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Dynamic Ultrasound Assessment and Targeted Taping for a Stiffness-Dominant Presentation of Heel Fat Pad Syndrome: A Case Report

Authors' Contribution:
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Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

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Patient: Female, 50-year-old
Final Diagnosis: Heel fat pad syndrome
Symptoms: Heel pain
Clinical Procedure: Athletic tape • ultrasonography
Specialty: Rehabilitation

Objective: Unusual or unexpected effect of treatment

Background: Heel fat pad syndrome (HFPS) is a common cause of plantar heel pain, typically characterized by macro-atrophy of the adipose tissue. However, some clinical cases present with severe pain despite preserved fat pad thickness. This report describes an underrecognized, stiffness-dominant HFPS presentation identified via layer-specific dynamic ultrasound, and details the short-term clinical course following targeted biomechanical taping.

Case Report: A 50-year-old woman presented with chronic, refractory heel pain exacerbated during weight-bearing activities. While dynamic ultrasound revealed preserved macro-chamber thickness compared to the healthy side, it demonstrated significant internal stiffening. The sonographic compressibility index (CI) of the affected macro-chamber was markedly reduced to 25.17%, indicating a profound loss of functional compliance. To address this, a “gathering” functional taping technique was applied with the aim of engaging the windlass mechanism and mechanically decompressing the tissue. This intervention was associated with immediate, significant pain relief and a restoration of the CI to a normal range (33.00%). Following a supplementary 3-week exercise program focusing on ankle mobility, the patient achieved complete functional recovery (AOFAS score: 100).

Conclusions: This case report describes a proposed stiffness-dominant phenotype of HFPS identified in a single patient; its diagnostic relevance remains to be established in larger studies. Reduced functional compressibility can be clinically informative in selected presentations with preserved thickness. Layer-specific dynamic ultrasound can help identify this presentation, and targeted taping warrants consideration as a pragmatic intervention pending broader validation. These findings suggest that functional compressibility assessment can complement conventional thickness-based evaluation in treatment-refractory heel pain.

Keywords: Bandages • Biomechanical Phenomena • Heel • Pain • Ultrasonography

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Introduction

Plantar heel pain is a highly prevalent musculoskeletal problem, often leading to significant functional impairment and accounting for millions of clinical visits annually [1,2]. While plantar fasciitis is the most commonly diagnosed cause, heel fat pad syndrome (HFPS) is a crucial differential diagnosis, primarily attributed to the loss of shock-absorbing elasticity secondary to aging, repeated micro-trauma, or chronic overuse [3]. In a population-based cohort of community-dwelling older adults, HFPS was identified in 4.2% of participants, representing the second most common cause of plantar heel pain after plantar fasciitis (6.9%) [4]; however, the proportion of these cases specifically characterized by mechanical stiffness with persistent pain, as distinct from thickness loss alone, has not been established in the existing literature. The human heel pad is a highly specialized adipose structure designed to attenuate impact forces during the heel-strike phase of the gait cycle [5].

Traditionally, the clinical and radiological diagnostic criterion for HFPS relies predominantly on the identification of macroscopic atrophy, conventionally referenced in a subset of the literature as a heel pad thickness below approximately 12 mm under uncompressed conditions—a threshold that reflects a historically used convention rather than a universally validated diagnostic cutoff—and a structural decline that is often accelerated by aging [6]. However, clinical observations and an ultrasound imaging study suggest that relying solely on static thickness can be diagnostically misleading [7]. A recent investigation utilizing advanced shear wave elastography has demonstrated that the mechanical stiffness (elastic modulus) of the fat pad correlates more strongly with patient-reported pain severity than absolute bulk thickness does [8].

Anatomically, the heel fat pad is structurally segregated into 2 functionally distinct layers by complex fibrous septa: the superficial micro-chamber and the deep macro-chamber [9,10]. The micro-chambers are densely packed with small fat lobules, functioning primarily to resist shear forces and protect the overlying skin. Conversely, the macro-chambers consist of larger fat lobules designed for vertical shock absorption and massive energy dissipation, which is highly dependent on the viscoelastic properties of the septal walls [9,10]. Standard diagnostic imaging often treats the heel pad as a homogeneous structure, overlooking pathological “stiffening” or distinct degenerative changes within these specific layers. It should be acknowledged that this two-layer framework, while clinically useful, is a simplification; the structural organization and degenerative changes of the heel fat pad are likely more complex than can be captured by a single anatomical model, and additional pathological features such as fibrosis, edema, and septal defects have also been qualitatively described [11].

The present report presents a case-based refinement of existing understanding: preserved bulk thickness can coexist with reduced functional compliance. Layer-specific dynamic ultrasound may help identify this presentation in selected patients. We present the clinical course, sonographic findings, and short-term management of this condition using a targeted biomechanical taping approach designed to engage the windlass mechanism. The relationship between preserved thickness, reduced compressibility, and symptom presentation proposed here remains a hypothesis-generating observation derived from a single case that warrants further investigation.

Case Report

A 50-year-old Japanese female homemaker presented with a two-month history of severe, unrelenting right plantar heel pain. The pain was profoundly exacerbated during weight-bearing activities, particularly during the initial heel-strike phase of walking, and was rated as 6/10 on the Numerical Rating Scale (NRS). Regarding her medical history, she had no co-existing diseases and was not taking any regular medications. Regarding health hazards, she had no history of tobacco smoking, alcohol consumption, or substance abuse. Prior interventions, including standard plantar fascia stretching exercises, had yielded no relief.

The objectives of this case report are to demonstrate the diagnostic value of layer-specific dynamic ultrasound for identifying functional stiffness in HFPS and to assess the effect of a biomechanically targeted intervention. Physical examination revealed localized, severe tenderness strictly at the central aspect of the heel pad. The structural integrity of the pad was clinically assessed using the “pinch test,” in which the affected right heel demonstrated marked induration, stiffness, and reduced pliability of the skin and subcutaneous tissue compared to the contralateral healthy side (**Figure 1A, 1B**).

Further biomechanical evaluation revealed limited right ankle dorsiflexion (5°) and significant hamstring tightness (Straight Leg Raise test: 45°, indicating severe muscular tightness without radicular signs). Prior research has identified limited ankle dorsiflexion as a risk factor for excessive mechanical loading on the plantar heel [12]. Furthermore, posterior fascial tightness, particularly in the hamstrings and gastrocnemius, restricts normal ankle kinematics [13,14]. These kinetic chain deficits suggested a compensatory, rigid gait pattern. Initial conservative management consisting of non-thermal ultrasound therapy and intensive dorsiflexion stretching yielded no symptomatic improvement after one week, representing a common clinical challenge in recalcitrant heel pain [15].

To thoroughly investigate the localized structural properties, a detailed layer-specific dynamic ultrasound assessment

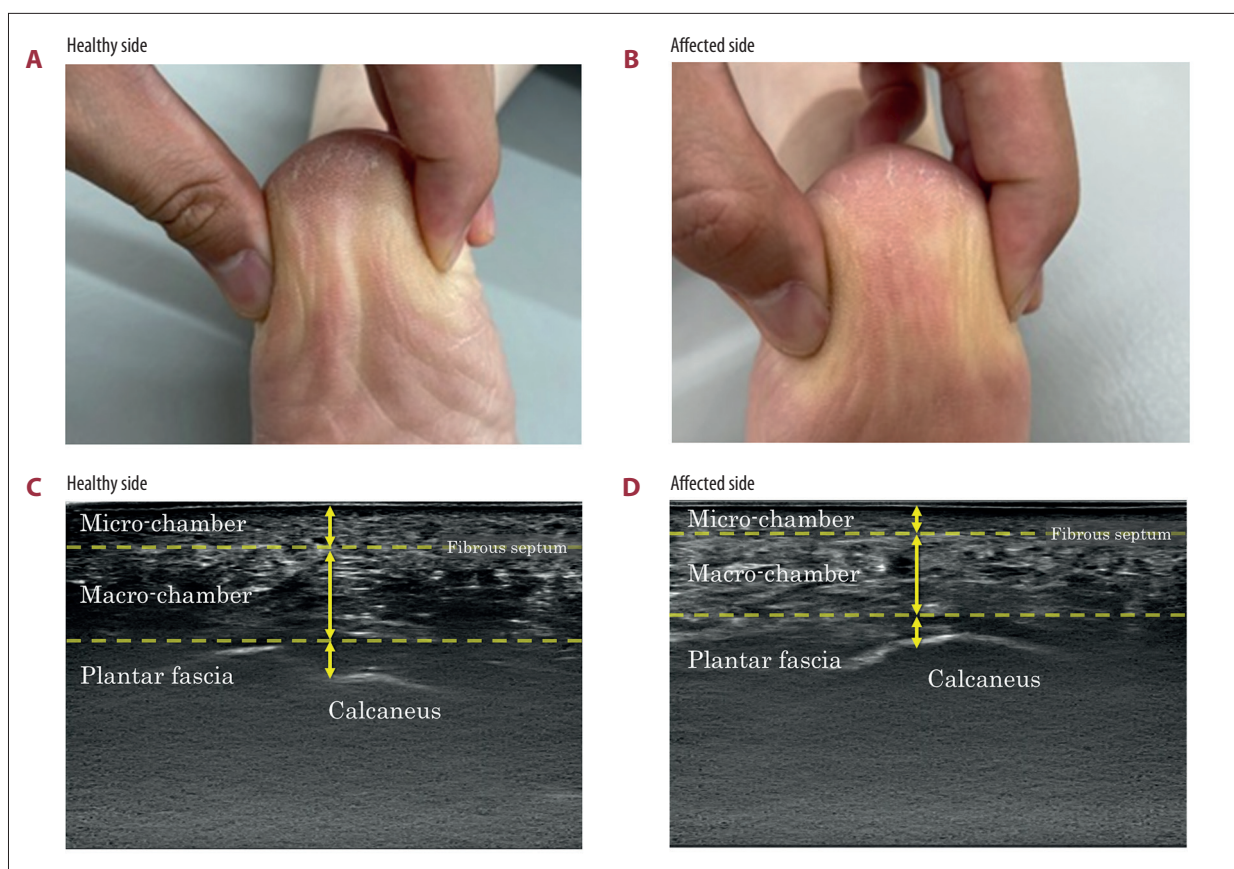


Figure 1. Clinical and sonographic assessment of the heel fat pad. **(A, B)** Clinical Pinch Test: **(A)** The healthy side demonstrates high tissue pliability, allowing the fat pad to be easily pinched. **(B)** The affected side shows marked induration and stiffness, reflecting a profound clinical loss of elasticity. **(C, D)** Longitudinal Sonograms: **(C)** The healthy side reveals a distinct two-layer compartmentalization with relatively hypoechoic micro- and macro-chambers. **(D)** The affected side illustrates the “dual pathology” of the stiff variant; the superficial micro-chamber (indicated by the upper double-headed arrow) exhibits significant focal atrophy, while the deep macro-chamber (lower double-headed arrow) shows diffusely increased echogenicity, suggesting underlying fibrotic degeneration despite preserved bulk thickness.

was performed using a SONIMAGE SNI BLE 2 system (Konica Minolta, Tokyo, Japan) equipped with an L18-4 high-frequency linear transducer (4-18 MHz). The patient was positioned prone with the knee flexed to expose the plantar heel. The probe was positioned longitudinally at the center of the heel pad; the unloaded state was defined as minimal probe contact sufficient to maintain acoustic coupling without tissue deformation, and the loaded state was produced by manual probe compression applied by the same examiner while observing the B-mode image in real time, until no further reduction in tissue thickness was observed, thereby standardizing the degree of compression across measurements without requiring pressure-sensing equipment; this approach was adopted as a pragmatic compression-standardization method feasible in routine clinical settings. To quantify the functional stiffness, the “Compressibility Index” (CI) was utilized and calculated as: $(\text{unloaded thickness} - \text{loaded thickness}) / \text{unloaded thickness} \times 100$. Maximal manual compression was

selected to approximate the loading condition experienced during the stance phase of gait and to capture the maximal compressibility attainable by the tissue; accordingly, the CI is interpreted as reflecting the extent to which the heel fat pad can deform under functional loading. It should be noted that the deformation pattern produced by static probe compression may not fully replicate the complex, multiaxial mechanical behavior of the heel fat pad during dynamic loading in gait, representing an inherent limitation of this functional approximation. The anatomical boundary separating the superficial micro-chamber and the deep macro-chamber was sonographically identified primarily by the change in echotexture between the finely granular superficial fat lobules and the coarser deep fat lobules, supported by a partially visible, horizontally oriented hyperechoic linear structure at the transition zone, consistent with the fibrous septum dividing the 2 compartments [9,10]. Echogenicity, reflecting tissue fibrosis or fluid accumulation, was calculated as the mean gray value

Table 1. Comparison of sonographic parameters between the healthy side, affected side (pre-taping), and affected side (with taping).

Parameter	Healthy side (left)	Affected side (right)	Affected side (with taping)
Superficial micro-chamber			
Thickness (mm)	3.14 ± 0.15	2.45 ± 0.29	2.64 ± 0.17
Echogenicity (0-255)	63.0 ± 1.5	94.1 ± 0.4	87.5 ± 5.2
Compressibility Index (%)	12.15 ± 3.35	7.11 ± 1.21	8.61 ± 2.27
Deep macro-chamber			
Thickness (mm)	5.99 ± 0.11	6.04 ± 0.11	7.28 ± 0.22
Echogenicity (0-255)	80.2 ± 3.2	98.0 ± 1.9	98.7 ± 2.2
Compressibility Index (%)	32.53 ± 5.43	25.17 ± 1.16	33.00 ± 3.23

Values represent the mean ± standard deviation of 3 repeated measurements obtained by the same examiner from a single case. For echogenicity, each of the 3 repeated measurements itself represents the average of values sampled at 3 ROI locations within the compartment. Echogenicity was measured using the mean gray value via ImageJ software (8-bit scale; 0: black, 255: white). *Compressibility Index (CI) = (unloaded thickness - loaded thickness) / unloaded thickness × 100*
 Bold values highlight the increase in thickness and the restoration of compressibility in the macro-chamber following the functional taping intervention.

using an 8-bit grayscale histogram. All thickness and CI values were obtained in triplicate by the same examiner and averaged to derive the reported values. Echogenicity was measured within a rectangular region of interest manually placed over each compartment; the ROI size was not fixed but was adjusted for each measurement to remain within the compartment boundaries, avoiding the fibrous septum and plantar fascia. For each acquisition, echogenicity was sampled at 3 ROI locations within the compartment and averaged to yield a representative value; this acquisition procedure was itself repeated 3 times by the same examiner, consistent with the thickness and CI measurements, and the resulting 3 representative values were averaged to derive the reported mean ± SD. Identical ultrasound gain and depth settings were maintained between the affected and healthy sides. While this two-layer micro-/macro-chamber framework provided a practical basis for the present sonographic assessment, the structural organization of the heel fat pad is likely more complex, and additional architectural changes beyond this simplified model may also contribute to symptom presentation.

Contrary to the typical presentation of gross atrophy in HFPS, the absolute thickness of the deep shock-absorbing macro-chamber on the affected side was remarkably comparable to that of the healthy side (6.04 ± 0.11 mm vs 5.99 ± 0.11 mm). However, layer-specific quantitative analysis revealed a distinct dual pathology (**Table 1, Figure 1C, 1D**). The superficial micro-chamber exhibited significant focal atrophy (2.45 ± 0.29 mm vs 3.14 ± 0.15 mm) and increased echogenicity (94.1 ± 0.4 vs 63.0 ± 1.5). Crucially, the deep macro-chamber exhibited a profound loss of internal mechanical compliance, evidenced

by a significantly reduced CI (25.17 ± 1.16% vs 32.53 ± 5.43% on the healthy side).

Based on this precise biomechanical diagnosis, the treatment strategy was shifted to a targeted clinical intervention. Functional taping was applied using elastic therapeutic tape (3M Kinesiology Taping). To ensure sufficient mechanical fixation force, 2 strips were layered. The tape originated below the sustentaculum tali, wrapped around the calcaneus with moderate tension to centripetally “gather” the fat pad, and extended distally toward the first metatarsophalangeal joint (**Figure 2A**). The tape was applied while passively extending the hallux to intentionally elevate the medial longitudinal arch, with the aim of engaging the windlass mechanism to facilitate load transfer [16]. Dynamic ultrasound immediately post-application confirmed that the taping mechanically reconstructed the macro-chamber, increasing its unloaded thickness to 7.28 ± 0.22 mm and restoring the CI to 33.00 ± 3.23% (**Table 1, Figure 2B**).

The patient was instructed on daily self-application of this specific taping technique. Leveraging this immediate mechanical pain relief, a proactive exercise therapy program was implemented, including forefoot weight-bearing training to promote the co-contraction of the triceps surae and gluteus maximus muscles. The exercise program was performed under direct clinical supervision at each visit and was supplemented by independent home practice; adherence was confirmed at each subsequent visit through patient interview and observed technique. No co-interventions other than taping and exercise therapy were administered during the follow-up period. By the second week of intervention, the pain had completely resolved

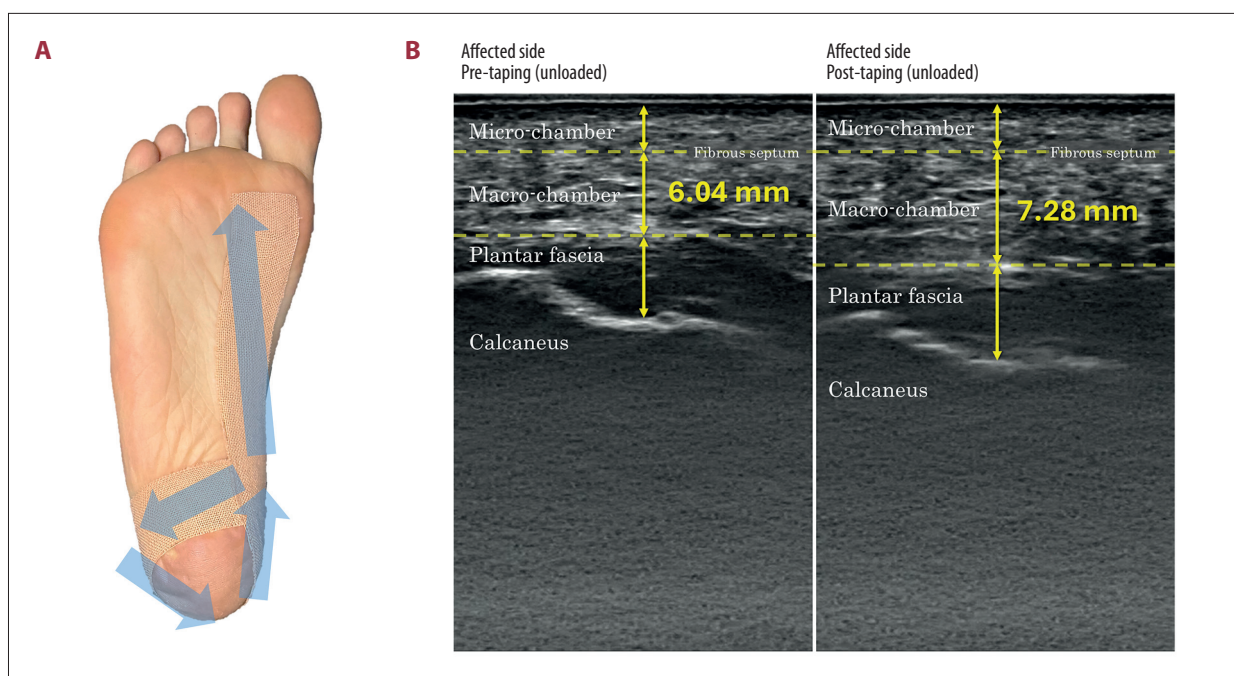


Figure 2. Intervention and immediate structural reconstruction. **(A)** Functional Taping Technique: Double-layered elastic tape is applied to centripetally “gather” the heel fat pad (short white arrows). The tape is extended distally toward the first metatarsophalangeal joint (long white arrow) while the hallux is in passive extension with the aim of engaging the windlass mechanism. **(B)** Sonographic Comparison Pre- and Post-application: The left image shows the unloaded affected heel pad prior to intervention (macro-chamber thickness: 6.04 mm). The right image demonstrates the immediate mechanical decompression following taping; the macro-chamber thickness increased to 7.28 mm, restoring the structural compressibility index to the level of the healthy side.

(NRS 0/10). After 3 weeks, taping was discontinued without recurrence of symptoms. The American Orthopaedic Foot and Ankle Society (AOFAS) Ankle-Hindfoot Scale improved from a baseline of 52 to a maximum score of 100 points, and ankle dorsiflexion improved to 10°. No complications or adverse reactions were reported throughout the treatment period.

Discussion

Precise structural and functional differential diagnosis is critical in managing refractory plantar heel pain. While plantar fasciitis is common, the absence of fascial thickening on ultrasound and the failure of fascia-specific interventions in this case made plantar fasciitis unlikely [17]. Neural etiologies, such as Baxter’s neuropathy and tarsal tunnel syndrome, were considered less likely given the strictly localized central tenderness and the absence of radiating paresthesia [18], although formal electrodiagnostic testing was not performed to definitively exclude a neural contribution. The specific, quantifiable sonographic finding of layer-specific functional stiffness was consistent with a heel fat pad origin of the pathology [19], illustrating a presentation of HFPS defined not by gross atrophy but by internal functional stiffness.

Our analysis suggests a plausible hypothesis regarding a sequential pathological cascade. We postulate that the patient’s kinetic chain deficits, specifically limited ankle dorsiflexion and hamstring tightness, contributed to a high-impact, rigid heel-strike pattern [20]. This chronic, excessive mechanical loading may reflect initial micro-trauma and subsequent fibrotic atrophy of the superficial micro-chamber. If the micro-chamber failed as the primary shear-absorber, the deep macro-chamber may have been forced to bear unattenuated, multidirectional loads. One possible explanation is that this chronic overstress induced secondary fibrosis and septal thickening within the macro-chamber, severely compromising its elastic properties despite preserving its structural dimensions.

The neuroanatomy of the heel provides a physiological basis that could be consistent with the severe pain observed in this presentation. The medial and inferior calcaneal nerve branches penetrate directly through the fibrous septa of the heel pad [21]. In a healthy, compliant heel pad, the honeycomb adipose structure cushions these neural elements during compression. However, as demonstrated in previous investigations, a loss of elasticity within the macro-chamber may impair its force-dissipating function [9,22]. We hypothesize that in our patient, the stiffened, fibrotic septa may have lost their ability

to deform safely, creating pathological traction and localized micro-compression of the penetrating nerve branches [23,24].

The immediate short-term success of our targeted biomechanical intervention is supportive of this neuromechanical hypothesis, although the temporal association is not sufficient to establish definitive causality. The “gathering” taping technique was designed to serve 2 proposed functions: first, we hypothesize that it mechanically decompressed the macro-chamber, potentially restoring its “cushioning stroke” and potentially reducing traction on the entrapped nerve branches; second, by directing the tape toward the first ray with the hallux extended, it was intended to engage the windlass mechanism [25]. This distal anchoring may have facilitated an “early heel-off” phase during the gait cycle, potentially shifting mechanical loading vectors away from the sensitized heel pad; however, as noted above, the specific contribution of this proposed mechanism to the observed pain relief cannot be confirmed from a single case.

Clinicians should be aware that HFPS can manifest with seemingly normal tissue thickness if it is masked by underlying dense fibrosis, a phenomenon biomechanically analogous to the “thick but pathologically stiff” heel pads documented in diabetic populations [26,27]. While taping is often considered a temporary measure with varying reported efficacy [28], our findings suggest its potential value in the immediate, layer-specific restoration of structural compressibility.

Although a small number of prior studies have reported compressibility indices for the heel fat pad, direct numerical comparison with the present case is precluded by substantial methodological heterogeneity. Using loaded-to-unloaded thickness ratios derived from radiographs, a compressibility ratio of 0.69 ± 0.14 has been reported in patients with HFPS versus 0.60 ± 0.11 in healthy controls [29], while a small ultrasound-based case series reported a ratio of 0.60 ± 0.09 [11]. These values are not directly comparable to the percentage-based CI used in the present report, both because of the differing calculation and because the compressive force applied by the ultrasound probe has not been standardized across studies, precluding meaningful cross-study comparison [30]. Physical activity may also have contributed to the observed pain relief: the structured ankle mobility exercise program, introduced after the initial taping-related improvement, may have further reduced symptoms by improving dorsiflexion and correcting the compensatory, high-impact gait pattern identified at baseline; however, as noted above, this potential contribution cannot be isolated from that of taping in the present single-case design.

This study has limitations. Importantly, the boundary conditions of this report must be defined: the observations apply to a single patient with central heel pad tenderness, preserved gross macro-chamber thickness, reduced compressibility on

dynamic ultrasound, and short-term improvement after combined taping and exercise. Because exercise therapy was introduced immediately after the initial taping-related improvement, the relative contribution of taping, exercise, and their combination to the observed short-term recovery cannot be disentangled in this single-case design. The applicability of these findings to broader HFPS populations remains uncertain. Additionally, the compressibility index relies on manual probe compression, which is operator-dependent. The lack of quantitative shear wave elastography and formal 3D kinematic gait analysis limits the absolute confirmation of the tissue’s elastic modulus and dynamic force vectors. Shear wave elastography offers highly reproducible, quantitative mapping of absolute tissue stiffness. Standard B-mode layer-specific dynamic ultrasound, by contrast, provides a pragmatic, widely accessible alternative in typical clinical settings; although it lacks absolute modulus values and relies on operator-dependent manual compression, it allows direct visualization of mechanical compliance and structural behavior under functional loading conditions. Future large-scale cohort studies are warranted to validate the diagnostic criteria for stiffness-dominant HFPS and to establish the long-term efficacy of compressibility-restoring interventions.

Conclusions

This proposed stiffness-dominant presentation of HFPS should currently be regarded as a preliminary clinical observation requiring validation in larger patient cohorts. This case suggests that HFPS can clinically present as functional stiffness even when macroscopic thickness is preserved. We hypothesize that reduced compressibility, quantified via the sonographic compressibility index—an operator-dependent but reproducible measure in this case—may be clinically informative in selected HFPS presentations. To our knowledge, no existing studies have standardized the compressibility index for the heel fat pad in relation to plantar heel pain, precluding direct comparison with the values reported here. Calculating the compressibility index using standard B-mode ultrasound may help identify a stiffness-dominant presentation of HFPS. Targeted taping may warrant consideration as a pragmatic active biomechanical intervention, alongside functional movement correction, aimed at structurally decompressing and offloading sensitized tissues, pending broader validation of this proposed mechanism.

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Institutions Where Work Was Done

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Informed Consent

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

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