Knee Surg Sports Traumatol Arthrosc (2011) 19:2131–2137
DOI 10.1007/s00167-011-1430-3

EXPERIMENTAL STUDY

Structure of retracted tendons after staged repair following continuous traction

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Received: 19 November 2010 / Accepted: 27 January 2011 / Published online: 12 February 2011

Abstract
Purpose The effect of staged repair involving continuous re-lengthening of the retracted musculotendinous unit after rotator cuff tear is not known. We quantified changes in chronically retracted tendons undergoing no repair or a staged repair involving an initial re-lengthening of the musculotendinous unit by traction in a sheep model of massive rotator cuff tear.
Materials and methods Infraspinatus tendons of 12 sheep were released and allowed to retract for 4 months. Repair was performed after the retracted musculotendinous unit had been progressively returned to its original length through continuous traction in 8 sheep (group I). In the other 4 sheep (group II) traction was not successful and the tendons remained retracted. Tendon structure was assessed macroscopically, by MRI, histology, and TEM.
Results Normalized to their contralateral controls, at sacrifice, tendon thickness was unchanged in group I (116%, n.s) and increased in group II (129%, $P < 0.05$), however with substantial shortening. Increased collagen fiber crimping and disorganization was found in group II, whereas in group I the differences from normal tendon were less pronounced.
Conclusion Retracted musculotendinous units have deteriorated tendons, characterized by increased collagen fiber crimp, and ultrastructural collagen fibril atrophy and disorganization. Continuous traction may arrest and partially restore degenerative changes in retracted tendon. The findings of this study might contribute to new approaches for the treatment of chronic "irreparable" rotator cuff tears.

Keywords Rotator cuff · Retraction · Continuous traction · Re-lengthening · Sheep · Tendon

Introduction

Tear of the rotator cuff tendons is a common cause of musculoskeletal morbidity, increasing in prevalence with advancing age [8, 20, 31, 32]. If not recognized early, the affected musculotendinous unit retracts progressively and an attempted repair to bone becomes increasingly difficult due to tension associated with a shortened musculotendinous unit [22].

Healing after repair is not always achieved and is limited by the quality of the structurally altered musculotendinous unit [13, 17, 18]. The recurrence rate of tendon ruptures ranges from 13 to 80% [11, 18], depending on the size of the tear, the degree of musculotendinous unit degeneration [17], patient demographics, and post-operative patient compliance [5].

A large body of literature [7] focuses on factors that can lead to tendon rupture [2, 23, 28, 29], on the immediate changes of the tendon architecture with disuse after rupture [19, 21], and on the repair and healing of the tendon [1, 4, 6, 12, 24]. Furthermore, various reports document the effect of continuous lengthening of the normal tendon [9, 10, 34]. While there is evidence that continuous traction

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can partially reverse pathological alterations in chronically retracted muscle [14], there is no information regarding the behavior of chronically retracted tendons undergoing lengthening. The purpose of this study was to explore the quantitative and qualitative histological changes of tendons that had undergone chronic retraction and to explore the hypothesis that a staged repair after re-lengthening by continuous musculotendinous traction may restore tendon structure.

Materials and methods

The present study is part of a series of experiments investigating the effect of experimental tendon release leading to retraction and subsequent traction applied continuously to the retracted musculotendinous unit [14, 25–27]. The animal experiments described in this article have been conducted according to state and local laws of animal welfare and approved by the Investigative Review Board (ZH-193/2004).

Animal model and groups

A previously established experimental sheep shoulder model of rotator cuff tear was used [14, 25–27]. The infraspinatus tendon was released through an osteotomy of the greater tuberosity. During the period of retraction, the bone chip and distal part of the tendon of the musculotendinous unit were shielded against spontaneous healing (scar formation) by encasement within a silicone tube as previously described [14, 15]. Twelve sheep were allocated for 16 weeks of retraction followed by continuous traction of the tendon to the original tendon reinsertion site, and then 12 weeks of rehabilitation. However, in only 8 (group I) of the 12 sheep was the re-lengthening procedure successful [14]. In the remaining 4 sheep (group II), traction of the musculotendinous unit failed, resulting in a resumption of retraction. All sheep were adult Swiss alpine sheep of similar body weight: 46.1 ± 4.8 and 43.2 ± 2.1 kg in group I and II, respectively. The mean age of the sheep was 16 ± 1.6 and 15.5 ± 2.4 months in group I and II, respectively. The sheep were killed 34 weeks after the initial tendon release, and both the operated and the contralateral shoulders that served as a control were harvested for analysis 12 weeks after explanation of the traction device and repair or solely explanation of the traction device in group I and II, respectively.

Macroscopic assessment

After harvesting the shoulder and imaging with MRI, the entire musculotendinous unit of the infraspinatus including the entire tendon from its bony insertion to the medial end of the muscle was dissected and fixed in formalin for further investigation. These samples were cut longitudinally through the middle of the tendon for macroscopic (optical) assessment of tendon thickness, muscle pennation angles, and surface area of the tendon. The samples were positioned in a standardized matter and digitally photographed alongside a metric scale. Digital image analysis was performed with Adobe Photoshop Professional 7.0 (Adobe System Inc., San Jose, CA, USA). Total length of the tendons was defined as the distance between bony insertion of the tendon and the medial intramuscular end of the (central) tendon, and measured by the same investigator. The bony insertion of the tendon was considered as either the bone chip in treated groups or the greater tuberosity in the control shoulders. Thickness of the tendon was measured as a function of the relative position along the tendon length in increments of 10% of the total length. Mean tendon thickness was calculated by averaging these incremental measurements of tendon thickness. The pennation angle was measured from the first distal insertion point of the muscle fibers at the tendon to 100% of the total tendon length. Tendon surface area was measured by contouring the margins of the tendon cross section from 0 to 50% and from 50 to 100% of total length.

MRI

MRI analysis (Siemens Symphony and Avanto; Siemens medical solutions, Munich, Germany) was performed to assess the volume of the tendon. For MRI, a standardized procedure described previously [15, 16] was used. The tendon proximal to the original insertion site (greater tuberosity) was examined. The volume of the tendon was measured using dedicated software, featuring semiautomated segmentation with interpolation (Myrian, Intrasense, France). The transversally acquired MR images depicting the cross section of the infraspinatus tendon were used for volumetry. The contour of the tendon was delineated on each MR image starting at the bone insertion to a predefined point corresponding to half of the tendon length. Volumetric results were obtained by the software volume rendering algorithm, multiplying the number of 3-D segmented voxels by voxel volume.

Histological analysis

Musculotendinous specimens were harvested for histological analysis at 15, 45, and 75% lengths along the tendon, embedded in paraffin, and processed with hematoxylin and eosin, Van Gieson, and periodic acid-Schiff stains. Samples were investigated with an Eclipse E600 microscope (Nikon, Egg, Switzerland) equipped with a fourfold

magnification objective and polarization optics (used to visualize collagen fiber crimping). Two representative fields of view were digitalized using a Kappa DX20 camera (Kappa optoelectronics GmbH, Gleichen, Germany) and analyzed with Adobe Photoshop Professional 7.0 to assess collagen fiber micro-architecture. Aberrant (abnormal) crimping of collagen fibers was defined before analysis by crimp wavelength smaller than 200 μm. Areas with aberrant crimp were quantified as a percentage of total visible collagen fibers.

Transmission electron microscopy

From the dissected musculotendinous units, standardized blocks of approximately 2 mm by 2 mm by 2 mm were cut at 10% of the total tendon length, and samples were fixed with 2.5% glutaraldehyde in PBS for 1 h and washed three times with PBS. Subsequently, the specimens were post-fixed with 2% osmium tetroxide in PBS for 30 min, washed once with distilled water, dehydrated in a sequence of ethanol solutions (70% ethanol in water 2 × 30 min, 96% ethanol in water 30 min, 100% ethanol 30 min, and 100% anhydrous ethanol 2 × 30 min), embedded in Epon (30% Epon in anhydrous ethanol for 1 h, 50% Epon in anhydrous ethanol for 2 h, 70% Epon in anhydrous ethanol over night, and 100% Epon for 2 h), and polymerized at 60°C for 48 h. Thin sections were stained with aqueous uranyl acetate 2% and Reynolds lead citrate, and imaged in a Phillips CM-12 transmission electron microscope (FEI, Eindhoven, Netherlands) using a Gatan Bioscan CCD camera (1,024 × 1,024 pixels) and digital micrograph acquisition software (Gatan GmbH, Munich, Germany). Collagen fibril diameter and alignment were semi-quantitatively documented.

Statistical analyses

Data were statistically analyzed using Student's paired *t* test for intra-group comparison and Spearman or Pearson correlation at a significance level of $P < 0.05$ (PRISM Version 5.01 for Windows; Graphpad, La Jolla, CA, USA). All statistical values refer to intra-group comparisons, comparing each group with itself before and after the intervention (paired controls). Values were normalized to the corresponding contralateral shoulder and denoted as a percentage of control (%).

Results

Macroscopic observations

There was a difference in macroscopic tendon dimension between the groups compared to their intact contralateral controls. Tendon shortening was more pronounced in group II (to 66.0 ± 6.2%, $P = 0.005$). The mean final relative length of the tendons was shortened in group I (77.7 ± 11.6%, $P = 0.001$). The mean tendon thickness was similar to controls (116 ± 25% of control, n.s.) in group I and slightly increased in tendons of group II (129 ± 11% of control $P = 0.025$) in accordance with their reduced length.

Tendon thickness was positively and significantly correlated with pennation angle of the inserting muscle fibers in both groups (group I: $r = 0.77$ and $P < 0.0001$, group II: $r = 0.75$ and $P < 0.002$). A similar correlation was observed in control tendons (group I: $r = 0.42$ and $P < 0.0001$, group II $r = 0.47$ and $P < 0.0025$).

The surface area of the distal half of the tendon (region closer to the humeral head) was not significantly different from their paired controls in both groups I and II (89 ± 29%, n.s. and 91 ± 15%, n.s., respectively). The surface area in the proximal half of the tendon appeared to have decreased in both groups (65 ± 22% in group I, $P = 0.008$ and 82 ± 10%, $P = 0.035$ in group II).

MRI

Compared with control tendons, there was a loss of tendon volume in the distal part of the tendon in group II (50 ± 12%, $P = 0.017$). In group I, the loss was slightly less pronounced and not significant (63 ± 24%, n.s.) (Fig. 1).

Histological analyses

Histological analysis by polarization microscopy of the collagen fibers showed areas of aberrant crimping in all groups compared to their paired controls. Crimping was more pronounced in group II if compared to group I. Group I showed mixed regions of normal and aberrant crimping of the collagen fibers (Figs. 2, 3).

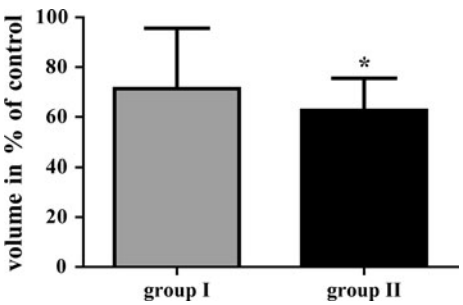

Fig. 1 Compared to the control, the volume of the tendons of *group I* was higher (63 ± 24%, n.s.) than of *group II* (50 + 12%, $P = 0.017$*)

Transmission electron microscopy

The collagen fibril structure of the tendon, revealed by TEM, indicated smaller (atrophied) fibrils in group II. In group I, collagen fibrils appeared more aligned with a tendency toward the bimodal distribution that characterized the intact control tendons (Fig. 3).

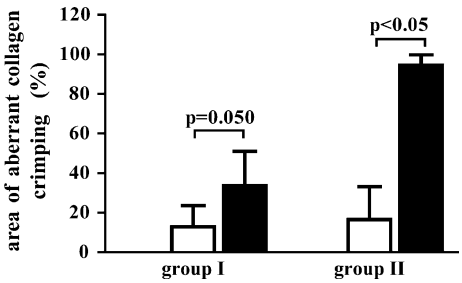

Fig. 2 The area of aberrant collagen fiber crimping is more pronounced in *group II* ($94 \pm 6\%$, $P = 0.001$) than in *group I* ($33\% \pm 17\%$ $P = 0.050$) (*black bars* are the treated tendons, *white* contralateral control)

Discussion

The most important finding of this study was that released and chronically retracted tendons undergo histological deterioration with associated macroscopic changes and that a reversal of this degeneration seems to be partially possible upon continuous muscle and tendon traction.

The retraction and degeneration of the musculotendinous unit represents a critically limiting factor in the repair of tendon tears, a yet unsolved but common clinical problem. While degenerative morphological changes in chronically retracted tendon are likely to play a role in poor repair outcome, to our knowledge such changes and their potential for reversal after staged repair have not been investigated. We hypothesized that as with the muscle, which has been a focus of most related research to date, the tendon itself may contribute to the retraction and degeneration process. This study explicitly considers not only the tendon between muscle and bone but also the central tendon, which can extend throughout the entire length of the muscle and serves as a mechanical backbone for insertion

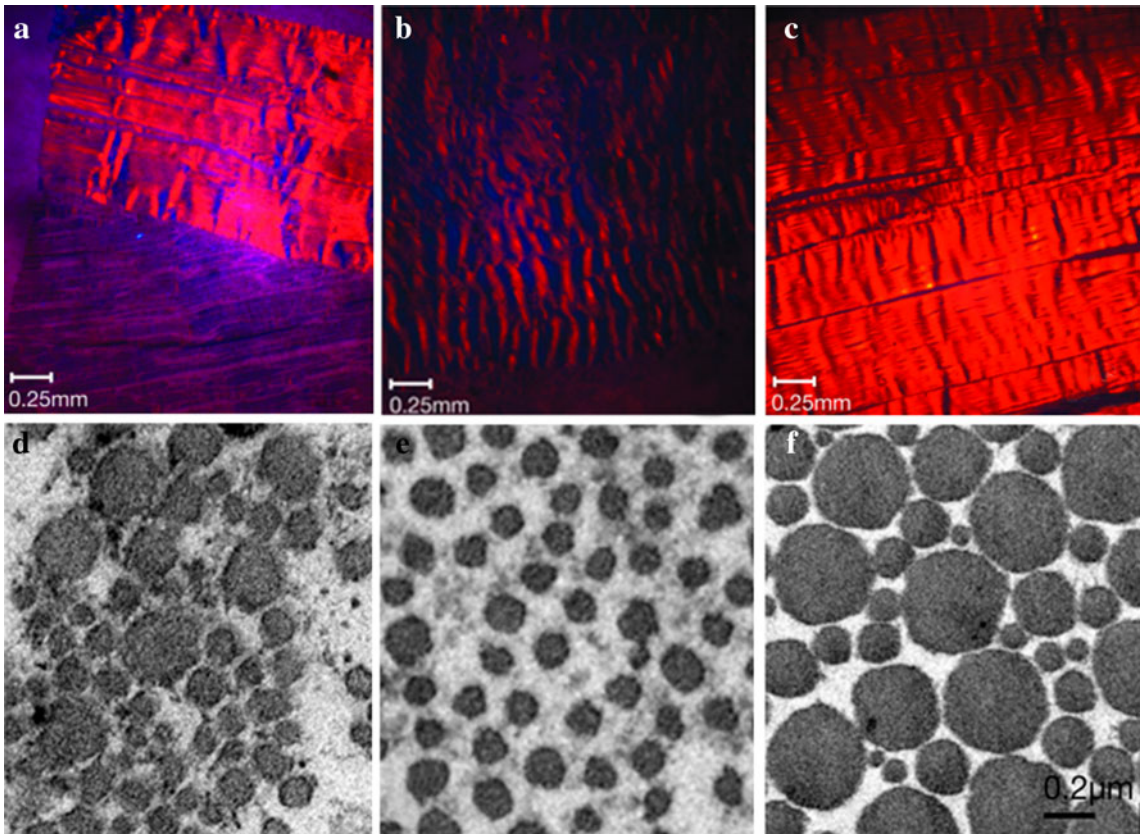

Fig. 3 Polarization microscopy (Hematoxylin Eosin) with 4× magnification of group I (**a**), II (**b**), and normal tendon (**c**). In comparison to the control (**c**), group I (**a**) represents the most similar crimp pattern. The crimping of the collagen fibers are abnormally enhanced the most in group II (**b**). Transmission electron microscopy (TEM) with 4,0000× magnification of group I (**d**), II (**e**), and a normal tendon (**f**). The fiber diameter is smallest in group II (**e**). Bimodal distribution of fibril diameters is noted in group I (**d**) and in the normal tendon (**f**)

of the muscle fibers. We hypothesized that this mechanical relationship may result in concomitant degenerative histological changes in both tissues after tear, or in this case, after experimental tendon release.

Tendon plasticity has been demonstrated in normal tendons that have been shown to adapt their length [9, 34] and alter their biomechanical properties [10] in response to changes in functional demands. However, there is little information about the morphological changes associated with tendons that have been torn (or released) and their potential to regenerate after repair. The purpose of this study was to investigate quantitative and qualitative changes in rotator cuff tendons that had undergone chronic retraction and subsequent repair. We specifically compared repairs performed using a multistage approach featuring continuous musculotendinous traction prior to reattachment.

While the results we present may lend valuable insight, there are limitations that must be considered when interpreting these results. First, group II ($n = 4$) was formed by sheep in which staged repair was attempted and not possible. Although a group that would have been compromised of animals in which no attempt of continuous traction would have been performed might be a better comparison, we believe that group II, by being build of animals in which the tendon remained retracted, can serve as a sufficient representation. A second limitation might be that single TEM images from each group cannot be considered as quantitative evidence, although changes indicated at the collagen ultrastructural level were consistent with our other findings. Further quantitative histology is notoriously accompanied by intra- and intertester reliability issues; quantification of the area of crimped collagen fibers as a percentage of total visible collagen fibers was done systematically with use of a software to limit potential biases. The observed differences between the groups were large, with effect sizes that likely precluded spurious statistical outcomes that could be induced by any uncontrolled bias in the analysis. A third limitation might be the surgical and technical challenges to perform the procedures in a highly standardized fashion (particularly the implantation of the traction device) contributing to a larger standard deviation of the results.

Although the sheep shoulder model is a well-established model for investigation of rotator cuff disease [14, 15, 35], this model has known limitations as a human surrogate [33]. Beside anatomical differences such as independent insertion of the rotator cuff tendons to the greater tuberosity in quadrupedal species, differences in histological appearance and biological tissue behavior such as the tendency of spontaneous reattachment through scar formation (necessitating our use of a silicon sleeve to prevent this) might make translation of the results of the sheep shoulder model to the human more challenging. Despite these limitations, the ovine infraspinatus remains the most widely employed large animal model of rotator cuff tear (with muscle degeneration characteristics that are analogous to the human condition), and the current study represents a potentially important advance in what we have learned using this model [14–16, 25–27].

As expected, all animals demonstrated profound changes in tendon histology at both the microscopic and ultrastructural levels. However, there were important differences between the groups; if left unrepaired (group II), tendons were thicker and had a smaller total volume as a result of substantial shortening. This macroscopic finding can be explained by the markedly aberrant collagen fiber crimping and evident atrophy of the individual collagen fibrils that was observed using TEM [30].

While those tendons with failed repair (groups II) had signs of severe degeneration, tendons of group I with staged and successful repair after musculotendinous traction to original length did possess a histology that was significantly and substantially more similar to a normal tendon. Nonetheless, despite traction of the tendon end to the original insertion site, staged-repair tendons were still shorter than intact control tendons and extended less into the persistently atrophic muscle, resulting in partially diminished tendon volume.

As we have previously described, an increase in muscle pennation angle associated with chronic tendon tears was accompanied by (and may reciprocally be accelerated by) shortening of the central tendon [27]. In this study, we observed a strong correlation between the thickness of the tendon and the pennation angle of the inserting muscle fibers, regardless of the treatment. While one could interpret increased muscle fiber pennation, tendon shortening, and tendon thickening as independent signs of muscle and tendon retraction, it could alternatively be speculated that changes in muscle drive changes in the tendon. In this model of musculotendinous retraction, higher muscle pennation angles draw the tendon into the muscle, resulting in abnormal collagen crimp that pushes the tendon outward. This pathomechanical process would thus simultaneously induce both a greater tendon thickness and a shorter tendon length. Using traction to restore a more normal muscle fiber pennation angle would then impart a concomitant reversal of aberrant tendon histology, with a reduction in over-crimping of the collagen fibers and a restoration of more normal ultrastructure (collagen fibril morphology). While our ultrastructural TEM findings are qualitative, it appears that staged-repair tendons returned to a bimodal distribution of collagen fibrils. Indeed, lateral fusion of individual collagen fibers could be observed, a well-known process in tendon development and healing that is essential to recovery of mechanical properties [3, 36].

Conclusion

In conclusion, we have shown that released and retracted tendons undergo histological deterioration with associated macroscopic changes (shortening and volume loss). Further, the degenerative processes in muscle retraction and tendon retraction following release are apparently coupled, with a strong correlation between increasing muscle fiber pennation angle and hallmarks of tendon retraction (aberrant crimp, reduced length, etc.). A reversal of this degeneration seems to be possible upon continuous muscle and tendon traction—a finding that has never been reported to our knowledge. It thus appears that the contribution of the central muscle tendon atrophy to difficulties in surgical repair of chronically retracted musculotendinous units such as the rotator cuff or Achilles tendon have been largely underestimated and will need further investigation.

The findings of this study might contribute to new approaches for treatment of chronic "irreparable" rotator cuff tears.

Acknowledgments The animal experiments in this study were funded by Swiss National Research Foundation, grant number 320-113424/1. The authors would like to thank Dr. sc nat. A. Käch and U.Lüthi at the University of Zürich, Center for Microscopy for their expertise and support in TEM imaging.

Conflict of interest The animal experiments described in this article have been conducted according to state and local laws of animal welfare and approved by the Investigative Review Board (ZH-193/2004).

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