

Cross-sectional area of the tendon and the muscle of the biceps brachii in shoulders with rotator cuff tears

A study of 14 cadaveric shoulders

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Background The intraarticular portion of the long head of the biceps tendon is often widened in shoulders with cuff tears. It is unclear whether this is a local phenomenon or is caused by muscle hypertrophy.

Methods We investigated morphological changes of the biceps brachii in 14 embalmed shoulders: 7 with intact rotator cuff and 7 with rotator cuff tears.

We measured the cross-sectional area (CSA) of the tendon of the long head of the biceps (LHB) at 9 levels between the glenoid origin and the musculotendinous junction. The muscle volume and the muscle fiber length of the long and short heads of the biceps were measured to calculate the physiological CSA (PCSA) by dividing the volume by the fiber length.

Results The CSA of the LHB tendon at the entrance to the bicipital groove was greater in cuff tear shoulders than in normal shoulders. The PCSA of the biceps was similar in normal and cuff tear shoulders.

Interpretation Hypertrophy of the LHB tendon appears to be a localized morphological change near the entrance to the bicipital groove.

cuff tear shoulders may be a hypertrophic change to compensate for the functional loss of the cuff (Matsen et al. 1998).

An et al. (1991) reported that tendon cross-sectional area (CSA) correlated well with the physiological CSA (PCSA) of associated muscles. If hypertrophy is caused by compensation or increased muscle activity, we should be able to observe hypertrophy of the entire LHB tendon as well as hypertrophy of the biceps muscles, both the LHB and the short head of the biceps (SHB). However, only the intraarticular portion of the tendon can be observed during surgery unless we open the bicipital groove. To our knowledge, there have been no such studies reported in the literature. We investigated morphological changes in the entire LHB tendon from the glenoid origin to the musculotendinous junction to clarify the PCSA of the LHB and SHB muscles in shoulders with rotator cuff tears.

Material and methods

Specimen preparation

It is well known that the tendon of the long head of the biceps (LHB) is often flattened and widened in shoulders with rotator cuff tears (Burkhead et al. 1998, Matsen et al. 1998). Since the LHB is a stabilizer (Kumar et al. 1989, Itoi et al. 1994) and depressor (Warner et al. 1995, Kido et al. 2000) of the humeral head, the widened LHB tendon in

We examined 14 embalmed cadaveric shoulders (7 with rotator cuff tears and 7 without rotator cuff tears) with an average age of 78 (54–94) years. None of these subjects had had muscle diseases such as progressive muscular dystrophy or myasthenia gravis. The position of the arm at the time of embalment was in adduction and slight inter-

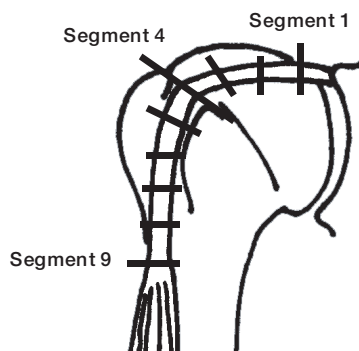


Figure 1. Sites of cross-sectional area (CSA) measurement. The CSA of the LHB tendon was measured at 9 different levels. The distal stump of each segment was sectioned into a thin slice and measured using NIH Image. Segment 1: glenoid origin. Segment 4: entrance to the bicipital groove. Segment 9: musculotendinous junction.

nal rotation. The arm dominance of these cadavers was unknown. We dissected the shoulder and the entire muscles and tendons of the LHB and SHB from the scapular origins to the forearm insertion, placing a mark on the LHB tendon at the entrance to the bicipital groove. There were 2 small tears (< 1 cm), 2 medium tears (1–3 cm), and 3 large tears (3–5 cm) according to the classification by Post (1978). The LHB tendon was covered by the remaining cuff tendon tissues in all the cuff tear shoulders except one.

Measurement of the CSA of the LHB tendon

The entrance point of the LHB tendon into the bicipital groove was located almost at the 4/9 level from the proximal origin. We thus divided the LHB tendon from its glenoid origin to the entrance to the bicipital groove into 4 portions, and the LHB tendon from the entrance to the musculotendinous junction into 5 portions (Figure 1) so that we obtained 9 free portions of LHB tendon of almost equal length. In each segment, the distal stump was sectioned into a thin slice and photographed with a scale. The CSA of each slice was measured using the image analyzing software NIH Image (National Institutes of Health, Bethesda, MD). We compared the CSA of the LHB tendon at each level, between normal and cuff tear shoulders.

Measurement of the PCSA of the muscles

After removing the remnant extramuscular tendons from the muscle belly, we measured the muscle

CSAs of the LHB tendon

Segment no.	CSA of shoulder (mm ²) normal	CSA of shoulder (mm ²) cuff tear	P-values
1	18 (10)	20 (10)	0.7
2	18 (21)	31 (9.5)	0.3
3	13 (27)	33 (6.5)	0.01
4	11 (15)	26 (3.9)	0.009
5	10 (10)	19 (3.7)	0.04
6	9.9 (4.7)	16 (3.3)	0.04
7	9.9 (3.5)	13 (3.1)	0.08
8	9.7 (2.6)	13 (3.1)	0.1
9	10 (2.7)	13 (3.5)	0.2

Segment no., see Figure 1.

volume by water replacement. The muscle fiber length was then measured at 5 different portions of the muscle. The PCSA was calculated by dividing the muscle volume by the muscle fiber length. We compared the PCSA of the LHB and SHB between the normal and cuff tear shoulders.

Statistics

Since the sample size was small and a normal distribution was not confirmed, we used nonparametric statistical analysis. The CSA of the LHB tendon and the PCSA of the muscles between the normal and cuff tear shoulders were compared using the Mann-Whitney U test. Significance was established at the 5% level.

Results

In normal shoulders, the CSA of the LHB tendon decreased with distance from the glenoid origin, and showed a constant value from the entrance to the bicipital groove to the musculotendinous junction (Table). In the cuff tear shoulders, however, the CSA of the LHB tendon increased with distance from the origin, showing the greatest value near the entrance to the bicipital groove, and decreased from the bicipital groove to the musculotendinous junction (Figure 2). The CSAs of the LHB tendon near the entrance to the bicipital groove (segment 3, 4, 5, 6) were greater in cuff tear shoulders than in normal shoulders.

The PCSA of the LHB was 174 (SD 42) mm² in normal shoulders and 175 (SD 15) mm² in cuff tear

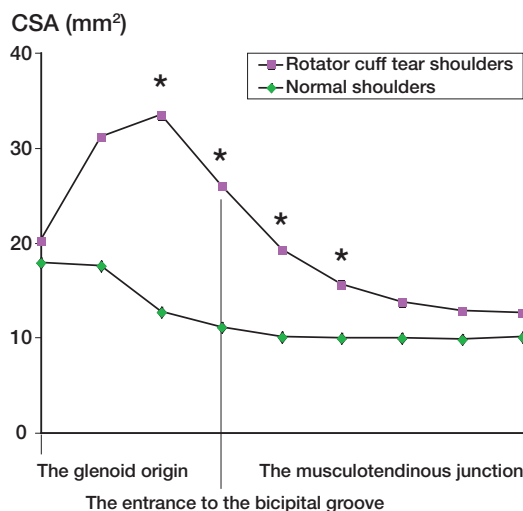


Figure 2. CSA of the LHB tendon (mm²). The CSAs of the LHB tendon near the entrance to the bicipital groove were significantly greater in cuff tear shoulders than in normal shoulders (* $p < 0.05$).

shoulders ($p = 0.95$). The PCSA of the SHB was 113 (SD 38) mm² in normal shoulders and 138 (SD 55) mm² in cuff tear shoulders ($p = 0.3$).

Discussion

Since the LHB tendon is an active stabilizer of the glenohumeral joint (Warner et al. 1995) and an active depressor of the humeral head in cuff tear shoulders (Kido et al. 2000), the widened LHB tendon may be due to hyperactivity of the biceps muscle in order to compensate for the functional deficit caused by rotator cuff tear. An EMG study by Kido et al. (1998) demonstrated that only one-third of patients with rotator cuff tears showed increased activity of the biceps during arm elevation compared to that in subjects without rotator cuff tears. On the other hand, Yamaguchi et al. (1997) reported that there was no significant activity of the biceps observed during arm elevation in patients with rotator cuff tears. Judging from these EMG studies, it is unlikely that the biceps is hyperactive in patients with rotator cuff tears.

It has been reported that CSA of the tendon increased with hypertrophy of the muscle in response to training (Woo et al. 1980). If hypertrophy of the LHB tendon was due to hyperactivity or

overuse of the biceps muscle, we should be able to observe not only hypertrophy of the LHB tendon but also hypertrophy of the LHB muscle. However, we did not find that in our series. We found that the CSA of the LHB tendon increased, while the PCSA of the LHB and SHB muscles did not show hypertrophy. From these data, it seems that hypertrophy of the LHB tendon is not due to hyperactivity or overuse of this muscle.

It has been reported that the origin of the LHB tendon is wider than the rest of the tendons in normal shoulders (Burkhead et al. 1998). This was confirmed in our study. However, although it has been reported that the CSAs of the LHB tendon—both at the intraarticular portion and at the intertubercular portion—are greater in cuff tear shoulders than in normal shoulders (Sakurai et al. 1998), there have been no reports of measurement of CSA of the LHB tendon along its entire length. Our findings show that hypertrophy of the LHB tendon in cuff tear shoulders is a localized hypertrophy at the entrance to the bicipital groove.

The localized hypertrophic change of the LHB tendon may be caused by a combination of many factors such as subacromial impingement, hidden lesion, and compression due to the superior migration of the humeral head. Hypertrophy of the LHB tendon may be affected by the tear size if mechanical impingement is one of the etiological factors of this hypertrophy. However, in our small series we could not find any significant correlation between the tear size and the CSA of the LHB tendon. Effects of these factors on hypertrophy of the LHB tendon should be clarified in a study with a larger sample size.

No competing interests declared.

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