Morphologic Change in Long Head of Biceps Brachii in Rotator Cuff Dysfunction

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The function of the biceps brachii (LHB) at the shoulder joint has been well accepted as a depressor or a dynamic stabilizer of the humeral head. This action becomes more important when there are rotatorcuff tears. Anatomically, the intra-articular portion of the LHB, which arisesfrom the supraglenoid tubercle and the posterosuperior glenoid labrum , runs across the superomedial aspect of the humeral head and enters the intertuberclular sulcus. The intertubercular groove is formed by the greater tubercle, the lessor tubercle, and soft tissue, including the insertion of the subscapularis tendon and the coracohumeral ligament. In individuals with rotator cuff tears, flattening or medial instability of the LHB is somettimes observed intraoperatively, and is thought to result from disruption of the bicipital gliding mechanism. The precise role of this tendon is controversial; its action as a depressor of the humeral head is thought to be increased in patients with impingement lesions. The purpose of this study is to clarify the involevement of the long head of the biceps brachii in rotator cuff dysfunction.

Materials and Methods

Rotator cuff and bicipital lesions were investigated in 170 shoulders of 85 embalmed cadaverers. The ages at the time of death ranged from 43 to 95 years (average, 70.8 years). Each rotator cuff was carefully dissected, and the shoulders were classified into three groups: group 1 (intact cuff), group 2 (incomplete cuff tears or full thickness tears with longest diameter less than 5 cm), and group 3 (full thickness tears with longest diameter more than 5cm). The rotator cuff lesions were evaluated, and the intraarticular and intertubercular portions of the bicipital tendons were then examined. After thorough gross examinations, the entire tendinous portions of the LHB was removed. Then the bicipital groove was resected en bloc, including the transverse humeral ligament and the soft tissue attached to the lesser tubercle, that is, the entrance to the shoulder joint. For morphologic exaamination, a cross section 3-mm-thick was made of each resected bicipital groove and the attached soft tissue. The cross section was located at the most proximal portion of the bicipital groove adjacent to the articular cartilage. To investigate the shape of the sulcus, radiographs were obtained for each specimen using a soft X-ray unit. Each radiograph was analysed using the NIH Image program on a personal computer.

Results

There were 87 shoulders ingroup 1 (51.2%), 59 shoulders in group 2 (34.7%), and 24 in group 3 (14.1%). Overall, bicipital lesions were observed in 86 shoulders (50.6%) on gross examination. There was flattening of the intra-articular portion in 68 specimens (40.0%), fraying in 5 (2.9%), rupture or absence of the tendon in 5 (2.9%), adhesions to the bicipital groove in 6 (3.5%), and medial displacement over the lesser tubercle in 16 (9.4%). In the shoulders of group 1, although there was slight flattening of the LHB in the intra-articular portion in some joints (21.8%), there were no specimens with intertubercular lesions. More than half of the specimens (55.9%) in group 2 exhibited flattening of the LHB in the intra-articular portion, while 11.9% exhibited medial displacement of the LHB. In the group 3 specimens, two-thirds (66.7%) revealed flattening of the LHB, while fraying of the LHB in the intra-articular portion occurred in 16.7%. Of the group 3 specimens, 25.0% were intact in the intertubercular portion, while 20.8% exhibited adhesions to the bicipital groove, 37.5% medial displacement, and 16.7% rupture of the LHB. Rupture of the transverse ligament was not seen in any specimen with subluxation or dislocation of the LHB.

Discussion

Bicipital lesions are an important caurse of shoulder pain, however, the role of the LHB in shoulder motion is still controversial. Some biomechanical and clinical studies have clarified its role in stabilizing the humeral head in the glenoid during abduction of the shoulder. Enlargement or flattening of the LHB is often observed during cuff repair surgery; this might be the result of compensation for inadequate cuff function. In the present study, relative stenosis in the proximal portion of the bicipital groove was found mostly in the group 2 specimens. However, we could not demonstrate attrition of the medial wall of the sulcus in group 2. Therefore, we believe that bicipital tendinitis develops notfrom subacromial impingement but from relative stenosis of the bicipital groove at the entrance of the shoulder joint. This stenosis result from the increasing volume of the LHB. It appears that this increase inLHB volume occurs to compensate for insufficient cuff function. In group 3, such stenosis wasnot apparent because of the decrease in LHB volume. The decrease in LHB volume in group 3 appears tobe the wear and tear of theLHB caused by the tendon degeneration. In our series, the incidence of medial displacement of the LHB was higher in group 3 than ingroup 2. In group 3, 37.5% of the specimens showed medial instability of the LHB. The hight of the medial wall was decreased in the shoulders with massive cuff tears (group 3) due to the extent of the wear and tear involving the anterior part of the rotator cuff, including the soft tissue attached to the lesser tubercle, possibly leading to medial subluxation or dislocation of the LHB. However, these changes were not observed in group 2. Also, in shoulders with cuff tears, the hight of themedial wall was lower in specimens with LHB dislocation than in those without LHB dislocation. Interestingly, complete rupture of the transverse humeral ligament was not seen in any specimen, even in those with LHB dislocation. This suggests that the transverse

humeral ligament may not acts a major restraint to the medial instability of theLHB. Accordingly, we presumed that the supero-medial wall of the bicipital groove, including the soft tissue, was the major constraint preventing LHB subluxation.

We concluded that the long head of the biceps brachii muscle has the potential to compensate for inadequate rotator cuff function. Enlargement of the LHB, induced by increased compensatory activity for the rotator cuff dysfunction, and the loss of soft tissue in the bicipital groove, such as subscapularis tendon tear, may then lead to disorder of the bicipital gliding mechanism.