Augmented Tendon Achilles Repair Using a Tissue Reinforcement Scaffold: A Biomechanical Study

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ABSTRACT

Background: Missed or chronic Achilles tendon ruptures may have muscle atrophy and tendon retraction resulting in a defect that must be augmented with endogenous or exogenous materials. The Artelon® Tissue Reinforcement (ATR) scaffold is a readily available synthetic degradable poly(urethane urea) material used to augment tendon repair. The objective of this study was to compare human cadaveric Achilles tendon repairs with and without ATR. Materials and Methods: Eighteen fresh frozen human cadaver limbs were dissected and the tendon transected 2 cm proximal to the calcaneal insertion. The control group of nine specimens was repaired with sutures, while the experimental group was repaired with sutures and reinforced with a tubularized patch of ATR. Specimens were tested for ultimate load to failure in an Instron machine after preloading to 10 N followed by cyclic loading for 20 cycles from 2 to 30 N. Results: The ultimate load to failure in the control group was a mean of 248.1 N ± 19.6 (202 to 293 at 95% CI) versus 370.4 N ± 25.2 (312 to 428 at 95% CI) in the ATR group. The ultimate load to failure was 370.4 ± 25.2 N (312 to 428 at 95% CI) and 248.1 ± 19.6 N (202 to 293 at 95% CI) in the experimental and control groups, respectively (p = 0.0015). Creep of the ATR augmented group was 2.0 ± 0.5 mm, compared to 3.1 ± 1.1 mm for the control group (p = 0.026). Conclusion: ATR provided a statistically significant improvement in load to failure when compared to control specimens in a cadaver model. Clinical Relevance: This finding may allow for development of more aggressive rehabilitation techniques following chronic Achilles tendon repairs.

Key Words: Achilles Tendon; Rupture; Repair; Tissue Reinforcement

INTRODUCTION

The Achilles tendon is the largest tendon in the body and the most commonly ruptured, affecting seven per 100,000 people and men four to five times more often than women.10 The tendon complex is composed of contributions of the gastrocnemius and soleus muscles that rotate 90 degrees to insert into the posterior surface of the calcaneus.15 This rotation creates a biomechanical spring to increase stored energy for higher shortening velocity and muscle power, allowing the tendon to withstand loads of up to 4,000 and 5,500 N during running and jumping.4,17 Degeneration of tenocytes occurs with age, and there is microscopic evidence of chronic tendon degeneration present in the biopsy of acute ruptures.9 Acute ruptures usually occur in patients 30 to 55 years old performing eccentric contractions during recreational activities, with 75% percent located 2 to 6 cm proximal to the Achilles insertion on the calcaneus.

Acute ruptures may be treated nonoperatively with cast or boot immobilization or operatively repaired with suture in an end-to-end fashion. Augmentation is rarely necessary. In contrast, patients with missed or chronic ruptures (greater than 6 weeks) may have muscle atrophy, tendon retraction, and inadequate tissue for repair.8 These patients present a unique challenge for the treating surgeon as augmentation with endogenous or exogenous material is required. The Artelon® Tissue Reinforcement implant (ATR) (Artimplant AB, Västra Frölunda, Sweden) is a synthetic degradable material used to reinforce soft tissue. It acts as a scaffold for cellular ingrowth to facilitate healing, stabilizing the tendon as well as allowing angiogenesis and neocollagenesis.7 This material has shown good biocompatibility in soft tissue and bone in animal studies and clinical use,5,14,18–20 Integration into the host tissue occurs after 6 months, which promotes the return of the normal physiological properties of the tendon.12 The implant is synthetic and has no risk of disease transmission or immunologic reactions toward donor collagen. It is these properties that make the ATR scaffold a good option for strengthening a direct tendon repair or tendon transfer.
The goal of this laboratory study was to evaluate the amount of strength added to an Achilles tendon repair reinforced with the Artelon® Tissue Reinforcement scaffold.

MATERIALS AND METHODS

Eighteen fresh frozen human cadaver lower limbs were separated into two groups of nine based on repair technique with or without an ATR augmentation. These specimens were unmatched for left versus right, sex, and age. A power analysis revealed that a minimum of seven specimens per group would be necessary. A triceps surae-Achilles-calcaneus dissection was performed and the ankle was disarticulated through the tibiotalar joint. The mid- and forefoot remained undissected.

To standardize the repairs and loading conditions, the Achilles tendons from specimens of both experimental groups were transected using a No. 10 scalpel to create a simulated rupture two cm from the insertion in the calcaneus. The simulated ruptures were subsequently repaired with two No. 2 ultra high weight polyethylene (Arthrex, Inc. Naples, FL) sutures in a modified Krackow fashion of two rows with four squared knots. In the first group of nine, the simulated cadaver limb tendon ruptures were repaired using only the standard locking loop suture described above (Figure 1). In the second group of nine, repair with the locking loop sutures was reinforced using the ATR, and a 4 × 4 cm patch of ATR was tubularized over the Krackow locking loop repair and secured to the tissue using three horizontal mattress 2-0 ultra high weight polyethylene sutures on each end (proximal and distal) of the defect (Figure 2). Two of the sutures were placed at each corner of the patch with one suture in the middle between the core sutures for a total of six sutures securing each patch.

Each specimen was tested on an Instron machine (Hydraulic model 8511, Instron, Canton, MA) using a standard load to failure test. Specimens were tested in a randomized order and the ultimate strength and load at failure were recorded.

RESULTS

Mode of failure

All of the specimens failed from suture breakage at the site of repair. None of the samples failed due to the Achilles tendon pulling off the calcaneus or the triceps surae complex slipping out of the tendon clamp; however, three specimens in the control group and one specimen in the experimental group did fail due to one or both of the knots of the Krackow complex coming untied. None of the suture complexes pulled through the Achilles tendon tissue. In the ATR group, no specimens failed via pull through on the ATR scaffold.

Ultimate load to failure

The ultimate load to failure of the experimental repair group with the Artelon Tissue reinforcement was 370.4 ± 25.2 N (312 to 428 at 95% CI) (Table 1 and Figure 3). The ultimate load to failure of the control group was 248.1 ± 19.6 N (202 to 293 at 95% CI) (p = 0.0015). The stiffness
Table 1: Results for Ultimate Load to Failure, Length to Failure, Stiffness, and Creep in the Non-Artelon Control Group (N) and Artelon Augment Group (A)

<table>
<thead>
<tr>
<th></th>
<th>N</th>
<th>A</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ultimate load to failure (N)</td>
<td>248.1 ± 19.6</td>
<td>370.4 ± 25.2</td>
<td>0.0015*</td>
</tr>
<tr>
<td>Length to Failure (mm)</td>
<td>54.272 ± 14.751</td>
<td>74.288 ± 17.495</td>
<td>0.033*</td>
</tr>
<tr>
<td>Stiffness (N/mm)</td>
<td>9.873 ± 2.511</td>
<td>12.370 ± 3.020</td>
<td>0.137</td>
</tr>
<tr>
<td>Creep (mm)</td>
<td>3.126 ± 1.141</td>
<td>2.051 ± 0.517</td>
<td>0.026*</td>
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*Statistically Significant

Fig. 3: Bar graph comparing the mean ultimate load to failure between the Non-Artelon Control Group (N) and Artelon Augment Group (A).

Fig. 4: Bar graph comparing mean stiffness between the Non-Artelon Control Group (N) and Artelon Augment Group (A).

Fig. 5: Bar graph comparing mean length to failure between the Non-Artelon Control Group (N) and Artelon Augment Group (A).

of the tendon repair construct was calculated using the force versus elongation curve for the ultimate load to failure curve. The difference in mean stiffness for the Artelon® Tissue Reinforcement group was 12.37 ± 3.02 N/mm, compared to the control group stiffness of 9.87 ± 2.51 N/mm, and was not found to be statistically significant (p = 0.137) (Figure 4).

**Ultimate length to failure**

Ultimate length to failure was defined as the distance of stretch where the construct was no longer able to withstand any further loading from the Instron machine. The mean length to failure for the ATR augment group was 74.3 ± 17.5 mm (Figure 5). The mean length of failure for the control group without the ATR augment was 54.3 ± 14.8 mm (p = 0.033).

**Creep**

Creep was calculated from the difference between the midcycle length of the construct during the first 30 N loading cycle and the last 30 N loading cycle. This is a measure of stretch in the system. For the ATR augment group this was 2.1 ± 0.5 mm, compared to 3.1 ± 1.1 mm for the control group (p = 0.026) (Figure 6).

**DISCUSSION**

Controlled biomechanical analysis of a simulated rupture and repair of cadaveric tendon is difficult to achieve in vitro. The ultimate load to failure of intact human cadaveric Achilles tendon has been reported in the literature to be between 843N and 5579N. Previous simulation of in vitro Achilles tendon ruptures have resulted in failure of the calcaneus bone or proximal muscle clamps rather than the tendon failure. To overcome this problem, Watson et al. utilized a scalpel to create an artificial rupture in cadaveric Achilles tendons 2 cm above the bony insertion. Testing of specimens repaired in this model demonstrated
failure of the suture near the repair site as anticipated, rather than failure at the calcaneus or tendon clamp. The locking loop suture technique was found to be 1.73 and 1.58 times stronger than Kessler and Bunnell methods, respectively.21

In a study of acute ruptures, repairs augmented with a gastrocnemius fascia turn-down procedure created a larger operative wound, had a longer operative time and experienced more wound infections. Only 56% of patients had an excellent overall ankle score after surgery, compared to 63% in the simple repair group.16 Missed or chronic Achilles tendon ruptures may have muscle atrophy; tendon retraction and a defect that must be augmented with endogenous or exogenous materials.8 The gap or interval scar tissue in the tendon can make a direct repair tenuous while increased time for healing of poor quality tissues delays the rehabilitation process. Depending on the quality of the tissues and gap, the different options for reconstruction include: flexor hallucis longus transfer with or without Achilles repair, gastrocnemius turndown, or augmentation with xenograft.

Elias et al. evaluated forty patients with chronic Achilles tendinosis at an average of 27 months after flexor hallucis longus transfer.3 They found no loss of plantarfexion strength or plantarfexion power in the postoperative ankles, an average AOFAS score of 96 out of 100 post-repair and a decrease in VAS from 7.5 preoperatively to 0.3 postoperatively.

In 2008, Barber et al., demonstrated that augmentation of an in vitro Achilles rupture with a xenograft provided greater strength and stiffness compared to repair without augmentation.1 The data presented in this investigation are similar; however, the ATR is thinner, has a potential for fibroblast ingrowth, and has improved handling characteristics compared to a xenograft. The ATR, a degradable polyurethane urea patch, has been shown to facilitate healing via cellular ingrowth, angiogenesis and neocollagenesis.19

Commercially available sizes range from 3 cm × 4 cm to 6 cm × 9 cm.

In this laboratory experiment, the control group without ATR augmentation ultimately failed at a load considerably lower than the group with ATR augmentation. Enhanced mechanical strength of the ATR group suggests that in the clinical setting, its use may facilitate an accelerated rehabilitation program and return to activity. The decreased creep and longer length at failure in the ATR group also supports the concept of greater resiliency which may allow for more aggressive rehabilitation after repair of a chronic Achilles tear with ATR.

Weaknesses of our study include a model with a cleanly transected Achilles tendon analyzed at one time interval in an ex vivo setting. Additionally, the tendon repair construct was loaded to failure at a non-physiologic rate of 6 mm/sec to accommodate the testing apparatus.

CONCLUSION

The superior mechanical properties of the ATR group when compared with the non-augmented group at time zero suggest that tendons repaired with ATR might be amenable to more aggressive rehabilitation techniques. More work is needed to observe the ATR repair throughout the healing process. Currently, a prospective study using ATR for repair of chronic Achilles rupture is underway at our institution.

REFERENCES