J Shoulder Elbow Surg (2011) 20, 698-707

ELSEVIER

Journal of Shoulder and Elbow Surgery

www.elsevier.com/locate/ymse

Decreased loading after rotator cuff tears leads to improved biceps tendon properties in a rat model

Cathryn D. Peltz, PhD, Jason E. Hsu, MD, Miltiadis H. Zgonis, MD, Nicholas A. Trasolini, David L. Glaser, MD, Louis J. Soslowsky, PhD*

McKay Orthopaedic Research Laboratory, University of Pennsylvania, Philadelphia, PA, USA

Background: The purpose of this study was to elucidate the mechanism of biceps tendon changes after rotator cuff tears. We hypothesized that increased loading on the biceps tendon after rotator cuff tears will result in further detrimental changes whereas decreased loading will result in increased organization and more normal tendon composition. In addition, we hypothesized that changes with altered loading will begin at the proximal insertion into bone and progress along the tendon length at later time points.

Materials and methods: Supraspinatus and infraspinatus tendon detachments in rats were followed by various loading protocols at various time points. Regional changes in cellularity, cell shape, collagen organization, and matrix proteins of the long head of the biceps tendon were determined by histologic measures and immunohistochemistry.

Results: Increased loading after detachments resulted in more disorganized collagen after only 1 week and compositional changes by 4 weeks. By 8 weeks, decreased loading resulted in increased organization, decreased cellularity, a more elongated cell shape, and more normal tendon composition. Organizational changes with increased loading began in the intra-articular space and progressed along the tendon length with time.

Conclusions: Combined with previous findings of decreased mechanics with increased loading, these results show that increased compressive loading away from the proximal insertion into bone is a mechanism for biceps tendon pathology in the presence of rotator cuff tears. The striking improvements with decreased loading further support increased loading as a mechanism for biceps tendon pathology because removal of this load led to improvements in tendon histology, organization, and composition.

Level of evidence: Basic Science Study.

Keywords: Biceps tendon; rotator cuff; animal model; tendon injury; tendon pathology; altered loading

Damage to the long head of the biceps tendon is common clinically, although it is rarely isolated and often seen in conjunction with rotator cuff tears.[1,7] However, there is some debate over the role of the long head of the biceps tendon after rotator cuff tears.[5,19] The mechanism responsible for the associated pathology is unknown, and its optimal treatment is somewhat controversial. There is evidence that the biceps tendon plays an increased role as a humeral head depressor when one or more of the rotator cuff tendons are torn.[5] Therefore, the pathology seen may be a direct result of the tendon experiencing loads not seen with an intact rotator cuff.

Animal study approval provided by Institutional Animal Care and Use Committee (protocol 802540).

*Reprint requests: Louis J. Soslowsky, McKay Orthopaedic Research Laboratory, Department of Orthopaedic Surgery, University of Pennsylvania, 424 Stemmler Hall, Philadelphia, PA 19104-6081.

E-mail address: soslowsk@upenn.edu (L.J. Soslowsky).

1058-2746/$ - see front matter © 2011 Journal of Shoulder and Elbow Surgery Board of Trustees.
doi:10.1016/j.jse.2010.11.033

The histology, organization, and composition along the entire length of the biceps tendon are not well characterized. It has been shown that the proteoglycan content is much higher, and very similar to rotator cuff tendons, near the insertion than it is in the intratubercular groove.[4,6] Both tensile loading and compressive loading are seen at this location and the proximal insertion into bone of a biceps tendon in an uninjured shoulder is less organized and expresses different amounts of various collagens and proteoglycans than the rest of the tendon.[6] Away from the proximal insertion into bone, the composition is markedly different, and the collagen fibers are organized along the long axis of the tendon as the tendon experiences primarily tensile loading in this location. The tendon's vascularity has also been shown to be more dense away from the proximal insertion into bone.[6] The variations in function and composition along the length of the biceps tendon may play a role in how and where pathology begins in the presence of rotator cuff tears.

Clinicians have noted that the biceps tendon is flattened, widened, and/or frayed at the time of rotator cuff repair and the damage has also been seen to increase with increasing tear size.[1,4] However, it is often not clear whether the changes are truly degenerate or are due to inflammation. The location where pathologic changes begin is also somewhat controversial. Some investigators believe that pathology occurs at the entrance to the intratubercular groove and suspect that the tendon first becomes inflamed and then damaged when it has difficulty sliding because it is hypertrophied. Neer[8] has long believed that the biceps tendon is susceptible to impingement under the acromion after a rotator cuff tear occurs and that changes begin near the tendon's attachment to the glenoid on its bursal side. It is also possible that changes may occur in this location but on the articular side, because increased compressive loading against the humeral head is present.

The effect of rotator cuff tears on the histologic, compositional, and organizational properties along the entire length of the biceps tendon was examined in a previous study in the rat.[11] The bony anatomy of the rat shoulder is very similar to the human and has previously been shown to be an appropriate model for studies of the rotator cuff.[16] In addition, anatomy of the biceps tendon is also very similar in the rat and the human. In both the rat and the human, the long head of the biceps tendon originates at the superior aspect of the glenoid (referred to here as the tendon's proximal insertion into bone) and passes through the intratubercular groove. It was shown that compositional changes appeared at the proximal insertion into bone as early as 1 week after rotator cuff tendon detachments and that the tendon was also more disorganized in the intra-articular space at this time point. By later time points, this increased disorganization had extended along the entire length of the tendon. It was therefore concluded that a degenerate process was occurring in the tendon and that increased loading near the proximal insertion into bone was a major contributor to biceps tendon pathology in the presence of cuff tears.

Subsequently, this model was used to examine the effect of altered loading after rotator cuff tendon detachment on biceps tendon mechanics to begin to elucidate its role as a possible mechanism for this pathology.[10,13] It was shown that increased loading resulted in detrimental changes along the entire tendon length as early as 4 weeks after detachments whereas there were no changes between detachment alone and decreased loading by 8 weeks. However, earlier time points were not examined, and therefore the biological processes, such as changes in the type of collagen present or decreased organization as a response to compressive loading, that result in the mechanical changes seen with increased loading are unknown. In addition, it is possible that improved biological properties, such as increased collagen organization and composition more like uninjured tendon, are present with decreased loading without yet having a mechanical effect. Therefore, the objective of this study was to determine the effect of altered loading after rotator cuff tears on the regional organizational, histologic, and compositional properties of the long head of the biceps tendon. Our hypotheses were that (1) changes with altered loading will begin at the proximal insertion into bone and progress along the tendon length at later time points, (2) increasing loading will result in further detrimental changes compared with detachment alone by 8 weeks, and (3) decreased loading will result in increased organization and more normal tendon composition by 8 weeks.

Materials and methods

Forty-two Sprague-Dawley rats (Charles River, 400-450 g) were used in this study, which was approved by the University of Pennsylvania Institutional Animal Care and Use Committee. The rats were divided into 3 groups based on postsurgical loading protocols, and rats from each group were killed at 1, 4, and 8 weeks after surgical tendon detachments: supraspinatus and infraspinatus detachment only (SI only) ($n = 4$ at 1 week and $n = 5$ at 4 and 8 weeks), supraspinatus and infraspinatus tendon detachment followed by decreased loading (SI + DEC) ($n = 4$ at 1 week and $n = 5$ at 4 and 8 weeks), and supraspinatus and infraspinatus tendon detachment followed by increased loading (SI + INC) ($n = 5$ at 1 and 4 weeks and $n = 4$ at 8 weeks). In all groups, a unilateral surgery was performed to sharply detach the rotator cuff tendons from their bony insertion, as previously described.[15] In brief, with the arm in external rotation, a 2-cm skin incision was made, followed by blunt dissection down to the rotator cuff musculature. The rotator cuff was exposed, and the tendons were visualized at their insertion on the humerus. The supraspinatus was first separated from the other rotator cuff tendons before sharp detachment at its insertion on the greater tuberosity with a scalpel blade, before detachment of the infraspinatus tendon in the same manner. Any remaining fibrocartilage at the insertion was left intact, and detached tendons were allowed to freely retract without attempt at repair, creating a gap approximately 4 mm from their proximal insertions into bone. The overlying muscle and skin were closed.