Functional Deficits Following Acute Achilles Tendon Rupture are Correlated with Changes in Muscle Structure

Todd J. Hullfish
Kathryn M. O'Connor, MD
Josh R. Baxter, PhD

Department of Orthopaedic Surgery, University of Pennsylvania

Summary

Plantarflexor functional deficits are associated with poor outcomes in patients following Achilles tendon rupture. In this study, we established the link between changes in gastrocnemius structure with plantarflexor function 3 months after injury. We found that deficits in peak isokinetic torque were positively correlated with decreases in resting fascicle length following tendon rupture. These findings suggest that patient function is explained by plantarflexor muscle remodelling, which is a rapid and permanent response to Achilles tendon ruptures.

Introduction

Two-thirds of patients who suffer Achilles tendon ruptures have limited plantarflexor function 1-year following the injury. While tendon elongation has been reported as a possible mechanism of functional deficits [1], our recent findings suggest that changes in muscle structure [2] are detrimental to patient function [3]. However, the link between muscle remodelling and isokinetic plantarflexor function has not been well described. Therefore, the purpose of this study was to establish the link between detrimental changes in medial gastrocnemius structure and plantarflexor function. We hypothesized that the magnitude of fascicle length shortening at 3-months following Achilles tendon rupture would be positively correlated with isokinetic plantarflexor deficits.

Methods

Eight adults (7 males; Age: 43.9 ± 12.9; BMI: 28.7 ± 6.5) who suffered acute Achilles Tendon ruptures and were treated non-operatively were enrolled in this IRB approved study. Subjects were prospectively tracked from the first clinical visit through 3 months after injury. Measurements of the medial gastrocnemius structure and function of the uninjured (contralateral) limb were made at the first visit and repeated for the injured limb at 3 months. Images of the mid-muscle belly were acquired with an 8 MHz ultrasound transducer (LV7.5/60/128Z-2, SmartUs, TELEMED) attached to the leg with a custom made cast. Measurements of muscle structure, characterized by fascicle length and pennation, were made while the ankle was passively positioned at 16° plantarflexion. Isometric and isokinetic dynamometry was performed to quantify plantarflexor function. Subjects lay prone with their knee fully extended and their foot affixed to a dynamometer foot plate. Each subject performed sets of 3-4 MVCs under 4 different contraction conditions. Isometric contractions were performed with the ankle at neutral, and isokinetic contractions were performed at 30, 120, and 210°/s.

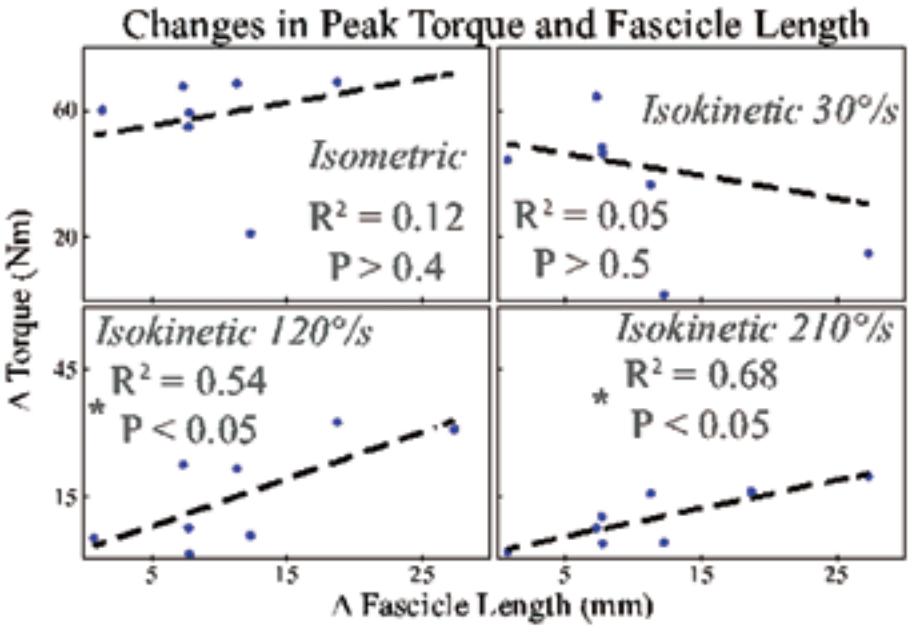

Figure 1: Decreases in fascicle length were positively correlated with decreases in isokinetic torque at 120 and 210°/s.

Results and Discussion

Medial gastrocnemius structure and plantarflexor function following an acute Achilles tendon rupture differed from the healthy contralateral limb 3 months after injury. Fascicle length was 27% shorter ($P = 0.005$) and explained functional deficits in the injured limb. These deficits in isokinetic torque were strongly correlated with changes in fascicle length at 210˚/s ($R^2 = 0.68$, $P < 0.05$) and moderately correlated at 120˚/s ($R^2 = 0.54$, $P < 0.05$). While peak isometric torque decreased by 51% ($P < 0.001$) and peak isokinetic torque measured at 30˚/s decreased by 28% ($P < 0.005$), these functional deficits were not explained by changes in fascicle length. Although and pennation angle was 33% greater ($P < 0.001$) 3 months after injury, plantarflexor kinetic deficits were not explained by these changes in pennation.

Conclusions

Plantarflexor kinetic deficits following Achilles tendon injury are explained by the magnitude of gastrocnemius muscle remodelling at 3 months after injury. These findings challenge the clinical paradigm that functional deficits are explained by tendon elongation [1]. Our ongoing work is focused on understanding the link between tendon elongation and muscle fascicle remodelling.

Acknowledgments

This study was supported by the Thomas B. McCabe and Jeannette E. Laws McCabe Fund. We thank Lauren McGarrity, Shilpa Donde, and Rena Mathew for their assistance with patient recruitment and data collection.

References

1. Silbernagel+ (2012) *Am J Sports Med*, 40(7): 1564-71.
2. Hullfish+ (2019) *bioRxiv [preprint]*, doi: 10.1101/445569
3. Baxter+ (2019) *J Biomech* [Online, Jan. 29, 2019]