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Case Report

Haglund's Deformity in a Patient with Long-Standing Type 2 Diabetes Mellitus: Implications for Tendon Pathology and Surgical Risk

Smit Christian[1*], Marion Yau[2], Sahar Abolhassani[2], Joseph Olivelle[1]

[1]Foot and Ankle Centre, England.

[2]Harley Medical Foot and Nail Laser Clinic London, England.

***Corresponding Author:** Smit Christian, Foot and Ankle Centre, England.

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Abstract

Background: Haglund's deformity is a posterosuperior calcaneal prominence frequently associated with insertional Achilles tendinopathy (IAT). Although traditionally considered a mechanical cause of posterior heel pain, the relationship between deformity size and symptomatic tendinopathy remains debated. Type 2 diabetes mellitus (T2DM) has been associated with increased risk of tendinopathy, likely through metabolic effects on tendon structure.

Case Description: A patient with long-standing T2DM presented with chronic posterior heel pain. Clinical and radiographic assessment revealed Haglund's deformity, posterior heel spur formation, increased calcaneal pitch angle, and ultrasound-confirmed insertional Achilles tendinopathy. Conservative management from a podiatric perspective included footwear modification, custom orthotic therapy with heel elevation, and extracorporeal shockwave therapy. Surgical options were considered in light of systemic metabolic risk.

Conclusions: Emerging evidence suggests that Haglund's deformity size alone does not predict insertional Achilles tendinopathy. Structural alignment, insertional degeneration, and systemic metabolic factors such as T2DM may be more significant contributors. Podiatric biomechanical management plays a central role in f irst-line treatment and may reduce the need for operative intervention in metabolically vulnerable patients.

Keywords: *Haglund's Deformity; Insertional Achilles Tendinopathy; Type 2 Diabetes Mellitus; Cavus Foot; Orthotic Therapy*

Introduction

Haglund's deformity describes a posterosuperior calcaneal prominence that may result in irritation of the retrocalcaneal bursa and Achilles tendon insertion.[1] When associated with posterior heel pain, bursitis, and Achilles pathology, it forms part of the clinical entity commonly referred to as Haglund's syndrome. The condition is often aggravated by rigid heel counters in footwear, leading to the colloquial term "pump bump."

Although Haglund's deformity has historically been implicated as a mechanical driver of insertional Achilles tendinopathy (IAT), the relationship between deformity magnitude and symptomatic disease is controversial. Recent radiographic evidence suggests that the actual size of the posterosuperior calcaneal prominence may not independently determine the presence of IAT.[2] Instead, structural alignment and degenerative tendon changes may play a more substantial role.

Concurrently, type 2 diabetes mellitus (T2DM) has emerged as a systemic risk factor for tendinopathy. A systematic review and meta-analysis demonstrated that individuals with diabetes are more than three times as likely to develop tendinopathy compared with controls.[3] Chronic hyperglycaemia promotes the formation of advanced glycation end products within collagen, increasing tendon stiffness and impairing reparative capacity. These metabolic changes may predispose to degenerative tendon pathology and influence clinical outcomes.

This case highlights the interaction between structural foot morphology, insertional Achilles pathology, and long-standing T2DM from a podiatric perspective.

Case Presentation

A patient with a prolonged history of T2DM presented with progressive posterior heel pain of insidious onset. Symptoms were exacerbated by prolonged ambulation and by wearing closed footwear with rigid heel counters. There was no history of acute trauma. The patient denied symptoms of intermittent claudication, rest pain, or previous vascular intervention.

Clinical examination demonstrated a prominent posterosuperior calcaneal enlargement with localized tenderness at the Achilles tendon insertion (Figure 1A). The ankle exhibited reduced dorsiflexion range of motion, and foot posture assessment revealed a rigid cavus morphology (Figure 1B). Palpation elicited tenderness at the insertional region of the Achilles tendon, with palpable thickening consistent with chronic degenerative change.

Weight-bearing lateral radiographs revealed a posterosuperior calcaneal prominence consistent with Haglund's deformity, associated posterior heel spur formation, and an increased calcaneal pitch angle suggestive of cavus alignment. Ultrasound imaging confirmed insertional Achilles tendinopathy characterized by tendon thickening and hypoechoic changes at the insertion.

Given the chronicity of symptoms and the patient's metabolic background, a conservative podiatric management plan was initiated.

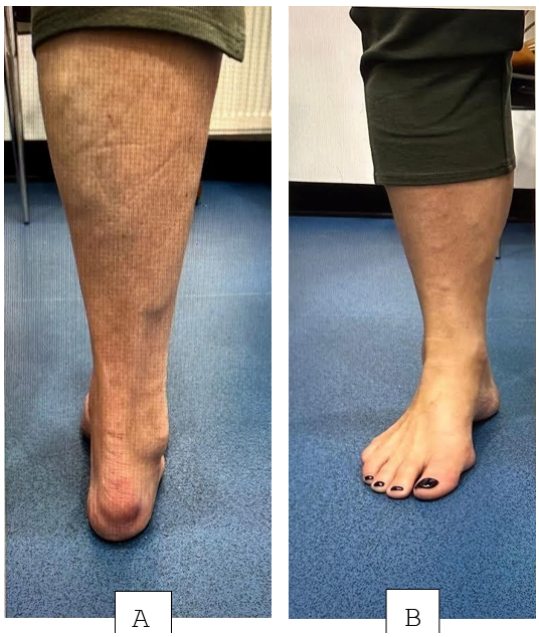

Figure 1. Clinical photographs of the affected foot. (A) Anterior view demonstrating cavus foot morphology. (B) Posterior view showing a prominent posterosuperior calcaneal enlargement consistent with Haglund deformity.

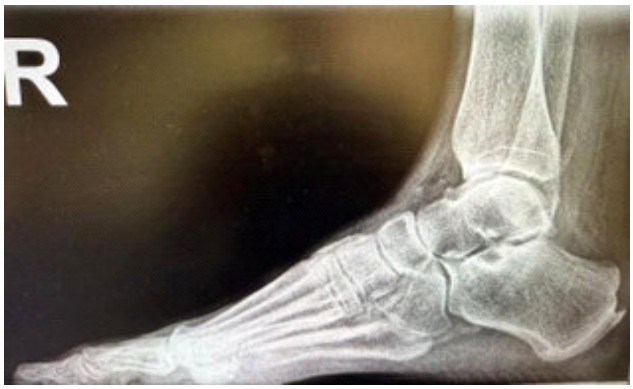

Figure 2. Weight-bearing lateral radiograph demonstrating a posterosuperior calcaneal prominence consistent with Haglund deformity and posterior heel spur formation. The calcaneal pitch angle appears increased, consistent with cavus alignment.

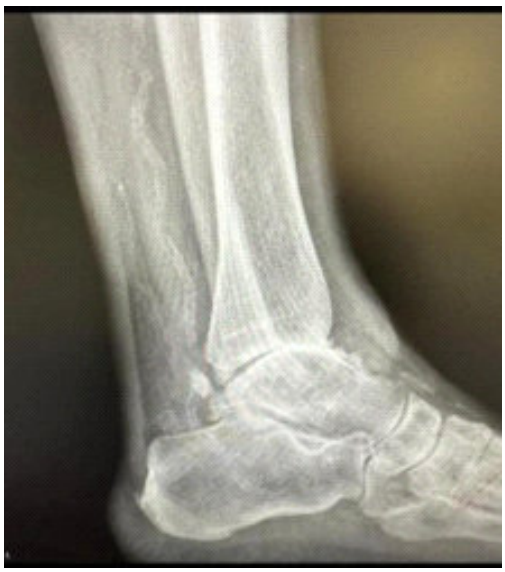

Figure 3. Lateral ankle radiograph demonstrating arterial calcification and mild tibiotalar joint space narrowing.

Management

Management focused on biomechanical load modification. Footwear advice emphasized avoidance of rigid heel counters and high-backed shoes that increase posterior compressive forces at the Achilles insertion. Shoes with softer heel counters and mild heel elevation were recommended to reduce insertional compression.

Custom foot orthoses were prescribed to address cavus biomechanics and reduce Achilles loading. The devices incorporated rearfoot control to improve frontal plane stability and a heel lift to decrease tensile strain at the insertion during gait. In cavus foot morphology, increased calcaneal pitch elevates resting Achilles tension and increases insertional compressive forces during dorsiflexion.[4] Orthotic intervention therefore aimed to reduce mechanical stress through sagittal plane load modification and shock absorption.

Extracorporeal shockwave therapy (ESWT) was administered as an adjunctive modality for chronic insertional tendinopathy. Randomized evidence suggests that ESWT may provide clinical benefit in chronic cases, particularly when combined with load modification strategies. [5]

Corticosteroid injection was not pursued due to concerns regarding tendon integrity. Although corticosteroids may offer short-term analgesic benefit, systematic review evidence indicates potential deleterious structural effects on tendon tissue and an increased risk of rupture in degenerative tendons.[6] These concerns are particularly relevant in patients with metabolic compromise.

Surgical management was discussed but deferred pending response to conservative treatment, given the increased risk profile associated with diabetes.

Discussion

This case demonstrates the multifactorial nature of IAT in the presence of Haglund's deformity and long-standing T2DM. Haglund's syndrome has traditionally been described as a mechanical cause of posterior heel pain, characterised by irritation at the posterosuperior calcaneal prominence.[1] However, contemporary evidence suggests that deformity size alone may not determine symptom development.

Lee et al[2] reported no significant difference in Haglund's deformity size between patients with and without IAT, indicating that the magnitude of the posterosuperior calcaneal prominence may not independently predict insertional pathology. Instead, posterior heel spur formation, intra-Achilles tendon calcification, and increased calcaneal pitch angle were independently associated with symptomatic disease.[2] These findings support the concept that structural alignment and degenerative change may be more clinically relevant than deformity size alone.

In the present case, the increased calcaneal pitch angle and cavus morphology likely contributed to elevated tensile and compressive forces at the Achilles insertion. A cavus foot posture increases resting Achilles tension and may amplify insertional load during gait.

Orthotic therapy has demonstrated benefit in redistributing plantar pressures and improving symptoms in painful cavus foot deformity.[4] From a podiatric perspective, this reinforces the importance of comprehensive biomechanical assessment, footwear modification, and orthotic intervention as first-line strategies aimed at reducing insertional stress.

The association between diabetes mellitus and tendinopathy is well documented. Ranger et al[3] demonstrated significantly higher odds of tendinopathy in individuals with diabetes compared with non-diabetic controls. Chronic hyperglycaemia promotes the formation of advanced glycation end products (AGEs) within collagen, resulting in increased cross- linking, reduced elasticity, and altered tendon mechanical behaviour. Experimental evidence further demonstrates that AGE cross-linking inhibits "discrete plasticity," a protective overload mechanism in which collagen fibrils form kinks to absorb energy and initiate repair signalling.[7] In cross-linked tendons, fibrils remain rigid and fracture abruptly rather than undergoing controlled deformation, reducing energy absorption capacity despite preserved normal-load stiffness.[7] These findings provide a plausible mechanistic explanation for the increased susceptibility to degenerative tendon pathology observed in patients with diabetes. Impaired microvascular perfusion and low-grade systemic inflammation may further compromise tendon healing capacity.[3]

In this case, the lateral ankle radiograph demonstrated linear medial arterial calcification, a finding frequently associated with long-standing diabetes.[8] The patient did not report symptoms of intermittent claudication, and no clinical features of critical limb ischaemia were present. Medial arterial calcification (Mönckeberg sclerosis) involves mineral deposition within the tunica media rather than intimal atherosclerotic plaque and may occur without flow-limiting peripheral arterial disease or classical claudication symptoms.[8] Nevertheless, its presence reflects systemic vascular pathology and reduced arterial compliance. Although a direct causal relationship between medial arterial calcification and tendon degeneration has not been definitively established, vascular alterations may influence tendon nutrition and reparative capacity, warranting cautious clinical assessment.

Conservative management remains central in such cases. Eccentric loading programmes and ESWT have demonstrated efficacy in chronic IAT.[5] Corticosteroid injections, while potentially providing short-term symptom relief, should be approached cautiously given concerns regarding tendon weakening and rupture risk.[6] In patients with diabetes, careful risk stratification is particularly important prior to invasive intervention due to potential delayed healing and increased complication risk.

Although surgical excision of the Haglund's prominence may be considered in refractory cases, operative decision-making should follow failure of comprehensive conservative management and appropriate systemic optimisation. From a podiatric perspective, biomechanical correction, load management, and metabolic risk consideration remain fundamental components of care.

Overall, this case highlights that posterior heel pain in patients with Haglund's deformity and diabetes should be interpreted within a broader mechanical, metabolic, and vascular framework rather than attributed solely to bony prominence size.

Conclusion

Haglund's deformity size alone may not predict the presence or severity of insertional Achilles tendinopathy. Structural alignment, insertional degenerative changes, and systemic metabolic influences, particularly longstanding type 2 diabetes mellitus, likely contribute more substantially to symptom development and impaired tendon resilience.

Recognition of diabetes-related collagen cross-linking and associated vascular changes further underscores the multifactorial nature of pathology in this population. This case highlights the importance of a comprehensive podiatric approach emphasising biomechanical correction, load management, and metabolic awareness prior to consideration of surgical referral.

Conflict of Interest

The authors declare no conflict of interest.

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