Integrating Functional Ultrasonography and Motion Analysis into the Clinical Treatment of Patients with Achilles Tendon Ruptures

Introduction
Achilles tendon ruptures can lead to elongated and abnormal tendons that limit patient function. While the links between musculo-tendon structure and locomotor function have been well described, the effects of altered tendon properties on muscle remodeling and locomotor function resulting from Achilles tendon injuries remain poorly understood. Animal studies have shown that muscles rapidly remodel in order to maintain sarcomere shortening dynamics and function. However, the muscular response to extreme changes in shortening demands imposed by Achilles tendon injuries have not yet been described and may provide critical information for clinical decision making.

Therefore, the purpose of this study was to establish a framework that can be integrated into the clinical setting to link muscle remodeling and functional deficits in patients recovering from Achilles tendon ruptures. In particular, we aim to identify clinically relevant benchmarks that will guide treatment and better manage expectations. This case presents a framework that is currently being implemented in a prospective patient registry to rigorously characterize the muscle remodeling response to Achilles tendon injuries and establish clinical benchmarks for patient success.

Methods
A 27-year old male (1.83 m and 84 kg) presented 2.5 years following an acute Achilles tendon rupture that was surgically repaired by another provider using an open repair technique within 1 week of the initial injury. He had a poor outcome that was confirmed by a clinical outcome score (ATRS score of 49) evaluation by a fellowship-trained foot and ankle surgeon, and an inability to perform a single-leg heel raise. This functional assessment is part of an IRB approved research registry.

Plantarflexor architecture was measured under ultrasonography and muscle remodeling was quantified by contrasting the medial gastrocnemius muscles of the affected and unaffected sides. Muscle thickness as well as fascicle length and pennation were quantified by identifying the superficial and deep aponeuroses and clearly identifiable fascicles.

Plantarflexor function was assessed through a battery of tests that included isometric strength testing, walking, and single-leg heel raise. During these activities, the ultrasonography probe acquired images synchronously with motion capture, dynamometer, and force plate recording of the electrical activity of the muscle, measurement of knee- and ankle-joint rotations, and measurement of ground reaction forces in six men during walking at 3 km h⁻¹. Plantarflexion motion, torque, and power along with fascicle shortening dynamics were calculated to establish the link between muscular and patient function. Fascicle shortening dynamics were quantified using an automated tracking routine utilizing MATLAB's Computer Vision System Toolbox.

Results
Plantarflexor muscle architecture of the affected limb was comprised of shorter and more pennated muscle fascicles as well as a muscle belly that was less thick compared to the unaffected side (Table 1). The resting fascicle length shortened by nearly 60% and demonstrated a 3-fold increase in pennation. Muscle belly thickness also decreased by one-quarter of the unaffected side.

Patient function measurements demonstrated varying degrees of sensitivity to the underlying skeletal muscle adaptations (Table 2). The affected limb demonstrated a 47% decrease in active plantarflexion torque while the fascicles on each limb went through similar amounts of relative shortening. Walking at a self-selected speed did not elicit any functional differences between the affected and unaffected sides. Single-leg heel raises were highly sensitive to deficiencies in muscle architecture, both at the

<table>
<thead>
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<th>Parameter</th>
<th>Affected</th>
<th>Unaffected</th>
<th>%change</th>
</tr>
</thead>
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<tr>
<td>Pennation</td>
<td>34°</td>
<td>13°</td>
<td>162%</td>
</tr>
<tr>
<td>Length</td>
<td>4.6 cm</td>
<td>11.2 cm</td>
<td>-59%</td>
</tr>
<tr>
<td>Thickness</td>
<td>2.2 cm</td>
<td>2.9 cm</td>
<td>-24%</td>
</tr>
</tbody>
</table>

Table 1. Architectural parameters of the medial gastrocnemius muscle of the affected side (surgically repaired tendon) and unaffected (healthy) sides.
Table 2: Plantarflexion biomechanics and fascicle shortening during isometric contractions, walking, and single-leg heel raise. (,) denotes nominal differences between groups.

<table>
<thead>
<tr>
<th></th>
<th>Affected</th>
<th>Unaffected</th>
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</tr>
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<tbody>
<tr>
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<td>133 Nm</td>
<td>−47%</td>
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<td>Shortening</td>
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<td>46% (5 cm)</td>
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<td>15°</td>
<td>~</td>
</tr>
<tr>
<td>Torque</td>
<td>160 Nm</td>
<td>156 Nm</td>
<td>~</td>
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<tr>
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<td>&lt;1 cm</td>
<td>~</td>
</tr>
<tr>
<td>Plantarflexion</td>
<td>12°</td>
<td>16°</td>
<td>47%</td>
</tr>
<tr>
<td>Torque</td>
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<td>140 Nm</td>
<td>~</td>
</tr>
<tr>
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<tr>
<td>Shortening</td>
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<td>5.4 cm</td>
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Discussion

This study introduces a framework that rigorously quantifies muscle remodeling in response to Achilles tendon injuries—establishing a means to prospectively study the implications of injury and treatments on lower extremity function. Despite the clinical understanding that patients recovering from Achilles tendon ruptures may have elongated tendons with intrinsic differences from a normal, never ruptured tendon resulting in reduced function, there has not yet been prospective research that characterizes the effects of tendon injury on plantarflexor muscle remodeling and patient function. Establishing this relationship will provide clinicians with a greater ability to plan treatment based on patient factors—leading to improved function and outcomes.

Our central hypothesis that tendon elongation and intrinsic abnormalities resulting from tendon injuries and treatment will elicit rapid muscle remodeling—which we propose as a mechanism of maintaining tendon tension (Figure 1)—will be tested in a large-prospective cohort using this framework. Initial testing is underway to identify measures of tendon, muscle, and patient function that are most sensitive to changes in patient outcomes and status in order to streamline this framework in order to implement it into clinical settings. Simple musculoskeletal models may also be informed by patient data as part of this prospective framework in order to predict patient function using cost-effective measures of musculoskeletal structure as model inputs.

This research framework is not without limitations. Prospective-functional registries are logistically challenging due to patient attrition and time constraints. In order to minimize these concerns, our methodology will collect a minimal amount of standardized information from all patients being treated for Achilles tendon pathology. Clinical experience suggests that other foot and ankle pathologies may result from compromised plantarflexor function following Achilles tendon rupture. For example, posterior tibialis tendon insufficiency—commonly referred to as flatfoot deformity—may result from increased mechanical demands placed on smaller muscles neighboring the triceps surae group, which was observed in this current case. Smaller muscles of the posterior compartment remodel in response to tendon injury and treatment as a compensatory mechanism to preserve some amount of foot function. To account for these compensatory changes in nearby muscles, we will acquire cross-sectional ultrasonography images of posterior compartment muscles.

Conclusions

We propose a framework for prospectively quantifying plantarflexor function and structure in patients treated for Achilles tendon ruptures. Current work is focused on streamlining this framework using low-cost measurement equipment in order to integrate these tests into the clinical setting. Characterizing the interaction between tendon structure and elongation, muscle remodeling, and functional limitations will aid clinicians in managing patient expectations, prescribing rehabilitation, and intervening in patients at risk of becoming functionally limited.

References


Figure 1. Achilles tendon elongation following repair, which appears to elicit muscle remodeling in order to maintain resting tendon tension. This muscle remodeling reduced muscle shortening and function in this study.

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