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The severity of the long head biceps tendinopathy in patients with chronic rotator cuff tears: macroscopic versus microscopic results

Po-Ting Wu, MD[a,b], I-Ming Jou, MD, PhD[b], Cheng-Chang Yang, MSc[c], Chii-Jeng Lin, MD, PhD[b], Chyun-Yu Yang, MD, PhD[b], Fong-Chin Su, PhD[a], Wei-Ren Su, MD, MSc[b,*]

[a]*Institute of Biomedical Engineering, National Cheng Kung University, Tainan, Taiwan*
[b]*Department of Orthopedics, National Cheng Kung University Hospital, Tainan, Taiwan*
[c]*Institute of Basic Medical Sciences, National Cheng Kung University, Tainan, Taiwan*

Background: This study investigated the histopathology of the long head of biceps (LHB) tendon and correlated the findings with the macroscopic appearances of the LHB and the size of rotator cuff tears (RCTs) in patients with chronic RCTs.

Methods: We compared biopsy specimens from LHBs in 34 patients with chronic RCTs and grossly normal LHBs in 8 patients undergoing shoulder hemiarthroplasty (controls). Duration of preoperative symptoms, the severity of RCTs, and macroscopic appearance of LHBs were recorded, classified, and compared with the histologic grading and apoptosis index of terminal deoxynucleotide transferase-mediated biotin-deoxy uridine triphosphate nick-end labeling (TUNEL) assays of LHBs.

Results: In the RCT group, there were 8 partial-thickness tears with 5 macroscopic LHB lesions, 12 full-thickness tears with 8 macroscopic LHB lesions, and 14 massive tears with 13 macroscopic LHB lesions. There were 6 LHB subluxations. However, the macroscopic grading and the symptom duration were not correlated with the severity of the histology. In patients with massive tears, no matter what the macroscopic appearance of the LHB, the proportion of end-stage (grade 4) histologic LHB tendinopathy significantly increased (85.7%, $P < .05$) compared with patients with other types of RCTs. There was a consistently high incidence of advanced LHB histology (grade 3 or higher) in each classification of RCTs (75.0%-100.0%). The 8 patients in the control group showed milder histopathology (grade 1 or 2). The apoptosis index significantly increased as the tendinopathy progressed ($P < .05$).

Conclusions: The macroscopic pathology of LHB may not fully reflect the severity of tendinopathy, and the coexisting size of RCTs plays a role in the severity of LHB tendinopathy.

Level of evidence: Basic Science Study, Histology.

Keywords: Long head of biceps; tendinopathy; rotator cuff tear; tenotomy/tenodesis; apoptosis; shoulder pain

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*Reprint requests: Wei-Ren Su, MD, MSc, Department of Orthopedics, National Cheng Kung University Hospital, 138 Sheng-Li Rd, Tainan 704, Taiwan.

E-mail address: suwr@ms28.hinet.net (W.-R. Su).

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Shoulder pain is common in chronic rotator cuff tears (RCTs) and is possibly caused by long head of biceps (LHB) tendinopathy.[4,5,23,30,32] Clinically, damage to the LHB is often seen in conjunction with RCTs but rarely in isolation.[9,23] Many studies[9,14,25,29] have shown that the LHB has a high incidence of macroscopically pathologic lesions in an RCT, especially in a massive tear (MT). How to manage concomitant LHB tendinopathy in procedures dealing with RCTs is still a challenge. Removing a potentially advanced pathologic LHB tendon when doing a rotator cuff repair or debridement may be acceptable because it may be mechanically deteriorated and, therefore, prone to rupture.

In a RTC repair, the current indications for an LHB tenotomy or tenodesis are based primarily on the morphology of the biceps.[26,27] A discrepancy between the macroscopic and microscopic findings in LHB lesions may exist, and the relationship between chronic RCTs and the histopathologic changes of concomitant LHB is important and helpful in decision making. However, there is still no such study in the literature.

The purpose of this study was to investigate the histopathology and apoptosis expression of the LHB tendon in a prospective case-control study of patients with chronic RCTs and correlate the findings with the macroscopic appearances of the LHB and the size of the RCTs. We hypothesized that the macroscopic appearance of the LHB is not consistent with its histologic severity and that LHB accompanying massive RCTs presents a higher incidence of histologically end-stage tendinopathy compared with those accompanying other sizes of rotator cuff lesions.

Materials and methods

Patient groups

The study recruited 34 consecutive patients (14 men and 20 women; mean age at surgery, 56.9 years; range, 34-73 years) undergoing arthroscopic treatment for an RCT with or without other ipsilateral shoulder lesions at our university hospital between January and December 2011. The inclusion criterion was a chronic rotator cuff lesion (duration $\geq$3 months[9]). The exclusion criteria were concomitant frozen shoulder syndrome, inflammatory arthritis, connective tissue disease, and age $<$18 years. The demographic data of patients were recorded, and the patients were assigned to 1 of 2 groups by the duration of their preoperative symptoms: $\leq$6 months and $>$6 months.[1] The mean $\pm$ standard deviation of symptom duration was 9.6 $\pm$ 5.0 months (range, 3-24 months). Fourteen patients (41.2%) underwent surgery $\leq$6 months after the onset of symptoms.

All patients were given a preoperative physical examination and a magnetic resonance (MR) image (MRI) scan or MR arthrography to evaluate the rotator cuff, the LHB, and the labrum lesion, if there was one. The final diagnosis was confirmed using the arthroscopic findings and then recorded. During surgery, the rotator cuff tendon lesion was identified and classified as a partial-thickness tear, a full-thickness tear, or a massive tear. Because of variations in the techniques of measurement and the sizes of the patients, complete RCTs that involved 2 or more entire tendons were defined as massive tears[13] to represent more severe tears.

A simplified classification modified from a prior study[6] was created to describe the macroscopic pathologic lesion of the LHB. Type 0 was the macroscopically normal tendon. The type I lesion was defined as tendinitis of the tendon, type II was fibrillation or delamination of the tendon, type III was a tendon tear of less than 50% of the tendon width, type IV was tendon tear of more than 50% of the tendon width (Fig. 1, *A-D*). The location of the LHB was classified as in the bicipital groove, subluxation, or dislocation.

The control group comprised 8 consecutive patients (2 men and 6 women) who were a mean age of 64.0 years (range, 56-72 years) who underwent shoulder hemiarthroplasty for proximal humeral fractures with an intact rotator cuff and a macroscopically normal (type 0) LHB inspected during surgery.[17] All patients provided written, signed, informed consent beforehand for all subsequent procedures.

Surgical procedure and biopsy

At the beginning of all procedures, the LHB was first evaluated using a "dry" scope examination with no pump pressure. The intertubercular groove portion of the LHB had to be retracted into the joint for evaluation. The rotator cuff lesions were treated as follows: if the tear thickness was less than 50%, the treatment was debridement with or without acromioplasty. If the tear thickness was more than 50% or was complete, the treatment was arthroscopic repair. Treatment for types I and II LHB lesions was only debridement. For type III lesions, if there was coexisting LHB subluxation or dislocation, or a massive RCT, resection or tenodesis was done; otherwise, only debridement was done. The treatment for all type IV lesions was tenodesis or resection.

The LHB tendons were arthroscopically harvested from the macroscopic lesion site (Fig. 2). For type 0 tendons, the specimens were harvested from the lateral border of the tendon 1 to 1.5 cm distal to the superior labrum, the location of the common tear site of the LHB.

Histologic grading and detecting apoptosis

The specimens were fixed in fresh 4% paraformaldehyde for 16 to 24 hours at 4°C, then subsequently dehydrated, embedded in paraffin, and longitudinally sectioned. Sequential 5-μm sections were stained with hematoxylin and eosin and examined under a light microscope. Light and polarization microscopy was used to evaluate tendons for changes in tenocyte morphology and collagen bundle characteristics. As detailed in previous studies,[12] we used a semiquantitative method to score each factor on a 4-point scale. According to the sum of scores, the tendinopathy was graded as 0 to 4 (0, $\leq$2, 3, 4, $\geq$5 points; Fig. 1, *E-H*). Grades 3 and 4 were defined as advanced tendinopathy; furthermore, grade 4 was considered end-stage tendinopathy. The histologic grading was assessed by 2 observers unaware of the clinical and arthroscopic findings of patients.

A terminal deoxynucleotidyl transferase-mediated biotin-deoxy uridine triphosphate nick-end labeling (TUNEL) assay was used to identify apoptotic cells by labeling nuclear DNA fragments (In Situ Cell Death Detection Kit, AP 1684817; Roche Diagnostics