). At follow-up (Figure 7), 23.9% (95% CI, 13.7-34.1) in the asymptomatic group had a normal fascia, 29.9% (95% CI, 18.9-40.8) a doubtful fascia, and 46.2% (95% CI, 34.3-58.2) a diseased fascia. In the symptomatic group, 5.7% (95% CI, 0.3-11.1) had a normal fascia, 45.7% (95% CI, 34.0-57.4) a doubtful fascia, and 48.6% (95% CI, 36.9-60.3) a diseased fascia. The odds ratio for finding a normal fascia by US was 4.4 times (95% CI, 1.4-15.4) higher in the asymptomatic group compared with the symptomatic group ($P = .02$). No significant difference was found for doubtful and diseased fascias.

At baseline, 55.5% of patients had a heel spur. A heel spur at baseline did not result in a poorer prognosis ($P = .88$) (Table 2). At follow-up in the asymptomatic group, 64.2% had a heel spur on the affected foot and 41.5% on the contralateral foot. In the symptomatic group, 65.7%

had a heel spur on the affected foot and 34.1% on the contralateral foot.

Elastography was measured in 126 affected feet and 98 contralateral feet. The mean stiffness was 1.6 (95% CI, 1.1-2.1) for the affected foot and 1.5 (95% CI, 1.0-1.9) for the contralateral foot. There was no difference between the affected and contralateral feet ($P = .96$). Neither did we find any difference between symptomatic and asymptomatic patients.

No partial or complete fascia rupture or color Doppler activity was observed in any of the patients.

US-Guided Corticosteroid Injection and the Association With Atrophy of the Heel Fat Pad

The mean fat pad thickness was 9.0 mm (95% CI, 7.0-10.9 mm) in the heels of patients who had received a corticosteroid injection and 9.4 mm (95% CI, 7.2-11.6 mm) in those who had not been given an injection ($P = .66$).

DISCUSSION

The long-term prognosis of PF is unknown, and patients are usually informed that the condition will resolve in 1 to 2 years. In this study, we investigated the long-term prognosis of PF based on the duration of symptoms and US evaluation of the fascia.

The first aim was to assess the long-term prognosis in patients with PF. The results showed that the risk of still having PF after 5 years was 50.0%, 45.6% after 10 years, and 44.0% after 15 years from the onset of symptoms. However, the symptomatic patients at follow-up had minor symptoms (pain level, 2.8 [range, 1-10] on the NRS). The patients with PF in this study belong to a subgroup with the most severe conditions, with a mean duration of symptoms at baseline of 304 days (range, 4-5478 days). In addition, 93% of the patients were treated with a US-guided corticosteroid injection, which is not a first line of treatment, and thus an indication that it was a “difficult-to-treat” cohort. Therefore, the participants investigated in this study are not representative for all patients with PF.

Of the 269 invited patients, 75 (28%) did not respond to our letter and were not included in the study. The patients who did not respond probably had few or no symptoms and therefore had little motivation to participate in the project. Even though many asymptomatic patients joined the study (54%), the prognosis would most likely have been better if the 75 patients who did not respond had joined. However, our results were similar to those of other studies investigating PF prognosis, with 18% to 49% of the patients being symptomatic after a follow-up of 1.5 to 5 years.^{4,5,10,22,35} Further, a study to assess the duration of symptoms in very light cases of PF is difficult to conduct, as this group of patients might never visit a physician because PF disappears quickly. We used the survival curve as our statistical tool to measure what the long-term prognosis was. PF is a condition with periods of improvement and relapse, and the survival curve is probably only a snapshot of reality because some patients will relapse again.

Patients with PF often receive a variety of (and often multiple) treatments. In our study, the patients tried, on average, 3.8 different treatments. Unfortunately, our observational study does not allow us to determine the efficacy of the different treatment strategies.

The majority of the symptomatic patients in this study had minor symptoms at follow-up (level 2-3 on the NRS). However, 5 patients still suffered considerably from the condition and scored >7 during walking on the NRS, and almost all the symptomatic patients did not run anymore because they were afraid of having PF again.

The second study aim was to predict the baseline characteristics that had an impact on the prognosis. A significantly higher risk for long-lasting symptoms was found for women and patients with bilateral heel pain in both the survival analysis and multiple Cox regression analysis (all P values $<.02$). This study is the first to find a higher risk for women. The reason is unknown but could be related to hormonal, physical, shoe wear, or some other factor. Further research is required to better answer the reasons for the higher risk in female patients. Bilateral heel pain was also a risk factor. People predisposed to PF should have the condition bilaterally because both sides are stressed equally. If there is pain in one heel, the other heel compensates, which eventually could result in pain in both heels. However, our study found only a small percentage of patients with bilateral heel pain. Most patients with unilateral pain do have US findings on their contralateral heel. Perhaps patients with bilateral involvement only focus on the more symptomatic side.

Bilateral pain could represent a manifestation of an inflammatory disorder, for example, spondyloarthritis or psoriasis, which is known to increase the risk of enthesopathy. Although none of the patients in this study had a history of an inflammatory arthritic condition and only 2 of 36 with bilateral pain had bony erosions, no patients underwent a physical or serological examination to rule out a diagnosis of inflammatory disease. Therefore, the possibility exists that an element of systemic disease in the present study group could have gone unrecognized. In the study by Furey¹⁰ with 116 patients, 29% were still symptomatic after 5.2 years of follow-up. In that study, 16% had systemic involvement, and most of these patients had bilateral pain.¹⁰ Yet, because the mean follow-up in our study was 9.7 years, we would expect that any systemic disease would become manifested. However, the majority of the patients had unilateral heel pain, which is the most common type.

There was no impact of BMI, age, smoking status, exercise-induced symptoms, physical work, time from onset of symptoms to baseline, fascia thickness, or presence of a heel spur at baseline on the prognosis in our study (all P values $>.05$). Wolgin et al³⁵ conducted a follow-up study of 100 patients with PF and found that 18% were symptomatic after a follow-up of 47 months (range, 24-132 months). In that study, overweight, bilateral pain, and those who had symptoms for a prolonged period (>6 months) before seeking medical attention had a significantly higher risk for persistent pain. None of the patients in that study had been investigated with US or magnetic resonance imaging. The cohort in our study was similar to the patients in

the study by Wolgin et al³⁵ with regard to age, sex, number of patients with bilateral pain, and duration of symptoms before medical attention was sought, but a higher proportion in our study had PF because of sports activities (76% vs 18% in the Wolgin et al study). In contrast to Wolgin et al,³⁵ we did not find any significant correlation with BMI. The reason could be that the cohort in our study was different because 76% were active in sports. The fact that we used the BMI from baseline in the calculation might also be a reason because patients' weight most likely changed over the years.

Our third aim was to assess the US findings in the fascia at follow-up. Fascia thickness of ≤ 6.5 mm or >6.5 mm had no impact on the prognosis ($P = .49$), and most of the patients still had a thickened and hypoechoic fascia at follow-up despite not having symptoms. We also conducted a Kaplan-Meier analysis of fascia thickness <7 and ≥ 7 , <8 and ≥ 8 , and <9 and ≥ 9 mm but were still unable to find a correlation between fascia thickness and PF prognosis. At follow-up, the fascia thickness was reduced significantly in both groups, demonstrating that the fascia approached normal thickness over time whether or not the patients had symptoms. The reason could be uncertainty in the measurements. Yet, because the same examiner (U.F.) measured fascia thickness at both baseline and follow-up and the interobserver (U.F. and L.H.) and intraobserver (L.H.) reliability was 0.5% to 6.7%, it seems unlikely.

The decrease in thickness could also be linked to age or the use of corticosteroid injections. Genc et al¹¹ showed that corticosteroid injections, which 93% of their patients received, reduced fascia thickness; the same result was found by Krogh et al²⁰ in patients with tennis elbow and Fredberg et al⁹ in those with Achilles and patellar tendinopathies. The corticosteroid reduces fascia thickness probably by making the edema disappear by reducing inflammation. However, because the corticosteroid's effect is short, fascia thickness should return to the same pathological thickness after this period if the triggering overload continues.

The reduced fascia thickness could also be a natural development in fascia healing. The "iceberg theory" first described by Fredberg et al⁸ could be an explanation for the lack of correlation between symptoms and US findings. The iceberg theory claims that an asymptomatic fascia undergoes a process involving progressive changes, which can be seen by US. When a given threshold is reached, the fascia will be symptomatic (the tip of the iceberg). During the treatment of a symptomatic fascia, many will be asymptomatic, but the US changes in the fascia will persist for a long time, which increases the risk of it becoming symptomatic again. This explains why 44% of elite soccer players in Denmark had an abnormal Achilles fascia on US and 18% had an abnormal patellar fascia without having symptoms at the start of the season, while a significant number of the players with an abnormal fascia developed symptoms during the season.⁷ It is likely that the same applies to PF.

No difference in elastography was found between the affected and contralateral feet ($P = .96$). A reason for this might be that we looked at chronic and not acute conditions. In addition, no positive color Doppler activity was found,

even though high-end US equipment was used and the scanner was set to maximum sensitivity. In general, Doppler is difficult to use in patients with PF because of the thickness and consistency of the skin and fat pad overlying the fascia. McMillan et al²⁴ also found no Doppler activity in patients with PF.

In the general population, heel spurs are feared because many think that they cause the symptoms. However, we found that a heel spur had no influence on either the development or prognosis of PF ($P = .88$) and that many patients had a heel spur without having symptoms. Karabay et al¹⁷ and Furey¹⁰ reached the same conclusions.

We did not find any correlation with PF symptoms at follow-up and fascia thickness, elastography, color Doppler, or heel spurs. Many of the asymptomatic patients also had an abnormal fascia at follow-up. The use of US at follow-up in patients with PF is of limited value, and clinical signs (NRS) may be a better way to follow the progress. However, it is reasonable to think that the patients with an abnormal fascia have a greater risk of having a relapse and that US is a good tool to diagnose PF when patients have symptoms and to rule out differential diagnoses.

The fourth aim of our study was to evaluate whether US-guided corticosteroid injections result in atrophy of the heel fat pad. A general fear with the use of corticosteroid injections is that they might result in atrophy of the heel fat pad or cause fascia ruptures. The heel fat pad was not reduced when the diseased, injected foot was compared with the contralateral, noninjected healthy foot ($P = .66$), and none of the patients experienced a rupture of the plantar fascia at follow-up. Genc et al¹¹ found similar results.

Limitations

As stated earlier, 28% of the invited patients did not respond to our letter, possibly skewing our results, since people are more motivated to participate in a study when they have symptoms compared to when they are asymptomatic. Recall bias might be a factor in this study because the patients had to remember when their symptoms started 5 to 27 years before. We did, however, seek confirmation of this information in the medical records at baseline. Furthermore, the asymptomatic patients had to recall when their symptoms disappeared. Information bias could also have been a problem because we do not know how many times the patients were treated with the different treatments, for how long they underwent rehabilitation, and many other aspects of their medical history. Also, we did not assess outcomes based on treatments because we had no specific guidelines for the variety of treatments. A randomized controlled trial must be conducted to investigate this.

Three different US machines were used in the study. There could potentially be a small measurement uncertainty between the machines, but it is most unlikely that this would affect the measurements of fascia thickness and heel fat pad thickness. In addition, the examiner (L.H.) was not blinded when performing the US scans with regard to knowing the status of the patient's symptoms. This could potentially influence the interpretation of the US findings.

However, the other examiner (U.F.) was blinded when he assessed the stored images. Finally, we did not include a healthy control group for US comparison. In future trials, this could help to distinguish between US findings that are specific to pathological changes in PF and findings that occur as a natural part of aging.

CONCLUSION

In patients with severe PF, the risk of still having PF was 50.0% after 5 years, 45.6% after 10 years, and 44.0% after 15 years from the onset of symptoms. Female patients and patients with bilateral heel pain had a significantly higher risk of having continuing symptoms. BMI, age, time from the onset of symptoms to baseline, smoking status, exercise-induced symptoms, and physical work had no significant impact on the prognosis in this study. The patients tried, on average, 3.8 different treatments, and a US-guided corticosteroid injection was the most frequently applied treatment (93%).

The US examination revealed that only 24% of the asymptomatic patients had a plantar fascia with a normal appearance. The mean fascia thickness decreased over time regardless of symptoms, and fascia thickness and the presence of a heel spur at baseline had no impact on the prognosis. Color Doppler activity and elastography had no role in the US evaluation of chronic PF. A US-guided corticosteroid injection was not a risk factor for atrophy of the heel fat pad or fascia ruptures.

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