



Biceps Variants and Shoulder Symptoms in Overhead Athletes

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Abstract

Background: Throwing athletes often present a painful shoulder due to several possible causes, including Superior Labrum Anterior to Posterior (SLAP) tears, biceps pathology, partial thickness rotator cuff tears, and “internal impingement” with an excessive external rotation, a Glenohumeral Internal Rotation Deficit (GIRD) and a posterior-inferior capsular contracture.

Methods: A narrative review about shoulder pathology in overhead athletes and LHB variants was performed and a case report of a tennis player with a biceps variant is presented.

Results: The tennis player recovered the full range of motion of the shoulder after releasing the limb of the LHB. This case highlights that a cause of pain and dysfunction in the tennis player can be associated to the anatomical variant of the LHB origin. The SPL-DO variant seemed to restrict the shoulder internal rotation in this throwing active patient. LHB anatomical variants should therefore be considered and treated in some overhead athletes, when identified and related to a significant clinical condition.

Conclusion: This case represents the first demonstration of the efficacy of releasing the limb of the LHB connected to the cuff, to regain the passive internal rotation.

Keywords: Shoulder; LHB variants; GIRD; Overhead athlete

Introduction

The cause of a painful shoulder in an overhead athlete is complex and controversial and its treatment is therefore challenging. It is also often difficult to distinguish a pathological lesion from a normal/adaptive one. Shoulder arthroscopy clarified several conditions of the disabled overhead athlete and showed many lesions, such as Superior Labrum Anterior to Posterior (SLAP) tears, biceps pathology, partial thickness rotator cuff tears, the “internal impingement” of the shoulder which is thought to be pathognomonic in overhead athlete pain. These conditions are, however, considered to be an end-stage in the disease spectrum caused by adaptation of the shoulder to overhead repetitive stress [1-3]. Most throwers exhibit an obvious motion disparity, whereby shoulder External Rotation (ER) is excessive and Internal Rotation (IR) is limited when measured at 90° of abduction. This loss of IR of the throwing shoulder has been referred to as Glenohumeral Internal Rotation Deficit (GIRD). Several investigators have documented that pitchers exhibit greater ER of the shoulder than do position players [4]. Throwing athletes often present the GIRD, with an excessive external rotation and a decreased Internal Rotation (IR) on the dominant shoulder, compared with the non-dominant side. GIRD has been regarded as a key component in abnormal throwing mechanics and injury risk. Posterior capsular tightness, which shifts the rotation center of the humerus postero-superiorly, was supposed to be a fundamental factor. Bony adaptation of the humerus was also related to this condition [5-8]. A SLAP lesion is often described in overhead athletes, such as swimmers and throwers, who can present an internal impingement (postero-superior) with a contact during the throwing between the articular side of the supraspinatus tendon and the postero-superior glenoid labrum [2,9,10]. A SLAP lesion can be associated to an AIOS (Acquired Instability in Overstressed Shoulder Surgery), which can be observed as minor instabilities in throwers or overhead workers. It is related to repetitive microtraumas that can produce degeneration or a dysfunction of the static stabilizing systems of the shoulder and a minor instability [5]. The anterior translation of the humeral head can produce a SLAP II lesion for a mechanism of traction on the biceps anchor [9]. The progressive detachment of the superior labrum known as “peel back” of the postero-superior labrum from the glenoid is a consequence of repetitive throwing microtraumas

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and can lead to a prevalent posterior SLAP II lesion with posterior component. In SLAP type II lesions, the biceps anchor peels off from the supraglenoid tubercle, with the associated detachment of the labrum, extending for a variable distance anteriorly and posteriorly; the biceps anchor can be displaced medially towards the glenoid neck on probing. A “peel back” of the anchor can be demonstrated by abduction and external rotation of the arm [1]. LHB is also known for several anatomical variations [11]. The overall incidence of variants is considered about 6.5%, divided into 4 groups: Mesotenon, Adherent, Split and absent [12]. Three case studies reported bilateral absence of the LHB tendon [13]. The LHB can be involved in several ways with isolated lesions or in combination with other pathological findings such as labral pathologies [14]. Also multiple reports of congenitally absent long head of biceps tendon have been reported in the literature. However there is no consensus on the clinical implications of this relatively rare entity [15]. Although the anatomic biceps variant may be benign, its presence might be associated with other shoulder pathology [16]. In the present paper we report a case of a painful shoulder in an overhead athlete, with an abnormal variant of the origin of the Long Head of the Biceps (LHB) [17]. It was assumed to be the primary cause of internal rotation deficit (GIRD) and pain during the overhead activities. The clinical outcome obtained by releasing the bicipital variant was optimal.

Case Presentation

A 28 year old male, right hand dominant tennis player complained of pain in his right shoulder for about 3 years, with worsening of the symptoms impairing him to play. The pain was described as achy, throbbing on the lateral and posterior area of the shoulder, especially during the service stroke. The patient did not report any dislocation, subluxation or any prior traumatic event. Connective tissue pathologies, such as Marfan syndrome, Ehlers-Danlos syndrome or Down syndrome, were clinically excluded. This research was conducted ethically according to international standards and this study meets the ethical standards of the journal.

Clinical examination

His right shoulder with fixed scapula had full passive range of motion in abduction and external rotation, while the passive Internal Rotation (IR) was only 10 degree. The passive motion of the left shoulder was normal, also for the internal rotation. Preoperative value of the ASES score was 77/120 and the Constant score was 73/100. Impingement signs (Neer, Hawkins, Yocum) were negative. Tenderness over the greater tuberosity, bicipital groove and lesser tuberosity were negative. He had no sulcus sign on both shoulders. In addition, O'Brien's test for SLAP lesion was negative, and relocation test of Castagna sign was negative [5]. Plain radiographs and Magnetic Resonance Imaging (MRI) scans did not reveal any obvious lesions, while MRI with contrast showed no fluid distention of the posterior capsule and a homogenous superior labrum. Neurological and vascular examination was negative too. Because of the worsening of pain, despite intense rehabilitation aiming also to recover IR and administration of NSAIDs, a shoulder arthroscopy was finally indicated.

Arthroscopic findings

Shoulder arthroscopy was performed in lateral decubitus and under general anesthesia. Examination under anesthesia showed passive Internal Rotation at 90-degree of abduction (IR2) limited at 20 degrees. The glenohumeral joint was examined routinely, including labrum, glenoid, humeral head, capsule and rotator cuff. Arthroscopy



Figure 1 (a+b): Right shoulder. The probe highlights the aberrant origin of the LHB.



Figure 2: With an arthrocare at hook, a release of the aberrant origin of the LHB is performed.



Figure 3: The passive motion in internal rotation is increased after the release of the aberrant limb.



Figure 4: Finally, a release of the postero-inferior capsule is performed.

revealed a split-type LHB variant, known as SPL-DO (split biceps with double origin), with one portion attached to the supraglenoid tubercle and the other limb presenting a separate attachment to the posterosuperior capsulolabral tissue 17 (Figure 1). The rest of the biceps tendon, labral complex, rotator cuff, chondral joint surface

were without any other atypical findings. Passive internal rotation at 30-degree of abduction in the scapular plane without traction was 10 degrees. Through the anterior portal, a 3-mm 90° Arthrocare was introduced into the joint to release the limb attached to the capsulolabral tissue (Figure 2). After releasing the mentioned limb, the shoulder passive Internal Rotation (IR2), which was measured by eliminating the traction of the arm, increased to 50 degrees (Figure 3). Nevertheless, the postero-inferior capsule was released from 6:30 to 8:00 and passive internal rotation angle was only slightly increased to 60 degrees (Figure 4). Postoperative forward flexion did not change from the preoperative values of 170°.

Rehabilitation protocol

After surgery, the shoulder was protected in a sling in neutral external rotation and 20 degrees of abduction for one month, while a passive motion of the shoulder was started after the first 3 weeks. After the first 5 weeks, active motion was allowed with a progressive program of gradual recovery of full strength. Competitive sport was allowed after the first 5 months. At 9 months after surgery, the athlete was completely asymptomatic.

Final follow-up

At final follow-up 3 years later, the ASES score was 118/120 and the Constant score was 100/100. Passive and active range of motion of the right shoulder was complete. The patient returned to its preinjury sport level. The patient gave us his informed consent for his clinical outcome to be treated in an anonymous way. The present studies have been approved by our ethics committee and have therefore been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and its later amendments.

Methods

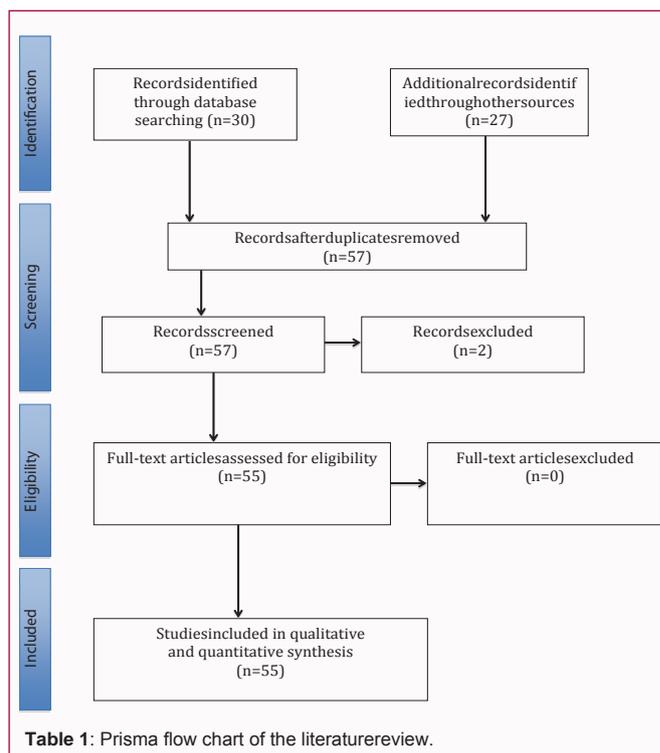
PubMed and EMBASE databases were searched to find a relationship between biceps variants and shoulder symptoms in overhead athletes, using the following keywords: Biceps variant, shoulder symptoms. A secondary search was performed by pearling the bibliography of all the full text articles obtained for additional reports. Pre defined inclusion criteria were used for abstract screening. Conference abstracts, narrative reviews and non-English articles were excluded from the review.

Results

The Keyword search identified 57 studies, out of which 55 studies were included for the final analysis (Table 1). Forty-nine were case series and 8 were case reports.

Discussion

Many articles in the literature associated GIRD and pain in athletes participating in overhead sports, however, no article reported the LHB variant as the primary cause of internal rotation deficit [8-10,17]. In this case, the release of the aberrant LHB origin, improved passive glenohumeral internal rotation of 50 degrees and the subsequent posterior capsular release provided a little additional gain (up to 60 degrees). Therefore, LHB variation seemed to be the primary factor of GIRD in this particular case. Dierickx et al. extensively examined the variations of the intra-articular portion of the LHB and proposed a classification of 12 types. In one type, well described in this case report, they included 9 cases, called the SPL-DO (split biceps with double origin) type. These were characterized by a Y-shape of the two splitted limbs of the LHB: One limb originated



from the inferior surface of the supraspinatus or from the medial capsular reflection at the superior glenoid and the other one, from the superior labrum or from the supraglenoid tubercle [17]. It is not clear if an absent LHB tendon is just an incidental finding or a pre-disposing factor to shoulder pain or instability. Lack of data on incidence of absent LHB tendon in asymptomatic shoulders compounds the problem [15]. Pal et al. based on a cadaveric study, reported a 25% incidence of actual tendon origin from the glenoid tubercle and a 70% incidence of the tendon blending with the posterosuperior glenoid labrum [18]. Gardner and Gray described the prenatal development of the shoulder joint in 65 human fetuses and embryos measuring 12 mm to 370 mm in length. In fetuses of 28 mm to 50 mm, the long head of the biceps tendon was separated from the joint cavity by a thin layer of tissue. The tendon was not seen freely in the joint cavity until a fetus reached 95 mm [19]. Most of LHB variants were considered to be benign and some researchers reported the existence of SPL-DO variants during a surgical treatment as a concomitant, accidentally found pathology. Wittstein et al. also reported two cases of SPL-DO variants arthroscopically observed in a 42-year-old male weight lifter and a 38-year-old female retired military officer [20]. However, they assumed that the variant LHB was a concomitant benign pathology and was not responsible for the patient's symptoms. In our case, IR restriction (GIRD) was solved after release of the limb adherent to the medial capsule. We assume that adhesion to the capsule might shift the rotation center of the glenohumeral joint and cause internal impingement, pain and dysfunction with throwing activity. Dierickx et al. reported that 5 of 9 cases of SPL-DO LHB type, showing a supraspinatus tear that needed repair [17]. They warned that this particular variant might be related to impingement in the young patient causing joint side cuff tears. Our case may suggest that SPL-DO variant restricted the glenohumeral motion, shifting the humeral head anterosuperiorly and causing internal impingement between the rotator cuff and the postero-superior rim of the glenoid [21-23]. It was difficult to

determine if this variant was congenital or acquired. Although many researchers agreed that these conditions are congenital as a result of partial detachment from the mesothelial or synovial fusion with the inferior surface of the capsule, repetitive overhead strains may cause partial detachment of the LHB and scarring to the surrounding tissue [24-27]. Alteration of the normal anatomical structure may prohibit the physiological motion of the LHB complex, such as "peel-back phenomenon". Isolated clinical reports of anatomic variants in the biceps have not linked aberrant anatomy of the biceps or its complete absence to a functional deficiency or pathologic condition [28]. The clinical picture at presentation is highly varied.

Conclusion

Although it is hard to conclude if a congenitally absent LHB tendon is a cause of shoulder pain or instability on its own, this case highlights that a cause of pain and dysfunction in the tennis player can be associated to the anatomical variant of the LHB origin. The SPL-DO variant seemed to restrict the shoulder internal rotation in this throwing active patient. LHB anatomical variants should therefore be considered and treated in some overhead athletes, when identified and related to a significant clinical condition.

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