Rotator Interval

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Abstract

Keywords
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► biceps tendon
► pulley lesion

The rotator interval is an anatomically complex region of the shoulder joint that is difficult to evaluate on clinical examination and by imaging. Abnormalities of its components may contribute to instability, shoulder stiffness, and pain and are challenging to diagnose and treat. This article gives an overview of the anatomy, MR anatomy, and normal variants of the rotator interval, together with basic technical aspects of MR imaging of this area. Pathologic conditions of the rotator interval capsule, the long head of biceps tendon, and the pulley system are reviewed and illustrated with several clinical examples.

The general dilemma of the shoulder as a ball-and-socket joint is to provide high mobility and, at the same time, maintain sufficient stability. This is reflected in the controversial pathoanatomical role of the rotator interval (RI), both as a dynamic stabilizer and also a structural weak spot, serving as a capsular reserve space and an outlet for the long head of biceps tendon (LHBT).[1,2] In experimental studies, a section of the RI leads to an increased anterior inferior and posterior translation of the humeral head, whereas surgical closure of the rotator interval limits external rotation, elevation, and extension.[3–5] Thus, although not generally agreed, the rotator interval structures are thought to contribute to joint stability, and consequently pathologic changes of these structures might affect glenohumeral stability and mobility.[1]

Imaging of the RI is mostly based on MR techniques. Normal anatomy and MR imaging findings in pathologic conditions involving the RI have been addressed by various review-type articles but only relatively few scientific studies. Therefore, in certain aspects, current practice is not fully based on reproducible results but also on personal experience.

This article further extends the list of review articles on the RI. With a focus on MR imaging and MR arthrography it discusses anatomical features, technical aspects, as well as pathology of the RI capsule, the LHBT, and the pulley system.

Anatomy

The rotator cuff has two tendinous gaps that are exclusively covered by capsular tissue: one anteriorly between the subscapularis (SSC) tendon and the supraspinatus (SSP) tendon, termed the *anterior rotator interval,* and one posteriorly between the supraspinatus tendon and the infraspinatus tendon, termed the *posterior rotator interval.* However, the term *rotator interval* usually refers to the anterior RI, and thus, to make things more simple, this convention is also assumed for this article.

The RI is a triangular shaped space bordered by the anterior free edge of the SSP tendon superiorly, the superior free edge of the SSC tendon inferiorly, and the coracoid process medially. The intertubercular sulcus and transverse ligament represent the lateral apex of this triangle, the coracoid process its base (►**Fig. 1**). The RI contains the intra-articular portion of the LHBT. It is covered by a fibrous capsule that represents an extension of the anterior joint capsule termed the *rotator interval capsule.* The RI capsule is reinforced by two ligaments: the coracohumeral ligament (CHL) on its bursal side and the superior glenohumeral ligament (SGHL) on its articular side (►**Fig. 1**). As the RI capsule forms the roof of the RI, its floor is formed by the articular cartilage surface of the humeral head.[6–9]

If viewed on sagittal sections (►**Fig. 1c**), the RI shows a quadrilateral configuration with the superior border of the SSC tendon inferiorly, the anterior border of the SSP tendon superiorly, the RI capsule anteriorly, and the humeral head posteriorly.[8] On midsagittal sections, the LHBT is located in the upper half of this quadrilateral.

Coracohumeral Ligament

As the most superficial layer of the RI capsule, the mediolaterally downsloping CHL has a broad origin from the lateral aspect of the base of the coracoid process. It forms two more or
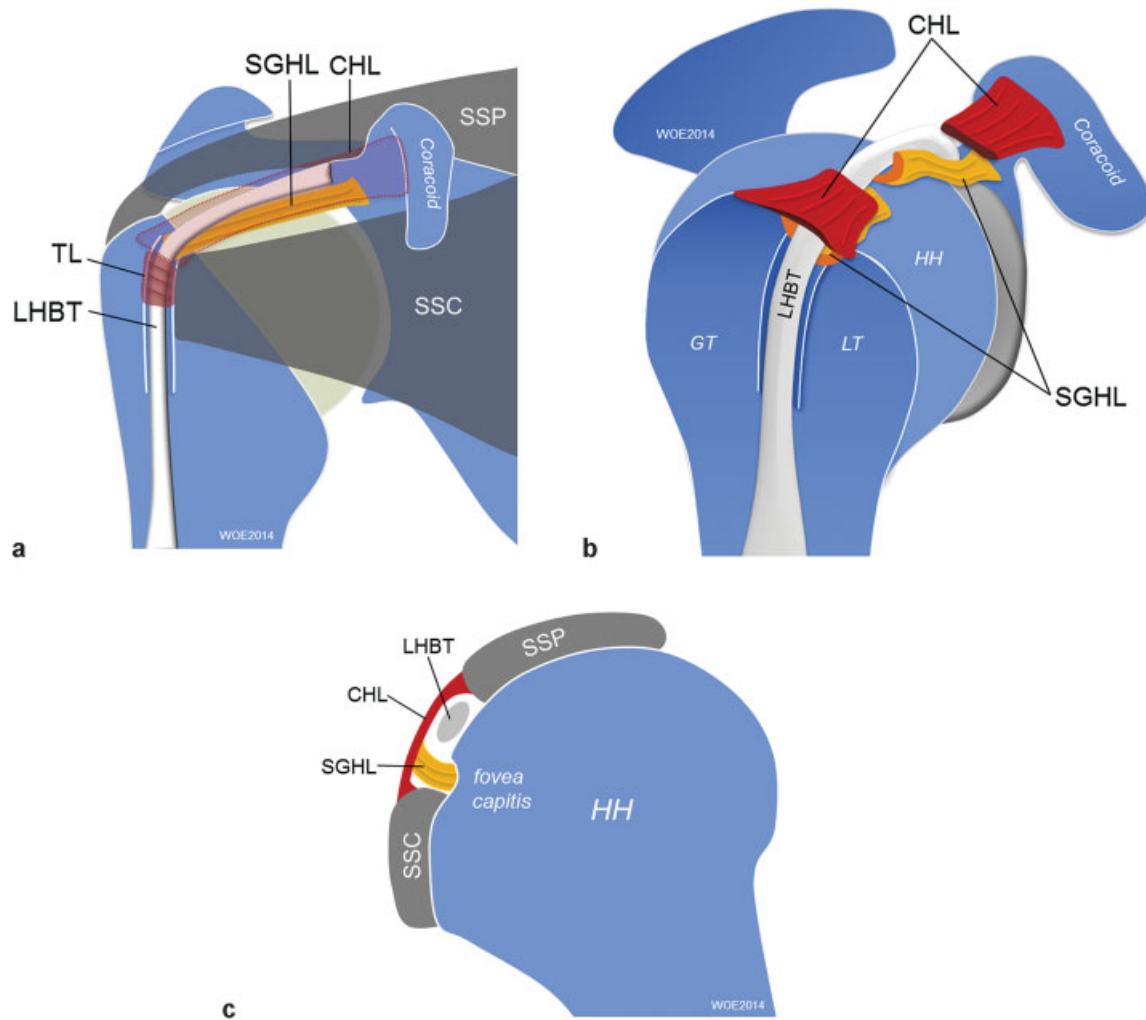
Fig. 1 Schematic drawings of the anatomy of the rotator interval. (**a**) Anterior view of rotator interval. (**b**) Anatomy of the pulley system. (**c**) Sagittal midsection through rotator interval. CHL, coracohumeral ligament; GT, greater tuberosity; HH, humeral head; LHBT, long head of biceps tendon; LT, lesser tuberosity; SGHL, superior glenohumeral ligament; SSC, subscapularis muscle and tendon; SSP, supraspinatus muscle and tendon, TL, transverse ligament.

less discrete bands that distally span the intertubercular (bicipital) groove of the humerus. The medial band of the CHL is smaller and blends with the SGHL distally. It crosses over the intra-articular LHBT and covers its medial and inferior aspect before it inserts onto the lesser tuberosity of the humeral head. The medial band merges with the RI capsule, the superior fibers of the SSC tendon, and the transverse ligament. The larger lateral band of the CHL surrounds the lateral and superior aspects of the intra-articular LHBT at the lateral portion of the RI (►**Figs. 1** and **2**). It merges with the anterior fibers of the SSP tendon and inserts to the greater tuberosity of the humeral head.[5–7,10,11] In most individuals the CHL can be identified as a well-developed anatomical structure that shows some variation in its thickness and the insertion sites of its different components.[5,10,12,13] The ligament is lax with the arm in internal rotation and adduction.[11]

On sagittal MR images obtained just lateral to the coracoid process, the medial portion of the CHL can be depicted as a broad linear structure of low signal intensity. More laterally it cannot be separated from the RI capsule.[11,12] The ligament may extend inferiorly over the anterior aspect of the SSC tendon (►**Fig. 3**).

Superior Glenohumeral Ligament

The SGHL typically originates from the supraglenoid tubercle of the scapula anterior to the LHBT. Possible variants are an origin from the superior glenoid labrum, the LHBT itself, the middle glenohumeral ligament (MGHL), or a combination of these. In the middle aspect and at the apex of the RI, the SGHL forms a U-shaped sling with the CHL around the LHBT, stabilizing the tendon as it passes through the rotator interval and changes its course from a horizontal to a vertical orientation (►**Figs. 1** and **2**).The ligamentous reinforcement formed by the combined fibers of the CHL and the SGHL has been termed the *biceps pulley, pulley sling,* or *reflection pulley of the biceps.*[8,13] The SGHL passes under the LHBT and inserts onto a small grove at the surface of the humeral head ("fovea capitis")
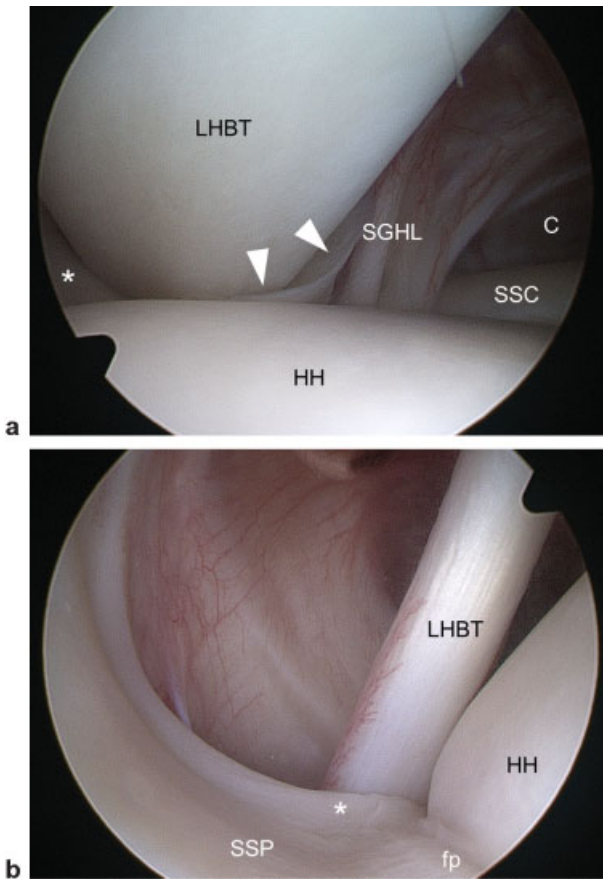
Fig. 2 Arthroscopic anatomy of the pulley system. (**a**) View from posterior portal over humeral head (HH) shows long head of biceps tendon (LHBT) and medial portion of pulley system with superior glenohumeral ligament (SGHL) forming the pulley sling (arrowheads). The superior border of the subscapularis tendon (SSC) and the inner surface of the rotator interval capsule (C) are seen more anteriorly. Asterisk shows lateral portion of pulley system. (**b**) More lateral view from posterior portal shows LHBT lying anterior to lateral portion of pulley system (asterisk) and supraspinatus tendon (SSP) before entering the bicipital groove. fp, footprint of supraspinatus tendon (Photographs: PD Dr. Sepp Braun, Sportorthopädie TU München; with permission).

just above the insertion of the SSC tendon onto the lesser tuberosity (►**Fig. 1c**), where it attaches to a thin slip from the superior SSC tendon.[6,7,11,14,15] By passing under the LHBT, the SGHL prevents it from inferior and medial dislocation in the RI. The SGHL represents a constant anatomical structure of variable shape and thickness.[5,13] It is often particularly well developed, if the MGHL is hypoplastic or absent. Congenital absence of the SGHL is rare, but anatomical variants, such as splits and duplications, are not uncommon.[6,7,11,15]

On sagittal MR images, the medial portion of the SGHL is seen as a round or linear intra-articular structure of low signal intensity that follows the LHBT, lying anterior to it. At the midportion of the RI, the SGHL fuses with the CHL to form a T-shaped structure. The SGHL is then interposed between the LHBT and the SSC tendon, where it can usually be seen as a curved structure with a striated appearance and a signal intensity slightly higher than that of the SSC tendon (►**Figs. 3a–d** and **4a**). Its insertion site at the fovea capitis is often visible as well (►**Fig. 3d**). On transverse images, the

SGHL is typically seen as a linear structure of variable shape and thickness arising from the anterior aspect of the supraglenoid tubercle and running anteriorly lateral to the coracoid process of the scapula. Redundancy of the ligament with the arm in neutral position represents a normal finding (►**Figs. 3e–f** and **4b**).[6,7,10,11]

Long Head of Biceps Tendon

The LHBT arises from the supraglenoid tubercle of the scapula and/or the superior glenoid labrum, takes an intra-articular horizontal course over the humeral head, traverses the RI, and enters the bicipital groove to become extra-articular and vertically oriented. The tendon passes the shoulder joint within a reflection of the synovial membrane and thus represents an intra-articular but extrasynovial structure. In the bicipital groove it lies under the transverse ligament surrounded by a tendon sheath that is formed by a simple extension of the joint capsule (►**Figs. 1** and **2**).[7,11,16] The pulley sling and the superior border of the SSC tendon are regarded as its main stabilizers and prevent the horizontal portion of the tendon from dislocating inferiorly and the vertical portion from dislocating medially.[1,10,14,15] In this system, the superior border of the SSC tendon is regarded as the most important structure for medial stability. Although controversial, the role of the transverse ligament as a stabilizer of the LHBT is probably negligible.[1,7,10,14,16] Whereas anatomical variations of the LHBT origin at the supraglenoid tubercle and glenoid labrum are quite common, variants of its intra-articular portion are not. Hypoplasia or congenital absence of the tendon, fusion with the rotator cuff, accessory synovial folds, and bifid tendons have been described. However, the clinical relevance of these rare anomalies is unknown.[16,17]

On MR imaging, the intra-articular portion of the LHBT can be best evaluated in the sagittal oblique plane (►**Fig. 3a–d**). The normal tendon is oval and homogeneously hypointense at cross section and shows a diameter of up to 9 mm. Toward the apex of the RI, it frequently appears more flattened. The groove portion, which is best seen on transverse images, usually appears more round and a little smaller than the horizontal part.[6,16,18] Because the tendon sheath communicates with the joint cavity, fluid around this portion of the LHBT should be considered a normal MR finding and not an indicator of tendon pathology.[7]

MR Imaging Techniques

On MR imaging, the structures of the RI can only be distinguished in the presence of joint fluid.[8,12] Therefore, MR arthrography (MRA) has been advocated as the modality of choice to evaluate this area of the shoulder.[7,10,11,15,19] At my institution, MRA is routinely used in cases where a lesion of the pulley system is clinically suspected. Sagittal T1-weighted arthrograms are preferably obtained without fat saturation to achieve high contrast between capsuloligamentous structures and extracapsular fat, on the one hand, and intra-articular contrast media, on the other hand. Furthermore, the MRA protocol includes coronal fat-suppressed T1-weighted and intermediate-weighted turbo spin-echo (TSE) sequences and a transverse T1-weighted TSE
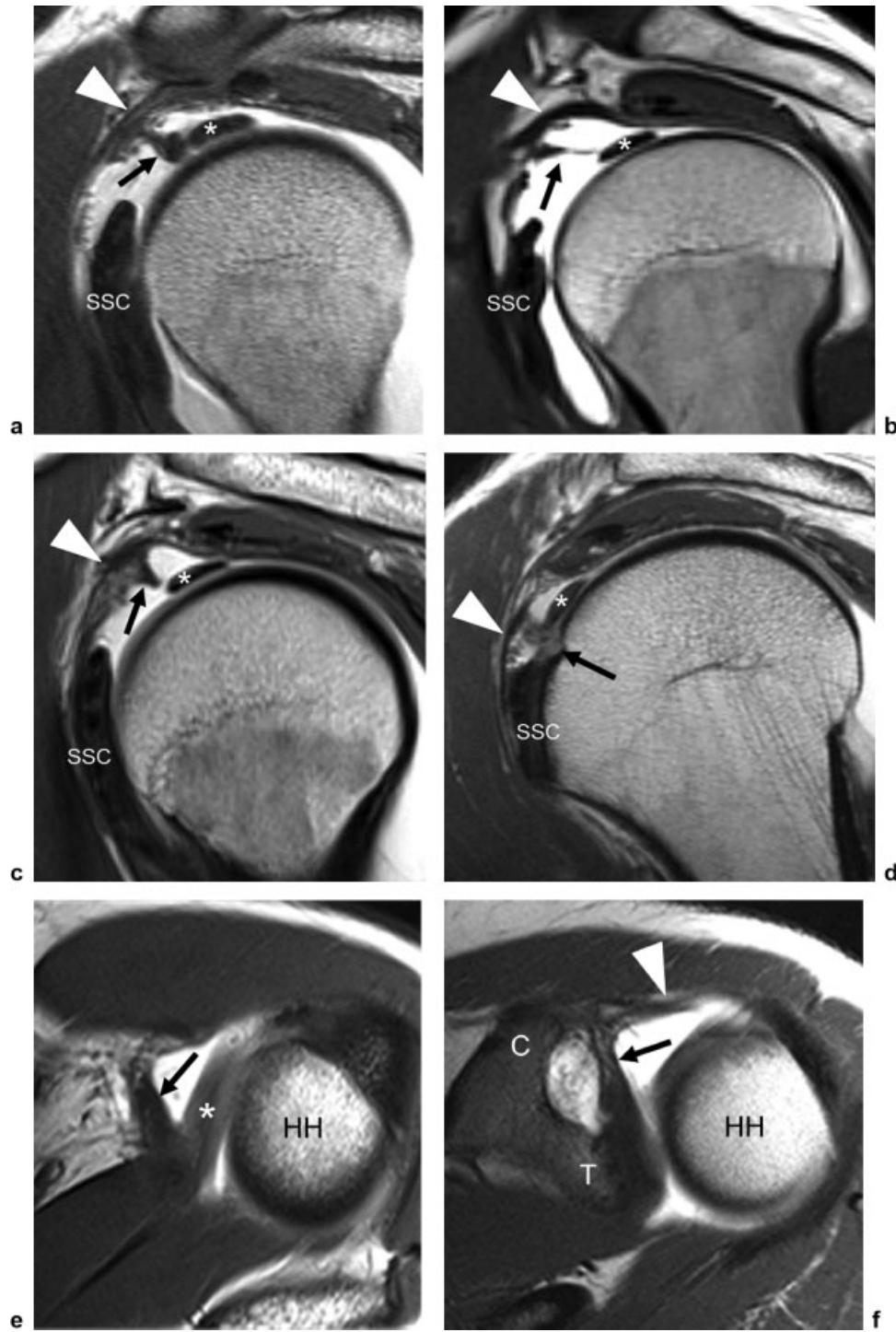
Fig. 3 MR anatomy of the rotator interval and pulley system. (**a–d**) Sagittal oblique T1-weighted MR arthrograms. Sections obtained at the medial aspect of the rotator interval just lateral to the coracoid (**a, b**) show the superior glenohumeral ligament (SGHL) (arrows) as a round (**a**) or linear (**b**) hypointense structure anterior to the long head of biceps tendon (LHBT) (asterisk). The coracohumeral ligament (CHL) is seen as a linear band at the outer aspect of the capsule (arrowheads). More medially the two ligaments unify to form one structure (**c**). A midsection through the interval (**d**) shows the pulley sling and insertion site of the SGHL (arrow). The CHL (arrowhead) extends inferiorly over the subscapularis tendon (SSC). (**e, f**) Consecutive transverse T1-weighted MR arthrograms obtained at the level of the superior aspect of the humeral head (HH) show the SGHL (arrows) originating from the supraglenoid tubercle (T) anterior to the LHBT (asterisk). The ligament runs anteriorly to fuse with the CHL (arrowhead). C, coracoid process.

sequence without fat suppression. If an anterior approach is used, contrast injection for MRA should be done carefully to avoid extravasation because extracapsular contrast material might be misinterpreted as a sequel of capsuloligamentous injury. Furthermore, care should be taken not to inject air because in the supine position air bubbles typically collect in the area of the RI and biceps tendon and hinder proper assessment of the anatomical structures.
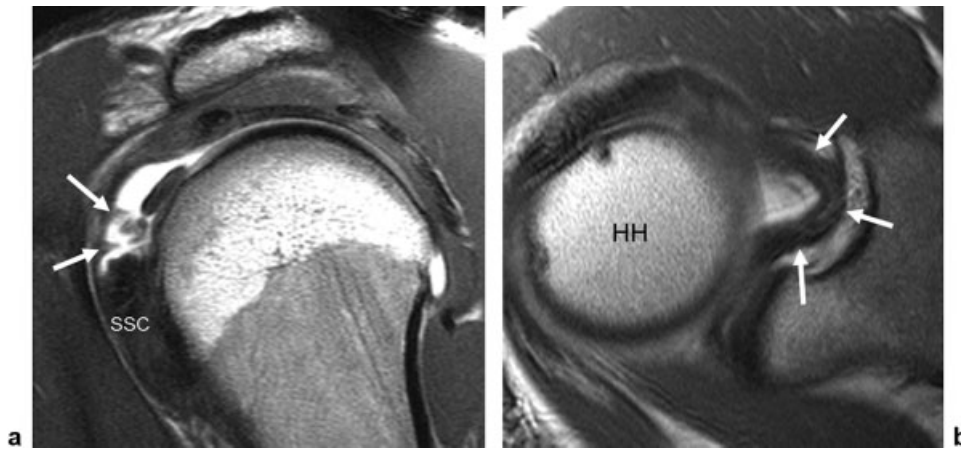
Fig. 4 Normal variants of the superior glenohumeral ligament (SGHL). (**a**) Sagittal oblique T1-weighted MR arthrogram shows duplication of the SGHL (arrows) at the level of the pulley sling. (**b**) Transverse T1-weighted MR arthrogram demonstrates a "cord-like" appearance of the SGHL (arrows) in an individual with congenital absence of the MGHL. HH, humeral head; SSC, subscapularis tendon.

On non-MRA examinations, the visibility of the RI structures markedly depends on the presence and amount of joint fluid. Due to their higher intrinsic contrast, intermediate-weighted TSE sequences with fat suppression should be used rather than T2-weighted or short tau inversion recovery sequences. At my institution, fat-suppressed intermediate-weighted TSE sequences with an echo time of 35 to 45 ms are obtained in all three imaging planes together with an additional coronal T1-weighted spin-echo sequence. A T1-weighted TSE sequence with a driven equilibrium pulse can alternatively be performed to achieve native arthrographic contrast.[20] Fat-suppressed T1-weighted sequences after intravenous contrast administration might be added to depict synovial proliferations or scar tissue in patients with adhesive capsulitis or rheumatic disease.

Regardless of the MR technique used, images should be obtained with a section thickness $\leq$ 3 mm and an in-plane resolution $\leq$ 0.5 $\times$ 0.5 mm.

Pathology of the Rotator Interval Capsule

Rotator Interval Changes in Adhesive Capsulitis

Adhesive capsulitis represents a common problem in general orthopedic practice.[11,21] It is a self-limiting disorder characterized by gradual loss of active and passive glenohumeral motion associated with severe pain that may lead to the clinical picture of a so-called frozen shoulder. The disease is more common in women, particularly between 40 and 60 years of age, but can occur in any age group.[6,21] Diabetes mellitus, thyroid disease, and autoimmune disease are believed to represent further risk factors.[21] The etiology of synovitis and capsular fibrosis in adhesive capsulitis is not fully understood. Its occurrence may be idiopathic without any initiating event or follow trauma or surgery. Inflammation and scaring typically involves the RI leading to a painful restriction of joint mobility especially in abduction and external rotation. Patients often also complain of pain at rest and at night, discomfort at the deltoid insertion, and tenderness in the area of the coracoid.[1] Spontaneous regres-

sion can be expected in most cases, but symptoms may be present for up to 2 years.[6,21] Most cases are treated conservatively by physiotherapy, systemic anti-inflammatory agents, or local steroid injections. Closed manipulation or surgical release are reserved for refractory cases.[1]

MR imaging for adhesive capsulitis is not generally utilized, particularly if patients present with the full clinical picture of the disease.[11] However, MR imaging can exclude underlying conditions that might otherwise be missed by clinical examination or point the way to the correct diagnosis in less severe cases with misleading clinical symptoms.[6,8] The RI and especially the CHL appear to be the central anatomical structures on both arthroscopic and radiologic evaluation.[1,6,22] In patients with adhesive capsulitis, several morphological changes involving the RI structures have been described on MRA including thickening of the CHL ($>$ 4 mm), thickening of the RI capsule ($>$ 7 mm), and synovial proliferation/scar tissue formation (►**Fig. 5**).[22,23] In a well-conducted study by Mengiardi and coworkers, complete replacement of the normal subcoracoid fat under the CHL by scar tissue seen on sagittal oblique arthrograms ("subcoracoid triangle sign") was the most specific finding (►**Fig. 5c**), whereas abnormalities of the CHL (signal intensity changes and/or contour irregularities) were most sensitive.[22] Other authors have studied the use of MR imaging after intravenous contrast administration in adhesive capsulitis and found that enhancing soft tissue proliferations in the rotator interval are a typical feature.[24–27] Correspondingly, ultrasound has been shown to allow identification of hypervascular hypoechoic tissue in the RI in patients who present with a frozen shoulder.[28]

Further imaging findings that have been described in adhesive capsulitis are a decreased capsular volume and shortening of the axillary recess on both conventional arthrography and MR arthrography,[22,29–31] as well as thickening, increased T2-weighted signal intensity, and contrast enhancement of the inferior joint capsule.[23,24,30–34] The latter criteria are less established due to the inconsistency of the data published in the literature.[22,24,30–34]
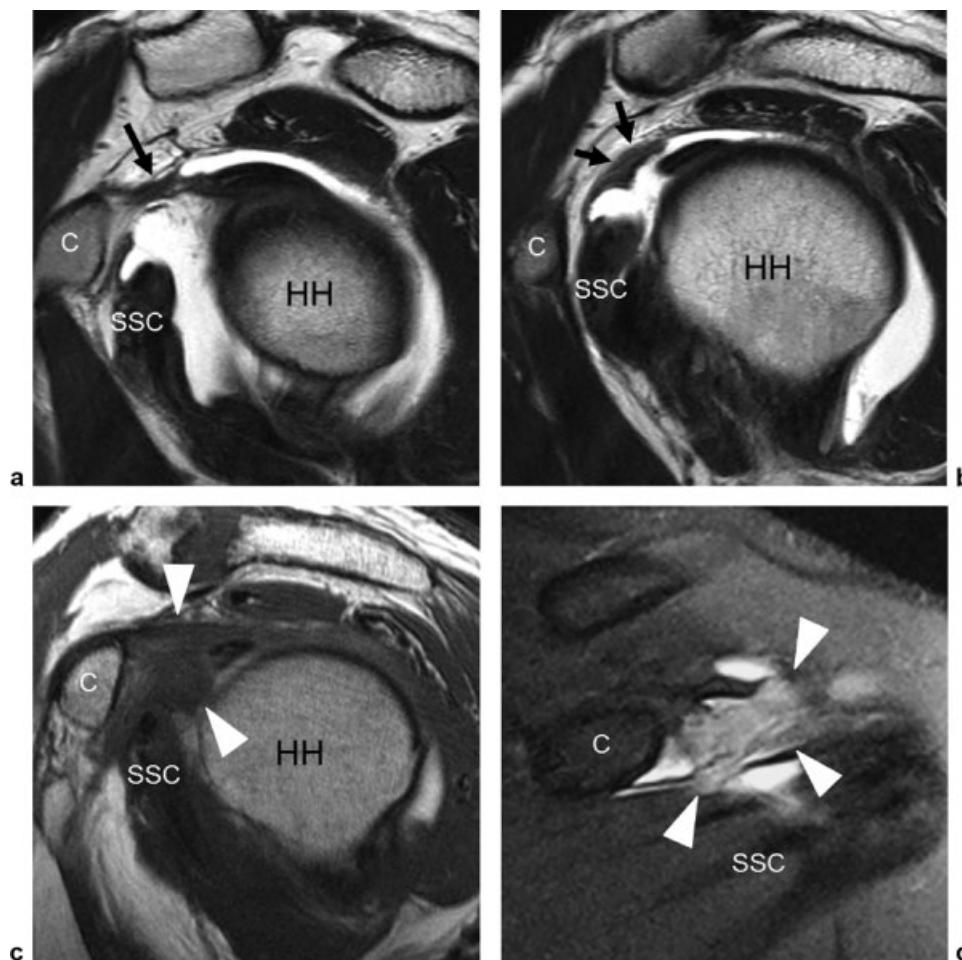
Fig. 5 Adhesive capsulitis: rotator interval findings. (**a, b**) Consecutive sagittal oblique T1-weighted MR arthrograms show thickening of the coracohumeral ligament (CHL) and rotator interval capsule (arrows). (**c**) Sagittal oblique T1-weighted MR arthrogram reveals complete obliteration of the fat triangle under the CHL (arrowheads) by hypointense scar tissue ("subcoracoid triangle sign"). (**d**) Corresponding coronal intermediate-weighted MR arthrogram with fat saturation demonstrates a soft tissue mass within rotator interval (arrowheads). C, coracoid process; HH, humeral head; SSC, subscapularis tendon.

Rotator Interval Laxity

The role of RI in shoulder instability is subject to an ongoing debate.[1,35] Although RI laxity may contribute to instability and pain,[1,3,5] it is unlikely to represent the only underlying pathology in most cases. Arthroscopic or open surgical closure of the RI, either alone or in combination with other stabilizing techniques, has been suggested as a means to treat different forms of shoulder instability, but the biomechanical effect of these procedures is doubted nowadays.[1,4,21,35]

The diagnosis of glenohumeral instability is mainly based on clinical history and physical examination.[6,36] Several studies have made attempts to quantify the dimensions of the RI on MR arthrograms of the shoulder (►**Fig. 6**) and to correlate these measurements with the results of clinical assessments.[35–38] Kim and coworkers found significant differences in RI dimensions between patients with chronic anterior instability and controls with stable shoulders.[37] Lee and coworkers reported significantly higher values for RI width in patients with multidirectional shoulder instability compared with controls.[38] These results could not be confirmed by Schaeffeler and coworkers, who did not observe any statistically significant differences in width and capsular outline of

the RI between patients with clinically established multidirectional instability and patients without shoulder instabilty.[36] The results of the latter study are in line with those of Provencher and coworkers, who found similar values for RI width in patients with anterior, posterior, or multidirectional instability and controls.[35] Possible explanations for these confusing and in some aspects contradictory results include differences in injected volume of contrast media and, consecutively, capsular distension at MRA, differences in shoulder rotation at imaging, inclusion of patients with different forms of shoulder instability, and the weaknesses of clinical assessment as the standard of reference.[36]

Rotator Interval Tears

Tears of the RI capsule can occur as an associated injury following acute traumatic anterior shoulder dislocation. Such defects typically show a horizontal orientation.[4,9,10] Le Huec and coworkers described isolated capsular tears at the RI as a sequel of shoulder trauma with forced internal rotation in a series of 10 patients with anterior shoulder pain.[39] However, little is known about the true incidence of such injuries, and their clinical relevance is probably low.

Fig. 6 Rotator interval laxity. Sagittal oblique T1-weighted MR arthrogram shows a relatively wide rotator interval (arrows) in a patient with multidirectional shoulder instability. SSC, subscapularis tendon; asterisk shows long head of biceps tendon.

The knowledge of MR imaging findings associated with a torn rotator interval capsule is so far more theoretical or based on personal experience with individual cases. Focal thinning, irregularity, and discontinuity of the RI capsule may be seen, particularly if MRA is used, but do not necessarily represent indicators of acute injury. Contrast material leaking from the rotator interval (►**Fig. 7**) or collecting in the subacromial/subdeltoid bursa in the absence of a rotator cuff tendon tear might represent more reliable signs.[8,40]

Biceps Tendon Pathology and Pulley Lesions

Pathologic conditions of the LHBT are a source of anterior shoulder pain that is often located over the bicipital groove. Pain may persist at rest and at night, be worse on rotation, and may radiate down the arm.[16] The most common causes are degenerative changes (tendinopathy), tendon instability, or a combination of the two.

Tendinopathy and Tear

In most cases, tendinopathy of the LHBT develops as an associated pathology in patients with subacromial impingement and degenerative rotator cuff lesions.[7,16] It can also occur as a consequence of chronic tendon instability or be observed as an isolated pathology caused by overuse in throwers or overhead athletes.[6,9,16,41] Tendinopathy typically affects the intra-articular (horizontal) portion of the LHBT. Initial intra-tendinous degeneration might be subtle or even invisible on inspection but might progress to fibrillation, splits, hypertrophy, or attenuation (partial tearing). Fusiform hypertrophy and the consequent inability to slide into the bicipital groove during elevation of the arm may cause entrapment ("hourglass biceps") and intra-articular buckling of the LHBT associated with pain and blocking of terminal elevation.[16,42] Rupture, as the end stage of this degenerative process, can occur with minor trauma or even during normal movement. The "Popeye sign" is the pathognomonic clinical presentation of a complete LHBT tear with distal retraction of the muscle belly. It may, however, be absent due to fibrous adherence or entrapment of the thickened tendon within the bicipital groove.[9,16]

The LHBT is difficult to evaluate on MR imaging. Tendinopathy of its intra-articular portion can cause an increased caliber and a more rounded shape on sagittal oblique sections, irregular contours, and increased signal intensity on short TE images (►**Fig. 8a, b**).[18,43] These findings may be subtle, and the
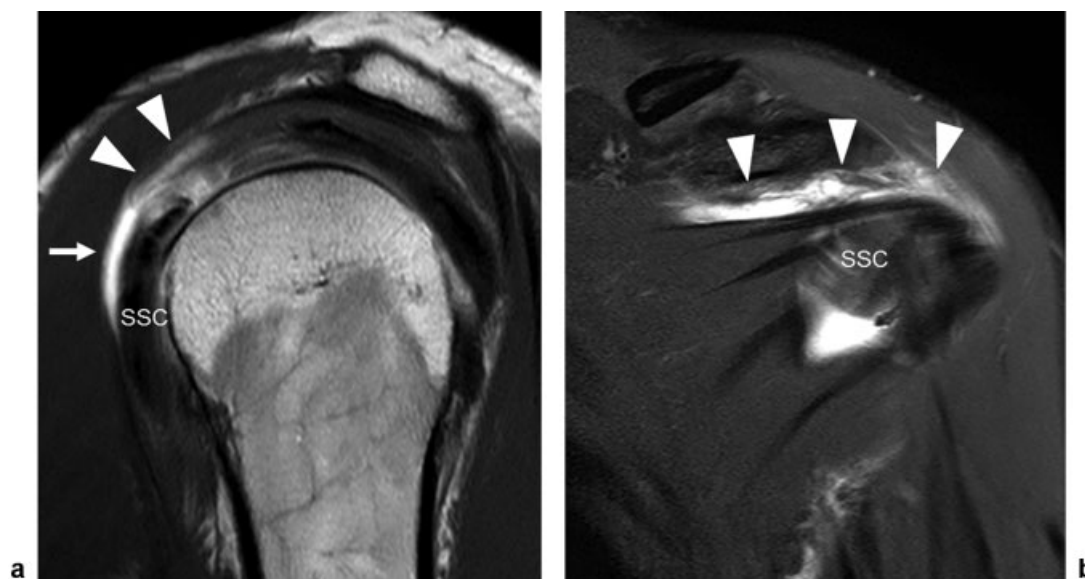

Fig. 7 Rotator interval tear. (**a**) Sagittal oblique T1-weighted MR arthrogram reveals defect of rotator interval capsule with contrast extravasation anterior to the subscapularis tendon (arrow) as an associated injury following anterior shoulder subluxation. A lesion of the anteroinferior labroligamentous complex was also present (not shown). (**b**) Corresponding coronal intermediate-weighted MR arthrogram with fat saturation demonstrates horizontal orientation of the capsular tear (arrowheads). SSC, subscapularis tendon.
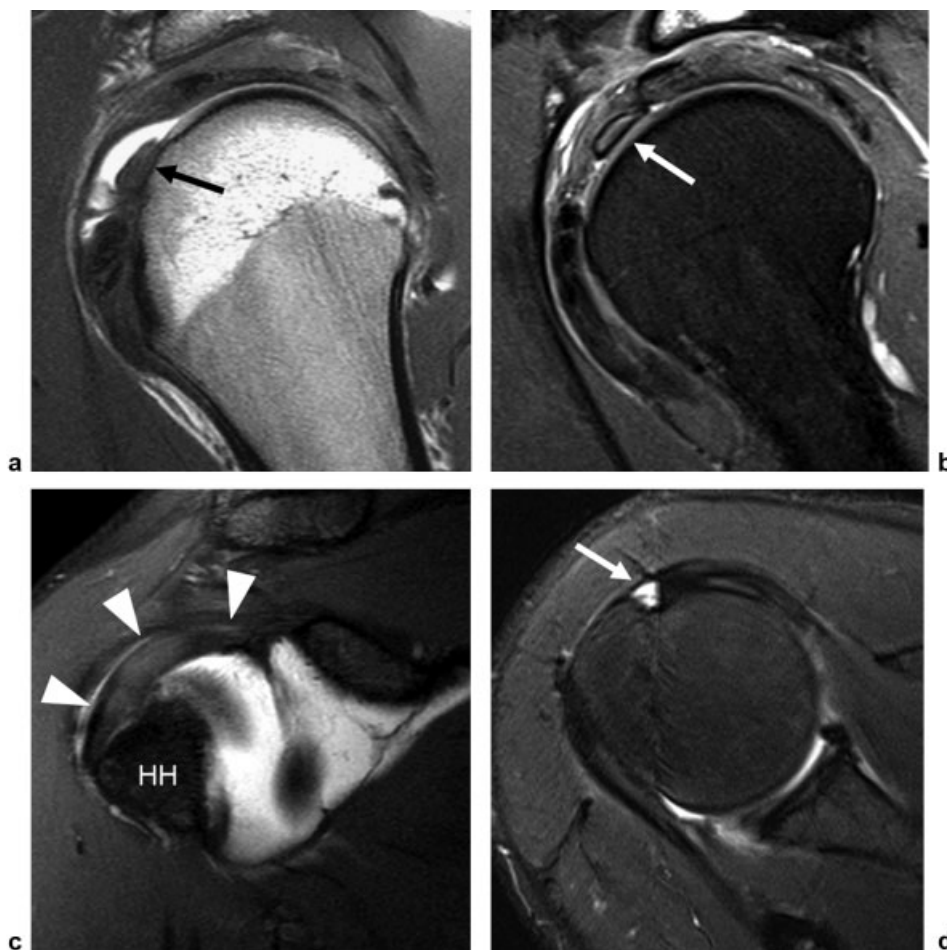
Fig. 8 Biceps tendon abnormalities. (**a**) Sagittal oblique T1-weighted MR arthrogram shows isolated tendinopathy in an overhead athlete with increased thickness and signal intensity of the intra-articular portion of the long head of biceps tendon (LHBT) (arrow). (**b**) Sagittal oblique intermediate-weighted turbo spin-echo (TSE) image with fat saturation demonstrates markedly increased signal intensity of LHBT (arrow) in a patient with rotator cuff disease. (**c**) Coronal oblique intermediate-weighted TSE image with fat saturation shows "hourglass" morphology with marked thickening and increased signal intensity of the entire intra-articular LHBT (arrowheads) until its entry into the bicipital groove. (**d**) Transverse intermediate-weighted TSE image with fat saturation shows "empty" bicipital groove (arrow) in a patient with complete tear of LHBT ("Popeye sign" on clinical examination).

diagnosis of tendinopathy is more reliable if more than one morphological abnormality is present. Magic angle effects are typically seen only at the most lateral aspect of the tendon just before it enters the bicipital groove and consequently can usually be differentiated from signal alterations secondary to tendon degeneration, particularly if the MR protocol includes an intermediate or T2-weighted sequence with fat suppression.[43] The typical morphology of the "hourglass biceps" may be identified on coronal oblique images (►**Fig. 8c**).[6] Partial tears can present with thickening and increased T1- and T2-weighted signal intensity as well as attenuation of the LHBT. Discontinuity of the horizontal portion or complete absence of visualization of the tendon are signs of complete rupture (►**Fig. 8d**).[9,18] In a study by Zanetti and coworkers, the overall sensitivity for detecting abnormalities of the LHBT on MR imaging was 89 to 92% and specificity was 56 to 81%.[18]

Medial Instability
Medial instability of the LHBT leading to tendinopathy, subluxation, or even dislocation can occur in isolation or in combination with other shoulder pathologies.[1] Subluxation is defined as a

partial or transient loss of contact, whereas dislocation represents complete and permanent loss of contact of the tendon with the bicipital groove.[16,44] Walch and coworkers described three basic forms of medial LHBT dislocation: intratendinous, intra-articular, and extra-articular (►**Fig. 9**).[44] All three forms are always associated with a pulley lesion and, except for the rare extra-articular subtype, with a subcapularis tendon tear.[16] In intratendinous dislocation, the LHBT cuts in between the CHL and the torn fibers of the superior edge of the SSC tendon. If the SSC tear progresses to complete detachment, the LHBT is allowed to dislocate intra-articularly. Extra-articular dislocation describes medial displacement of the LHBT superficial to the SSC tendon that can develop with a tear of the lateral CHL.[9,44] These basic patterns are also recognized in the more complex arthroscopic classification system suggested by Bennett (►**Table 1**).[45,46]

Pulley Lesions
Lesions of the biceps pulley and its adjacent structures can develop due to acute trauma, repetitive microtrauma, impingement, degenerative changes, and extension of rotator cuff tears over the RI.[1,7,15,47,48] With an overall arthroscopic
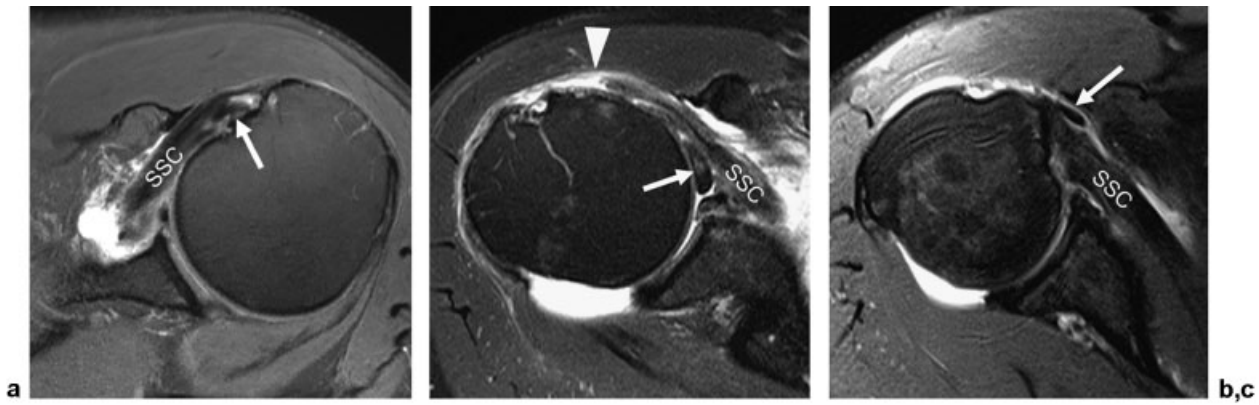
Fig. 9 Biceps tendon: medial dislocation patterns. (**a**) Transverse intermediate-weighted turbo spin-echo (TSE) image with fat saturation demonstrates (intratendinous) dislocation of the long head of biceps tendon (LHBT) (arrow) into a deep surface tear at the superior border of the subscapularis tendon. (**b**) Transverse intermediate-weighted TSE image with fat saturation shows intra-articular dislocation of the LHBT (arrow) in a patient with complete subscapularis tendon tear (arrowhead). (**c**) Transverse intermediate-weighted TSE image with fat saturation reveals extra-articular dislocation of LHBT (arrow) onto intact subscapularis tendon in a patient with full-thickness supraspinatus tendon tear (not shown). SSC, subscapularis tendon.

Table 1 Bennett classification of long head of biceps tendon instability[11,45,46]

Type	Direction of instability	Underlying structural lesion
1	Intrasheath subluxation	Isolated SSC tendon tear
2	Intrasheath subluxation	Medial CHL (CHL/SGHL) tear
3	Intra-articular dislocation	SSC tendon tear + medial CHL (CHL/SGHL) tear
4	Medial dislocation superficial to SSC tendon (extra-articular)	Anterior SSP tendon tear + lateral CHL tear
5	Intra-articular dislocation	SSC tendon tear + anterior SSP tendon tear + medial and lateral CHL tear

Abbreviations: CHL, coracohumeral ligament; LHBT, long head of biceps tendon; SGHL, superior glenohumeral ligament; SSC, subscapularis; SSP, supraspinatus.

prevalence of 7%, pulley lesions are not uncommon and frequently are found in association with rotator cuff defects.[15,47,49] They typically result in instability of the proximal LHBT and consecutive tendinopathy.[1,15,45,46,50] The term *hidden lesions* was coined by Walch and coworkers, who reported that on an open repair of SSP tears, defects of the pulley sling and superior border of the SSC tendon were invisible under the intact superficial fibers of the CHL until the RI was surgically opened.[44] Although it originally referred to open surgery, the expression has also been used in relation to the difficulty in diagnosing pulley lesions by clinical tests, imaging, and even arthroscopy.

Habermeyer and coworkers introduced a classification system that distinguishes four types of pulley lesions (►**Fig. 10**). A type 1 lesion is an isolated tear of the SGHL (pulley sling) with an intact superficial portion of the CHL. In types 2, 3 and 4, the SGHL tear is associated with a tear of the adjacent SSP tendon, SSC tendon, or both tendons, respectively.[49] It is important to note that 29 to 72% of all pulley lesions are isolated SGHL tears (type 1 lesions).[15,47,49,50] The preferred surgical treatment for pulley lesions is tenodesis and, if present, repair of an associated rotator cuff defect. The clinical outcome has been shown to be more favorable with isolated SGHL lesions than with combined defects.[47]
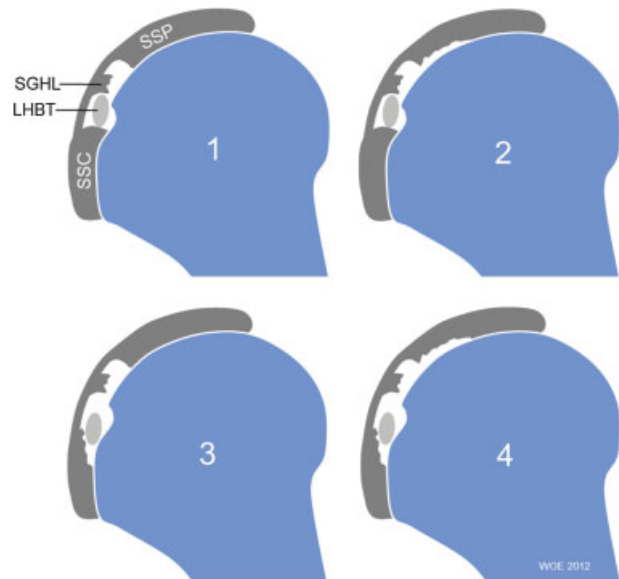

Fig. 10 Pulley lesions: Habermeyer classification (schematic drawing). 1. Isolated superior glenohumeral ligament (SGHL) tear. 2. SGHL tear and adjacent supraspinatus tendon (SSP) tendon tear. 3. SGHL tear and adjacent subscapularis tendon (SSC) tendon tear. 4. SGHL tear and adjacent SSP and SSC tendon tears. LHBT, long head of biceps tendon.
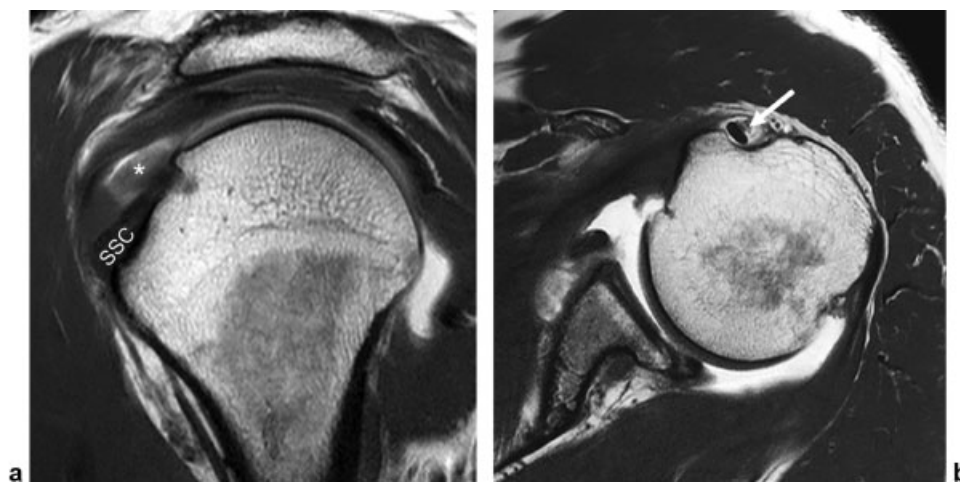
Fig. 11 Pulley lesion: type 1 (isolated superior glenohumeral ligament [SGHL] tear). (**a**) Sagittal oblique T1-weighted MR arthrogram demonstrates the "displacement sign." The horizontal portion of the long head of biceps tendon (LHBT) (asterisk) is displaced inferiorly onto the subscapularis tendon on a midsection through the rotator interval. The SGHL is not visible. Note markedly increased caliber and signal intensity of LHBT due to tendinopathy. (**b**) Corresponding transverse T1-weighted MR arthrogram shows an unremarkable vertical portion of the LHBT (arrow) centered in the bicipital groove. SSC, subscapularis tendon.

The value of MR imaging in the detection of pulley lesions was studied by Schaeffeler and coworkers and Weishaupt and coworkers. In both studies, the diagnostic performance of MRA was retrospectively analyzed with arthroscopy as the standard of reference.[15,19] Weishaupt and coworkers found an overall sensitivity of 86 to 93% and specificity of 80 to 100% with abnormalities of the superior border of the SSC tendon as the most accurate indirect finding. The direct assessment of the SGHL and CHL did not prove helpful in this study. The authors concluded that differentiation from an isolated SSC tendon lesion might therefore be impossible.[19] In the more recent study by Schaeffeler and coworkers, MRA performed comparably well in diagnosing pulley lesions with a sensitivity of 82 to 89% and a specificity of 87 to 98%; however, the diagnostic criteria were reevaluated. The most accurate criteria in their study were the "displacement sign," nonvisibility or discontinuity of the SGHL, and tendinopathy of the intra-articular LHBT on sagittal oblique MR arthrograms (►**Fig. 11**). The "displacement sign" describes caudad displacement of the horizontal portion of the LHBT leading to direct contact with the SSC tendon on a sagittal oblique midsection through the RI caused by the lack of inferior support of the LHBT by the torn SGHL (►**Fig. 11a**). Medial subluxation or dislocation of the vertical portion of the LHBT on transverse images and the presence of an adjacent SSC tendon tear proved to be highly specific but insensitive signs of a pulley lesion.[15] On MR imaging, a normal position of the LHBT within the bicipital groove does not therefore exclude a pulley lesion (►**Fig. 11b**), particularly if it represents a Habermeyer type 1 or 2 lesion.[15] This statement is in line with the arthroscopic observation by Braun and coworkers, who described that the LHBT remained centered in the bicipital groove in two thirds of pulley lesions when the arm was in a neutral position.[50] If, however, the LHBT appears medially subluxed or even dislocated on transverse MR images, the diagnosis of a pulley lesion is straightforward, but the SSC tendon as the most powerful medial stabilizer is probably injured as well.[9,15] Due to the close association of instability and tendinopathy, a lesion of the pulley sling is unlikely in the absence of degenerative changes of the intra-articular LHBT.[15]

Lateral Instability

Lateral instability of the LHBT is very rare. It has mainly been described in the context of greater tuberosity fractures. Posttraumatic deformity of the humeral head with flattening of the bicipital groove and anterior contour of the greater tuberosity may allow the LHBT to sublux or dislocate laterally. At times, dynamic lateral instability of the LHBT can also be found in association with SSP tendon tears. The tendon may then roll over the lateral rim of the bicipital groove when the arm is in abduction and internal rotation.[16]

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