

CASE REPORT

Absence of the Long Head of the Biceps Tendon Associated With Glenoid Dysplasia and Posterior Labral Tear

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Anatomic variations in the insertion of the long head of the biceps tendon and superior labral complex have been described (Erickson et al. [1992] *AJR Am. J. Roentgenol.* 158:1091-1096; Kreitner et al. [1998] *AJR Am. J. Roentgenol.* 170:599-605; Mariani et al. [1997] *Arthroscopy* 13:499-501; Vangsness Jr. et al. [1994] *J. Bone Joint Surg. Br.* 76:951-954). To the authors' knowledge, there have been only five reported cases of congenital absence of the long head of the biceps tendon. Three of these cases were associated with anterior shoulder instability, one with a superior labral anterior posterior lesion and one simply with shoulder pain. This is the first reported case of congenital absence of the biceps tendon associated with glenoid dysplasia and a posterior labral tear. *Clin. Anat.* 21:728–732, 2008. © 2008 Wiley-Liss, Inc.

Key words: shoulder; biceps tendon; posterior labral tear; glenoid dysplasia

CASE PRESENTATION

An 18-year-old, right-hand dominant, male, high-school football player and wrestler presented with complaints of right shoulder pain and instability. The patient had a history of recurrent painless voluntary posterior shoulder subluxation since childhood. Several months prior to presentation, the patient had two separate episodes of traumatic posterior dislocation, each of which spontaneously reduced. The first episode occurred during a football game and the second occurred during a wrestling match. With each episode, the patient had his outstretched, adducted, and internally rotated arm forced posteriorly. The patient then complained of painful involuntary posterior shoulder subluxation, night pain, and inability to participate in sports. The pain was exacerbated by activities performed with an outstretched right arm, and weightlifting activities, especially the bench and military press. A 4-month course of physical therapy for rotator cuff strengthening and scapular stabilization was attempted but was unsuccessful.

On examination, there was no atrophy noted in the upper extremity or about the shoulder. There was full, symmetric range of motion of both upper extremities. Strength to manual muscle testing was normal bilaterally, especially resisted shoulder abduction and external rotation as well as elbow flexion

and supination. There was no evidence for prominence of the biceps muscle belly or "Popeye" sign (Kelly et al., 2005). Lift-off maneuver was negative and nonpainful. There were no signs of subacromial impingement. The anterior apprehension test was negative, and the patient had a 1+ sulcus sign (Altchek et al., 1991). Posterior apprehension test (Pollock and Bigliani, 1993) was positive for both pain and apprehension. There was Grade II laxity on load and shift testing (Hawkins et al., 1984). Speed's test (Bennett, 1998) was negative, there was no tenderness to palpation in the bicipital groove, and the jerk test (Kim et al., 2004) was positive.

A CT scan was performed and showed marked glenoid dysplasia, with 20° of glenoid retroversion, which was correlated with plain radiographs according to the method of Tetreault et al. (2004) (Fig. 1). MR arthrography demonstrated an extensive posterior labral tear with tiny associated paralabral cysts

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Fig. 1. Axial CT scan showing 20° of glenoid retroversion.

(Figs. 2A–2C). The intraarticular segment of the long head of the biceps tendon was absent with a shallow bicipital groove. The extraarticular segment of the biceps tendon was initially identified at the inferior aspect of the bicipital groove (Fig. 2D).

Arthroscopic examination, in the lateral arthroscopy position, confirmed complete absence of the intraarticular portion of the long head of the biceps tendon (Figs. 3 and 4). The superior labrum was without evidence of trauma to suggest biceps rupture (Fig. 3). Additionally, there was no evidence of a foramen for the long head of the biceps tendon in the rotator interval between the subscapularis and the supraspinatus tendons (Fig. 4). The patient had a very patulous posterior capsule and complex labral tear (from the 5:30 to the 11 o'clock position) which was partially detached from the posterior glenoid. There was no evidence of a Hill-Sachs or a reverse Hill-Sachs lesion. The patient underwent uncomplicated arthroscopic posterior labral repair with capsular shift using multiple bioabsorbable suture anchors. The patient was placed in an external rotation-type shoulder immobilizer (UltraSling ER; Donjoy, Vista, CA), which held the shoulder in 30° of external rotation. This was continued for 6 weeks after surgery during which time he was allowed to perform pendulum exercises and scapular retraction only. Between 6 and 12 weeks, progressive range of motion and light

pain-free strengthening were performed. Between 12 and 16 weeks, a more aggressive strengthening program was performed in preparation for the Army Physical Fitness Test (APFT) at 6 months post-op. At most recent follow-up, he was able to perform 30 push-ups in the office and had passed the APFT.

DISCUSSION

To the authors knowledge, there have been only five case reports documenting the absence of the long head of the biceps tendon (Mariani et al., 1997; Smith et al., 2002; Glueck et al., 2003; Franco et al., 2005; Keefe and Lowe, 2005). This is the first report on the absence of the intraarticular portion of the long head of the biceps tendon associated with glenoid dysplasia and symptomatic posterior instability.

Glenohumeral instability has been reported to affect 2% of the population (Hovelius, 1982). Posterior shoulder instability is a rarer condition that affects 2–5% of those patients who have glenohumeral instability (Arciero and Mazzocca, 2004). Posterior shoulder instability can be classified as traumatic or atraumatic. The traumatic type is much more common (Hawkins et al., 1984). Atraumatic posterior instability should alert the treating physician to a possible congenital abnormality, connective tissue

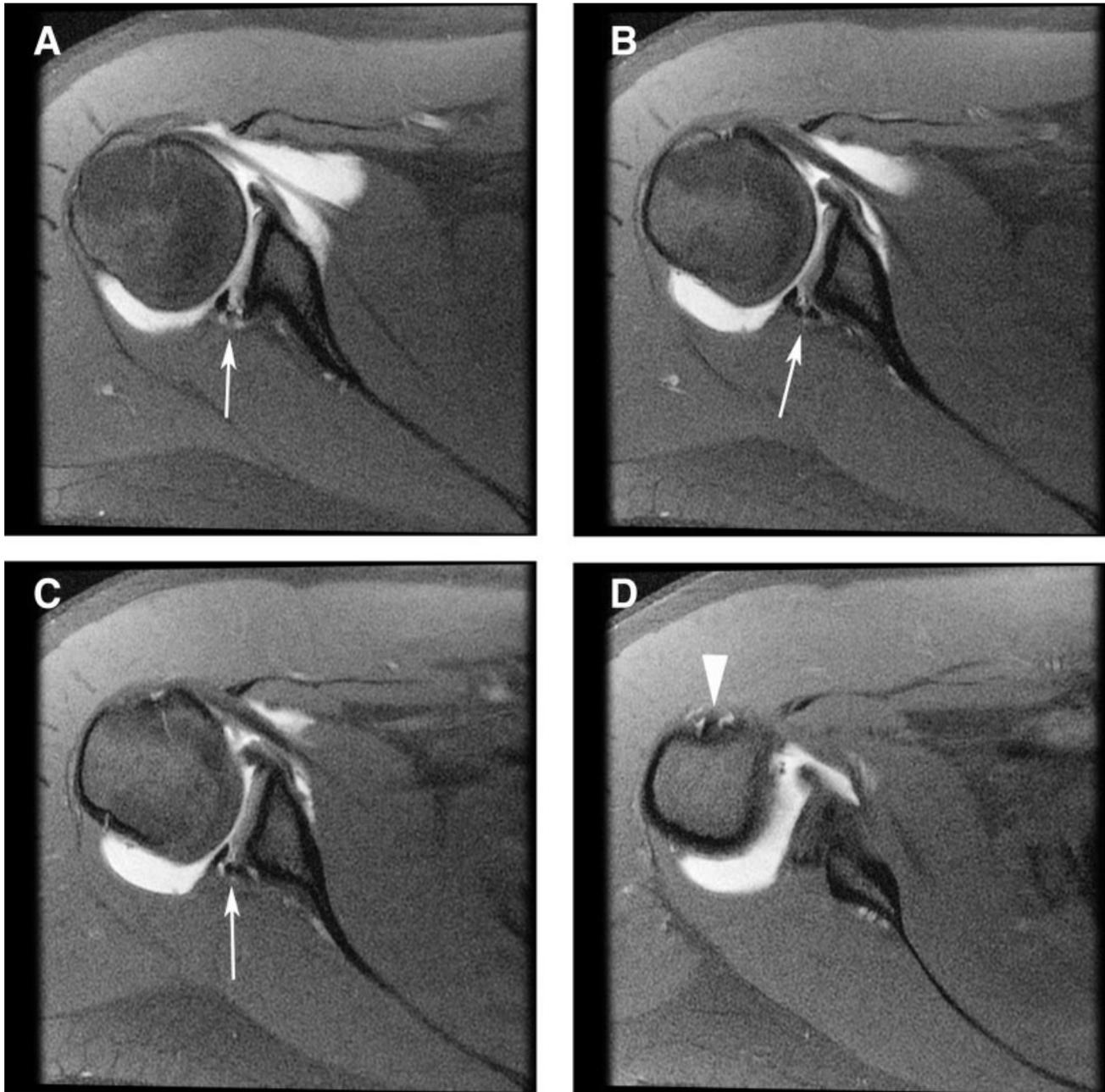


Fig. 2. Axial T1-weighted MR-arthrogram with fat-suppression. Sequential axial images from superior to inferior (**A–C**) demonstrate extensive tearing of the posterior labrum with tiny associated paralabral cysts (white arrows). The intraarticular segment of the long

head of the biceps tendon is absent and the glenoid is retroverted. The extraarticular segment of the biceps tendon is first seen at the inferior aspect of the bicipital groove several axial slices more inferiorly (**D**, white arrowhead).

disorder, or increased glenoid retroversion. Our patient had signs of both atraumatic and a later traumatic posterior instability.

The posterior instability in this case was due to several factors. Glenoid dysplasia is a known risk factor for posterior instability and undoubtedly played a role in the patient's voluntary subluxation episodes prior to his traumatic injury (Millett et al.,

2006). We believe the patient developed laxity of the inferior glenohumeral ligamentous complex and posterior capsule after many years of voluntarily subluxating his shoulder. The patient then developed pain following a traumatic injury to his posterior labrum resulting in symptomatic posterior labral detachment. Devgan et al. (2006) reported a case of a voluntary posterior subluxator which converted to a

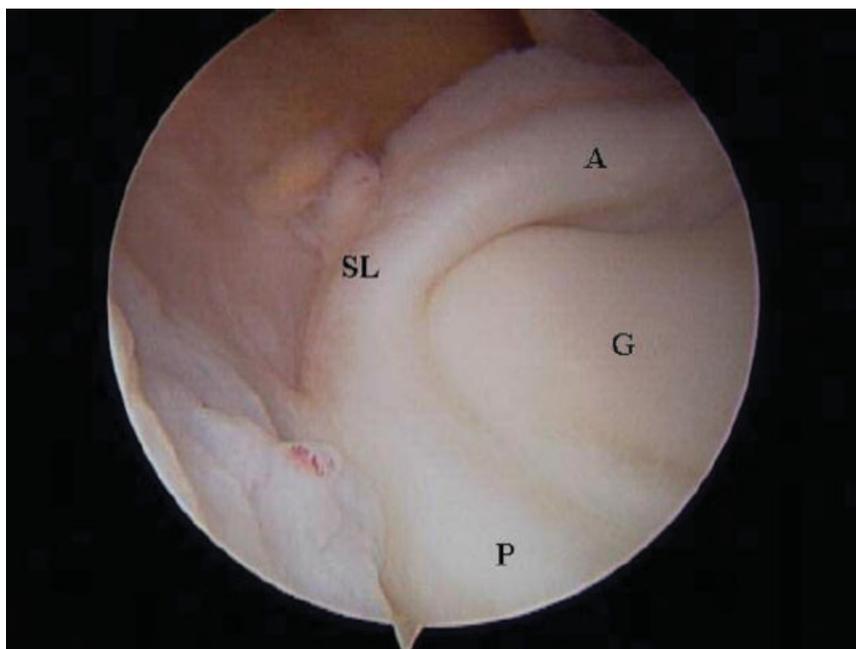


Fig. 3. Arthroscopic view of the superior aspect of the glenoid with the shoulder in the lateral position. Complete absence of the long head of the biceps tendon is noted. SL, superior labrum; A, anterior; P, posterior; G, glenoid.

traumatic instability pattern. The authors postulated that a combination of genetic predisposition and athletic microtrauma rendered the inferior glenohumeral ligament complex loose, which increased the patient's susceptibility to a traumatic posterior labral tear (Devgan et al., 2006). There was no mention of an absent intraarticular biceps tendon.

In contrast to the findings in our case, three of the five previously reported cases of absence of the long head of the biceps tendon have described anterior instability as the presenting clinical finding (Smith et al., 2002; Glueck et al., 2003; Franco

et al., 2005). Cadaveric and electromyographic studies have suggested an increased activation of the biceps in response to anterior glenohumeral translational forces (Itoi et al., 1993; Rodosky et al., 1994; Kim et al., 2001). Congenital absence of the long head of the biceps tendon may have contributed to the clinical findings of anterior instability in these three cases. A fourth case reported a Type II SLAP tear in association with absence of the long head of the biceps tendon (Keefe and Lowe, 2005). The authors concluded the causative factor for the superior instability and subsequent SLAP tear was the

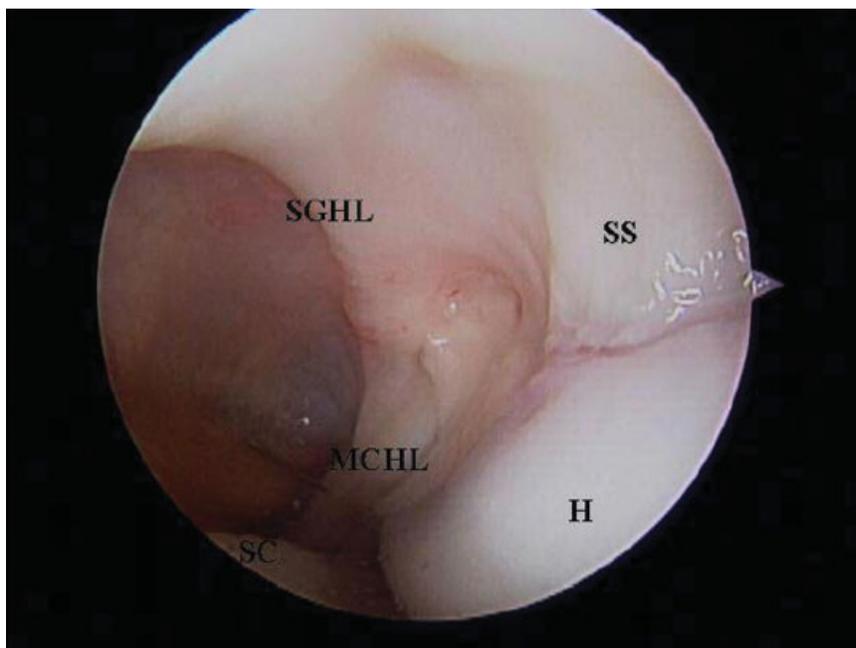


Fig. 4. Arthroscopic view of the rotator interval with the shoulder in the lateral position. Complete absence of the foramen for the long head of the biceps tendon is noted. MCHL, medial coracohumeral ligament; SS, supraspinatus; SC, subscapularis; SGHL, superior glenohumeral ligament; H, humeral head.

absence of the biceps tendon. Without the long head of the biceps tendon there was no humeral head depressor effect to help control superior humeral head translation (Keefe and Lowe, 2005). A recent report by Gaskin et al. (2007) described two cases of anomalous biceps tendons associated with anterior labral tears and instability. In both cases the long head of the biceps was present but hypoplastic.

It is unclear whether the absence of the long head of the biceps played a role in our patient's posterior instability. The biceps tendon has been proposed to play a dynamic protective role with respect to anterior instability by decreasing the stress placed on the inferior glenohumeral ligament complex (Rodosky et al., 1994). In a cadaveric study by Blasier et al. (1997), the long head of the biceps was found to both increase and decrease the resistance to posterior subluxation depending on the rotational position of the arm. Thus, the exact role of the long head of the biceps in posterior shoulder instability has not been fully elucidated.

Smith et al. (2002) reported a case of absence of the long head of the biceps in a patient with VATER syndrome, and suggested that when an absent long head of the biceps is encountered, a search for associated congenital anomalies should be performed. Our patient was noted to have bilateral glenoid dysplasia, also presumed to be a congenital anomaly. The long head of the biceps tendon is well formed by the seventh week of gestation. The tendon arises from the same anlage that forms the glenoid, labrum, and capsule (Giuliani et al., 1977). Given this, it is reasonable to assume that a congenital absence of the long head of the biceps tendon can be associated with glenoid dysplasia. Because of the relatively small numbers of reported cases, the exact cause and magnitude of such presumed associations remains unknown.

This case highlights a very rare clinical entity, the absence of the long head of the biceps tendon associated with glenoid dysplasia and posterior instability. The diagnosis was made utilizing standard imaging modalities, and confirmed at arthroscopy. It is likely that a combination of the patient's genetic predisposition and superimposed trauma led to the patient's symptoms. Patients with an absence of the long head of the biceps, although usually an isolated finding, could possibly have other minor congenital anomalies, as well as glenoid labral pathology.

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