8 Imaging Diagnosis of SLAP Tears and Microinstability

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8.1 Normal Labrum

8.1.1 Anatomy and Biomechanics

The glenohumeral joint is the most mobile joint in the body. Static and dynamic stabilizers play a vital role in maintaining the stability of the shoulder, negotiating the fine balance between physiologic mobility and pathologic laxity. The glenoid labrum is an important static stabilizer of the glenohumeral joint, consisting of a ring of fibrous and fibrocartilaginous tissue along the glenoid rim. The bulk of the labrum consists of dense fibrous tissue and collagen with a small amount of fibrocartilage at the chondrolabral junction [1, 2].

The labrum serves to increase the depth and surface area of the glenoid fossa, contributing 50% of the glenoid fossa depth and increasing the surface area of the glenoid by approximately one-third [3, 4]. In conjunction with intra-articular fluid, the labrum also creates a suction effect on the humeral head, helping to maintain the humeral head centered in the glenoid cavity [5]. It functions as a bumper-like mechanism to help protect the articular cartilage from compression and shear damage [1]. Perhaps even more importantly, the glenoid labrum allows other glenohumeral stabilizers to function by providing an attachment site for the glenohumeral ligaments and long head of the biceps tendon (LHBT).

The glenoid labrum can have a wide range of shapes. A study by Park and colleagues looking at labral morphology on MR arthrograms in asymptomatic volunteers found that triangular (64% anteriorly, 47% posteriorly) and round (17% anteriorly, 33% posteriorly) shapes were the most common [6]. Flat, cleaved, notched, or absent labral morphologies were also seen. Significant variability also exists in labral size, ranging from 2 to 14 mm in normal individuals [7]. Normally the labrum is larger at its superior and posterior aspects, compared to the inferior and anterior aspects [8]. The labrum typically has low signal intensity on all MRI sequences. However, increased linear or globular signal has been described in up to a third of arthroscopically normal labra [7].

The glenoid labrum is conventionally divided into four quadrants—anterosuperior, anteroinferior, posterosuperior, and posteroinferior—by a horizontal line bisecting the labrum into superior and inferior halves and a vertical line bisecting

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the labrum into anterior and posterior halves. The labrum is also commonly divided into a clock face, with 12 o'clock designating superior and 3 o'clock anterior.

Significant variations exist between the labral quadrants, both in the incidence of labral pathology and normal variants. The posterior and inferior portions of the labrum are most firmly attached to the glenoid [9, 10]. This helps explain the preponderance of variants in labral attachment found in the superior and anterosuperior portions of the labrum. The superior labrum, and particularly the anterosuperior quadrant, is the site of attachment of the LHBT and most of the glenohumeral ligaments. The LHBT attaches at the level of the supraglenoid tubercle at approximately the 12 o'clock position. Both the superior (SGHL) and middle (MGHL) glenohumeral ligaments attach to the anterosuperior labrum. The anterior band of the inferior glenohumeral ligament (IGHL) has traditionally been thought to arise from the anteroinferior labrum. However, a recent cadaveric study by Ramirez Ruiz and colleagues found high origin of the anterior band of the IGHL at or above the 3 o'clock position in four of ten cadaveric shoulders [11]. The intimate relationship between the labrum and these vital capsular structures partly accounts for the disproportionate amount of pathology that occurs in the superior and anterosuperior labrum.

8.1.2 Superior Labral Variants

The superior and anterosuperior labrum are common sites for labral anatomic variants.

These same locations are also common sites for labral pathology, making the distinction between pathology and anatomic variant both difficult and clinically relevant. Knowledge of the array of anatomic variants that may occur here is crucial to avoid mistaking them for labral abnormalities.

8.1.2.1 Cartilage Undercutting

The glenoid hyaline cartilage may sometimes undercut the deep portion of the superior labrum, creating an extended chondrolabral interface (Fig. 8.1). This variant has been found in up to 32% of asymptomatic shoulders [6]. Superficially, this may resemble a superior labrum anterior-posterior (SLAP) tear. However, the cartilage has intermediate signal similar to the rest of the glenoid articular cartilage, compared to the high-signal-intensity fluid (on T2-weighted images) or gadolinium (on T1-weighted MR arthrogram images) insinuating into a labral tear. The cartilage also parallels the contour of the glenoid rim, unlike a SLAP tear, which typically curves laterally, away from the glenoid [12–14].

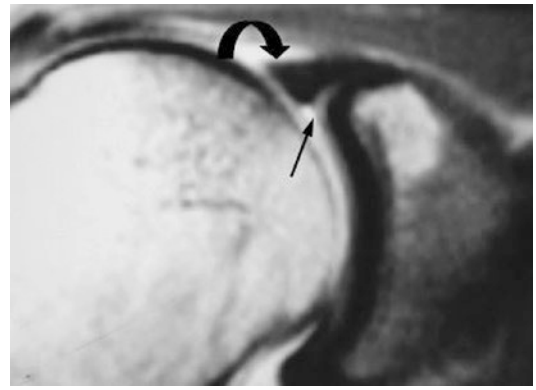

Fig. 8.1 Cartilage undercutting. Coronal proton density image demonstrates glenoid hyaline cartilage (arrow) undercutting the deep portion of the superior labrum (curved arrow). The cartilage parallels the contour of the glenoid rim and shows similar intermediate signal intensity to the rest of the glenoid articular cartilage

8.1.2.2 Sublabral Recess

The sublabral recess or sulcus is a small cleft found between the biceps labral complex and the glenoid cartilage (Fig. 8.2b, c). It is the most common anatomic variant of the superior labrum [15], present in up to 73% of shoulders and deeper than 2 mm in 39% [16, 17]. Like cartilage undercutting of the labrum, it can also be confused for a SLAP tear. However, a smooth contour cleft that parallels the curvature of the glenoid rim suggests a sublabral recess rather than a SLAP tear [14, 18, 19]. Although initially thought to never extend posterior to the LHBT insertion [9], studies have shown that a sublabral recess can extend posterior to the LHBT insertion in the absence of a SLAP tear [17, 20].

8.1.2.3 Biceps Labral Complex

Three distinct types of biceps labral complexes (BLC) have been described (Fig. 8.2) [10]. In

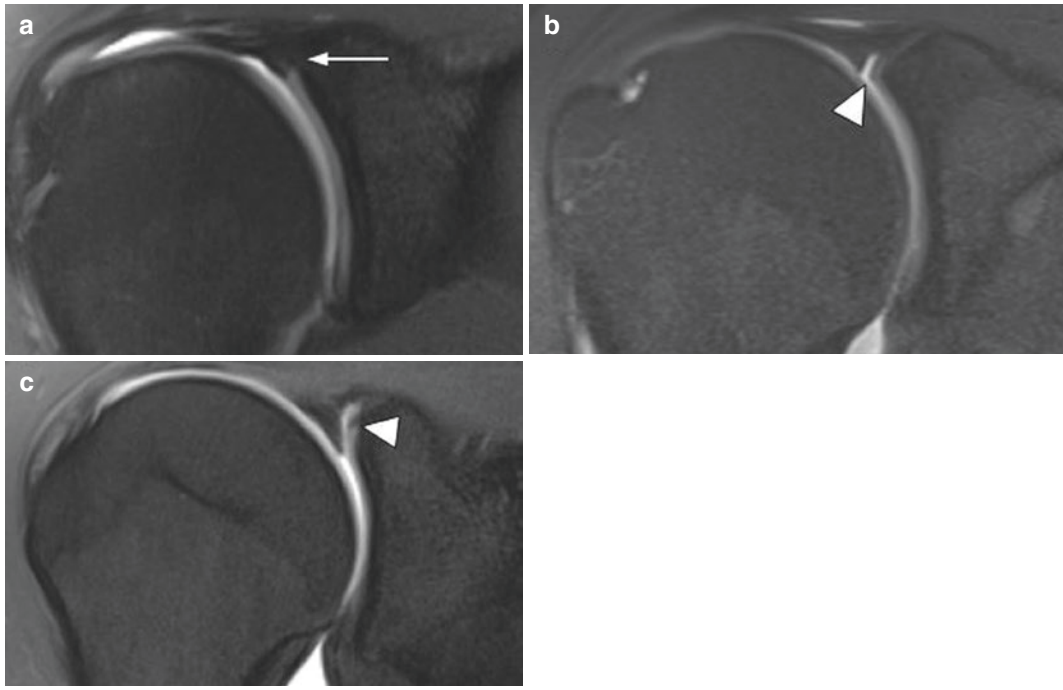

Fig. 8.2 Biceps labral complex (BLC). Coronal fat-suppressed T1-weighted MR arthrographic images demonstrate the three distinct types of biceps labral complexes. (**a**) Type I BLC with firm attachment of the labrum to the underlying glenoid cartilage without cartilage undercutting or sublabral recess (arrow). (**b**) Type II BLC with the labrum projecting slightly more medially over the glenoid articular cartilage and a small sublabral recess paralleling the contour of the glenoid (arrowhead). (**c**) Type III BLC with a meniscoid labrum projecting into the joint space and accompanied by a deep sublabral recess (arrowhead)

type I BLC, the labrum is firmly attached to the glenoid without cartilage undercutting or sublabral recess present. In type II BLC, the labrum projects more medially over the glenoid articular cartilage and there is a small sublabral recess paralleling the contour of the glenoid. In type III BLC, a prominent triangular meniscoid labrum projects into the joint space and is accompanied by a deep sublabral recess.

8.1.2.4 Bicipital Labral Sulcus

A shallow cleft can sometimes be found on the undersurface of the proximal intra-articular biceps tendon at the junction with the superior labrum (Fig. 8.3). This bicipital labral sulcus has been reported to have a prevalence of 30% on MR arthrography [6].

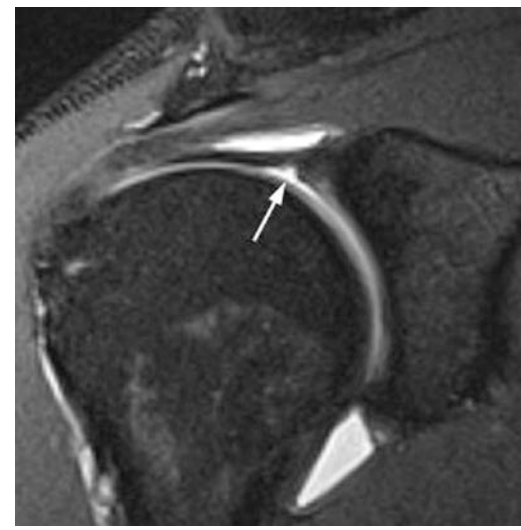

Fig. 8.3 Bicipital labral sulcus. Coronal fat-suppressed T1-weighted SPACE image from a direct MR arthrogram demonstrates a shallow cleft on the undersurface of the proximal intra-articular biceps tendon at the junction with the superior labrum (arrow)

8.1.2.5 Buford Complex

An absent or hypoplastic anterosuperior labrum accompanied by a thickened cordlike MGHL is

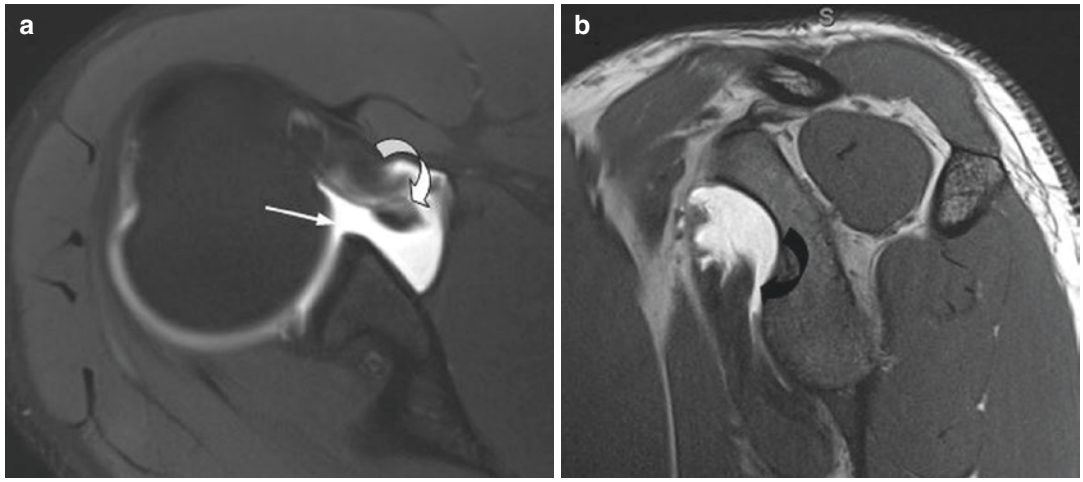

Fig. 8.4 Buford complex. (**a**) Axial fat-suppressed T1-weighted MR arthrographic images demonstrate an absent anterosuperior labrum (arrow) and a thickened cord-like MGHL (curved arrow). (**b**) Sagittal T1-weighted image from an MR arthrogram confirms a thickened MGHL (curved arrow) coursing deep to the subscapularis tendon

known as a Buford complex (Fig. 8.4). This relatively uncommon entity has been reported in 1.5–7.4% of patients [21, 22]. The Buford complex can sometimes be mistaken for a displaced labral tear. This pitfall can be avoided by following the thickened MGHL to its insertion on the humeral neck or as it blends with the anterior joint capsule beneath the subscapularis tendon. Correlating with the sagittal images is also important since the thickened MGHL can be well appreciated in the sagittal plane (Fig. 8.4b).

8.1.2.6 Sublabral Foramen

A sublabral foramen is a focal detachment of the anterosuperior labrum between the 1 o'clock and 3 o'clock positions (Fig. 8.5) [9]. It can be seen in up to 18.5% of shoulder arthroscopies and has been described in association with a thickened MGHL [23]. Although generally thought not to extend below the level of the midglenoid notch or glenoid equator, Tuite and colleagues have reported that a sublabral foramen may indeed extend to the anteroinferior labral quadrant [24]. The features distinguishing a sublabral foramen from a labral tear include focal detachment of the anterosuperior labrum without involvement of the biceps origin, labral displacement less than 1–2 mm, and a smooth labral contour [15]. Interestingly, although a sublabral foramen in and of itself is considered a normal labral variant, association between sublabral foramen and SLAP tears has been found [22, 23, 25]. This may be related to a sublabral foramen leading to alterations in biomechanics that produce greater forces on the superior labrum, thus predisposing to labral pathology.

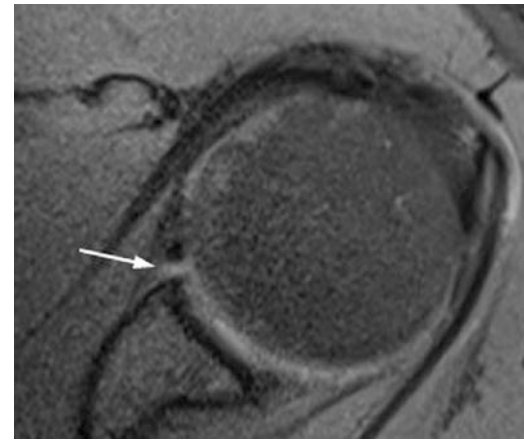

Fig. 8.5 Sublabral foramen. Axial fat-suppressed proton density image shows focal detachment of the anterosuperior labrum (arrow). There is a smooth labral contour and lack of significant labral displacement, differentiating the sublabral foramen from a labral tear

8.2 Microinstability

8.2.1 Definition

Microinstability is a heterogeneous set of disorders with complex pathophysiology that presents a particular challenge both in its clinical and imaging evaluation. No universally accepted definition exists in the literature. Microinstability is generally understood to consist of poorly localized shoulder pain related to pathologic laxity without frank dislocation [26]. Historically, shoulder instability has been characterized as either TUBS (traumatic unidirectional Bankart lesion, responds to surgery) or AMBRII (atraumatic, multidirectional, bilateral, responds to rehabilitation, inferior capsular shift, and interval closure) [5, 27]. TUBS typically presents with the classic Bankart and Hill-Sachs lesions seen in anterior shoulder instability, whereas in AMBRII no obvious structural lesions are found. Microinstability encompasses the wide stretch of territory between the two extremes of TUBS and AMBRII. It is helpful to subdivide microinstability into two types, one associated with repetitive overhead motions—AIOS (acquired instability in overstressed shoulder)—and one without—AMSI (atraumatic minor shoulder instability) [27–30].

8.2.2 Classification and Pathophysiology

AIOS is a common type of microinstability typically seen in young overhead athletes, such as baseball pitchers, volleyball players, and tennis players. However, individuals whose professions require repetitive overhead motions, such as painters and builders, share a similar mechanism that predisposes them to the development of microinstability. Much of our knowledge of microinstability and AIOS is grounded in research performed to better understand the mechanics of the throwing athlete. It is this model that we will focus on to help understand the pathophysiology of AIOS.

The performance of an elite throwing athlete, the ability to throw both with high velocity and accuracy, relies on the complex interplay between static and dynamic stabilizers of the shoulder. Furthermore, the entire body is involved in the "kinetic chain" that transmits force from the ground up, coordinating the sequence of muscle contractions that culminate in tremendous force generation in the throwing shoulder. Imbalances in the components of the "kinetic chain" can contribute to the deleterious effects on the supporting structures of the shoulder seen in throwing athletes [29, 31–33]. Repetitive distracting forces on the throwing shoulder require internal adaptations that increase mobility to achieve peak performance. At the same time, shoulder stabilizers are tasked to prevent pathologic laxity and instability. The fine balance of these competing forces on the throwing shoulder is known as the "thrower's paradox" [34]. The disturbance of this balance can lead to injury.

8.2.2.1 Internal Impingement

There are two main theories that attempt to explain the mechanisms of injury and etiology of microinstability in the throwing athlete. In the anterior laxity internal impingement theory, repetitive abduction and external rotation experienced by the throwing shoulder during the late cocking and early acceleration phases of the throwing motion leads to adaptive changes in the anterior joint capsule. The anterior joint capsule stretches, causing anterior capsular laxity and leading to anterior instability [35–38]. The lax anterior capsule allows for increased external rotation of the shoulder, which leads to pathologic contact between the greater tuberosity, posterosuperior labrum, and rotator cuff in a process known as internal impingement. Internal impingement typically consists of the triad of posterosuperior labral tears, articular surface tearing of the posterior supraspinatus or anterior infraspinatus tendons, and cystic changes in the posterior aspect of the humeral head [39]. Posterior humeral head articular cartilage lesions can also be found in the setting of internal impingement [40].

8.2.2.2 Posterior Capsular Contracture/ Glenohumeral Internal Rotation Deficit

The alternative theory that attempts to explain the spectrum of shoulder pathology seen in overhead athletes proposes that the initiating event occurs in the posterior capsule, rather than the anterior capsule as suggested by the anterior laxity internal impingement theory. The posterior capsule must withstand tensile forces of up to 750 N during the deceleration and follow-through phases of throwing [41]. This repetitive tensile loading on the posteroinferior capsule during the follow-through phase eventually causes posteroinferior capsular hypertrophy and contracture [41, 42]. Since the posterior band of the IGHL is located directly below the humeral head during maximum abduction and external rotation seen in the late cocking and early acceleration phases, a contracted posterior band causes posterosuperior shift of the humeral head relative to the glenoid [41, 43]. Such posterosuperior humeral shift allows for increased humeral external rotation due to increased clearance of the greater tuberosity over the glenoid, but also leads to more profound internal impingement between the humeral head, posterosuperior labrum, and rotator cuff. Thus, the ultimate result is the same as proposed by the internal impingement theory—pathology involving the posterosuperior labrum and adjacent rotator cuff. The posterior capsular contraction theory further suggests that the posterosuperior displacement of the humeral head causes functional redundancy and slackening of the anteroinferior capsule, producing a pseudolaxity that may simulate true laxity related to anterior capsular stretching.

Contraction of the posterior capsule also produces loss of internal rotation in the throwing shoulder compared to the non-throwing shoulder, a concept known as glenohumeral internal rotation deficit (GIRD). GIRD is defined as a loss of internal rotation >18° compared to the contralateral side, which can be easily assessed on physical examination [42]. Indeed, research has corroborated that GIRD has a significant association with pathology in the throwing shoulder. In a study by Verna of 38 overhead athletes with SLAP II tears, all were found to have significant GIRD, with an average of 33 degrees [44]. A study by Kibler assigned high-level tennis players to two groups, one that performed daily posteroinferior capsular stretching to minimize GIRD and one that did not [45]. During the 2-year follow-up period, the stretching group experienced both a significant increase in internal rotation and a 38% decrease in the incidence of shoulder injury compared to the control group.

8.2.2.3 Superior Labrum Anterior Cuff (SLAC) and MGHL Lesions

There are several types of microinstability not necessarily related to overhead activity. These are generally related to injuries to the supporting ligamentous structures of the shoulder, in particular the superior (SGHL) and middle (MGHL) glenohumeral ligaments, as well as the rotator interval. The rotator interval includes the SGHL, coracohumeral ligament, joint capsule, and biceps tendon [46]. The SGHL is particularly important in restraining anterior and superior translation of the humeral head in shoulder flexion and lesser degrees of abduction [47–49]. Injury to the SGHL can lead to pathologic anterosuperior translation of the humeral head with pathologic contact between the humeral head, anterosuperior labrum, and rotator cuff. The constellation of anterosuperior labral tears, articular surface tearing of the anterior supraspinatus tendon, and SGHL injury is known as the superior labrum anterior cuff (SLAC) lesion [50]. In the original work by Savoie and colleagues, 39 of 40 patients had avulsion of the SGHL, thought to be the inciting event precipitating a SLAC lesion [50].

The MGHL is the primary anterior stabilizer of the shoulder at 45 degrees of abduction and also serves to limit external rotation [27]. Dysfunction of the MGHL has long been recognized as a potential cause of microinstability [51]. In a study by Savoie and colleagues, 33

patients with isolated avulsions of the MGHL demonstrated evidence of anterior instability [52]. Subsequent arthroscopic repair led to improvement in pain and function in all patients.

8.2.2.4 Atraumatic Minor Shoulder Instability (AMSI)

A rare form of microinstability not related to overhead activity is atraumatic minor shoulder instability (AMSI). AMSI generally presents as shoulder pain after a period of inactivity, such as during pregnancy or immobilization [27]. These patients may have static anatomic variants of the MGHL, including absence, hypoplasia, or a large sublabral foramen or Buford complex [23, 25, 30].

8.2.3 Imaging Diagnosis of Microinstability

The findings of internal impingement are well depicted on MR imaging. The classic constellation of findings on MRI includes posterosuperior labral tears, articular surface tears of the posterior supraspinatus or anterior infraspinatus tendons, and cystic changes in the posterior aspect of the humeral head (Fig. 8.6) [39, 53]. However, not all findings may necessarily be present. MR arthrography with ABER (abduction external rotation) view is optimal for the evaluation of internal impingement since it has greater sensitivity for labral pathology and articular surface rotator cuff tears [53–55]. The ABER view may even depict impingement of the rotator cuff between the greater tuberosity and posterosuperior glenoid/labrum since ABER recreates the abduction and external rotation position in which internal impingement occurs. However, care must be taken not to misinterpret contact between the undersurface of the rotator cuff and the posterosuperior glenoid/labrum in the ABER position as internal impingement in the absence of other associated pathology, since such contact can be seen in normal individual placed in the ABER position [35, 56, 57].

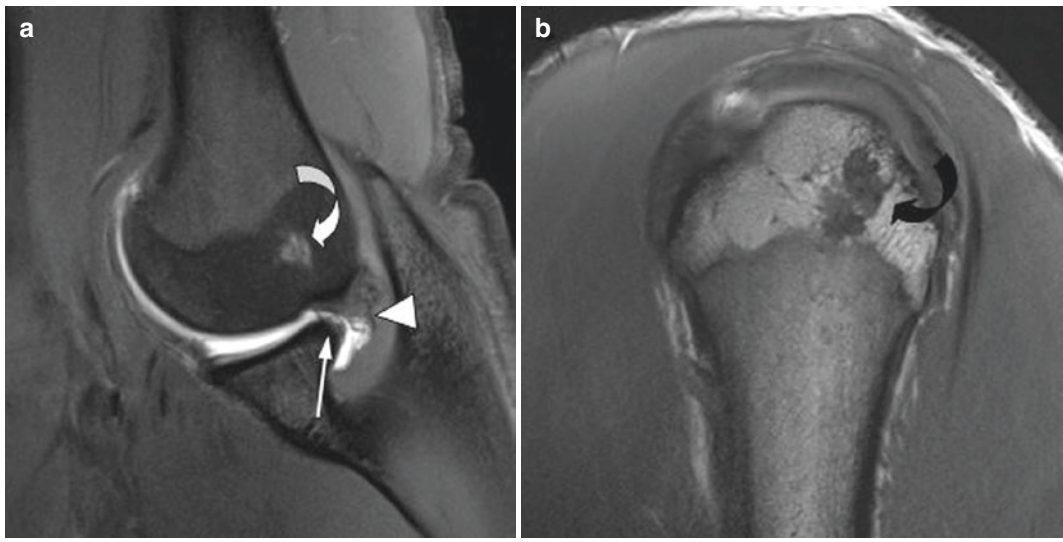

Fig. 8.6 18-Year-old baseball pitcher with shoulder pain. (**a**) Fat-suppressed T1-weighted abduction external rotation (ABER) view from an MR arthrogram of the shoulder demonstrates tearing of the posterosuperior labrum (arrow), articular surface tearing of the posterior supraspinatus tendon fibers (arrowhead), and cystic changes within the posterior aspect of the humeral head (curved arrow). Findings are consistent with posterosuperior internal impingement. (**b**) Sagittal T1-weighted image confirms that the cystic changes are present at the anterior aspect of the greater tuberosity middle facet (curved arrow)

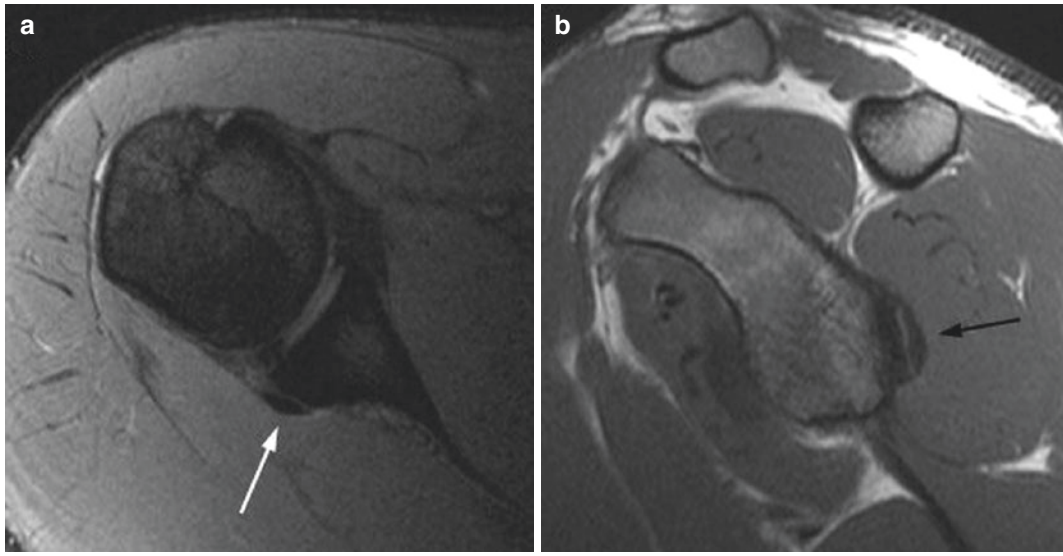

Fig. 8.7 21-Year-old baseball pitcher with shoulder pain. (**a**) Axial gradient-echo and (**b**) sagittal T1-weighted images demonstrate a crescent-shaped focus of low signal intensity adjacent to the posteroinferior aspect of the glenoid rim (arrow), consistent with mineralization in the setting of a Bennett lesion

Several theories exist that attempt to explain the occurrence of the posterior humeral head cysts. Traditionally, they have been thought to be the sequela of impaction injury as the humeral head abuts the posterosuperior glenoid during the late cocking and early acceleration phase of throwing. However, more recently, it has been proposed that inflammatory changes in the posterior humeral head secondary to internal impingement may lead to increased vascularity and result in cyst formation [39].

A number of imaging findings are associated with GIRD in the throwing athlete. A study by Tuite and colleagues found that patients with GIRD have a longer posteroinferior labrum, thicker posteroinferior capsule, and shallower posterior capsular recess [58]. In a study of professional baseball pitchers with GIRD, Tehranzadeh and colleagues also observed a thickened appearance of the posterior band of IGHL on MR imaging [59]. A variant of GIRD characterized by a thickened posteroinferior capsule is the Bennett lesion, a crescent-shaped focus of extra-articular mineralization at the posteroinferior aspect of the glenoid rim from calcification of the posterior band of IGHL and adjacent labrum (Fig. 8.7) [60, 61].

Imaging in the classic SLAC lesion reveals tears of the anterosuperior labrum, articular surface tearing of the anterior supraspinatus tendon, and injury of the SGHL. However, tears of the cranial fibers of the subscapularis tendon can also be seen in the setting of SLAC given their close proximity to the anterior supraspinatus tendon. SLAC lesions are also associated with lesions of the intra-articular biceps tendon and other components of the rotator interval, such as the coracohumeral ligament and rotator interval capsule (Fig. 8.8).

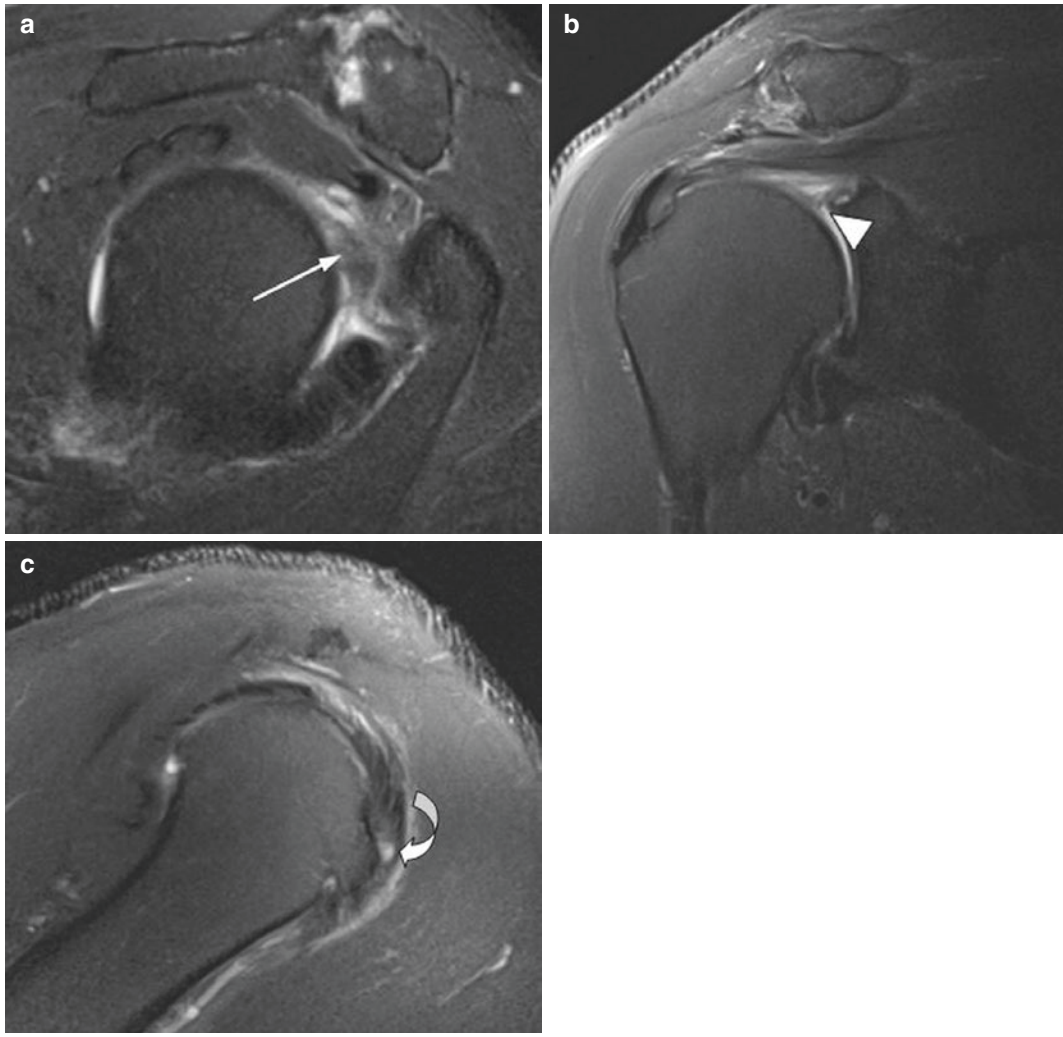

Fig. 8.8 60-Year-old male with shoulder pain. (**a**) Sagittal fat-suppressed T2-weighted image demonstrates absence of the biceps tendon, coracohumeral ligament, and superior glenohumeral ligament (SGHL) in the rotator interval (arrow), consistent with tears. Instead, debris and organizing hemorrhage fill the rotator interval. (**b**) Coronal fat-suppressed T2-weighted image demonstrates a superior labral tear (arrowhead). (**c**) Sagittal fat-suppressed T2-weighted image shows an articular surface tear of the far-anterior supraspinatus tendon (curved arrow). Constellation of findings can be seen in the setting of superior labrum anterior cuff (SLAC) lesion

8.3 SLAP Tears

8.3.1 Definition and Classification

Superior labral tears or SLAP (superior labrum anterior-posterior) lesions all involve the superior labrum at the level of the biceps origin, or approximately the portion of the labrum from 11 o'clock to 1 o'clock [62]. The significance of SLAP tears lies in their relationship to the biceps labral complex. As a result, many SLAP tears lead to instability of the biceps anchor and result in functional impairment and even microinstability of the glenohumeral joint.

Snyder and colleagues were the first to use the term SLAP lesion and described the original four types of SLAP lesions [63]. The classification system carries treatment implications, since the

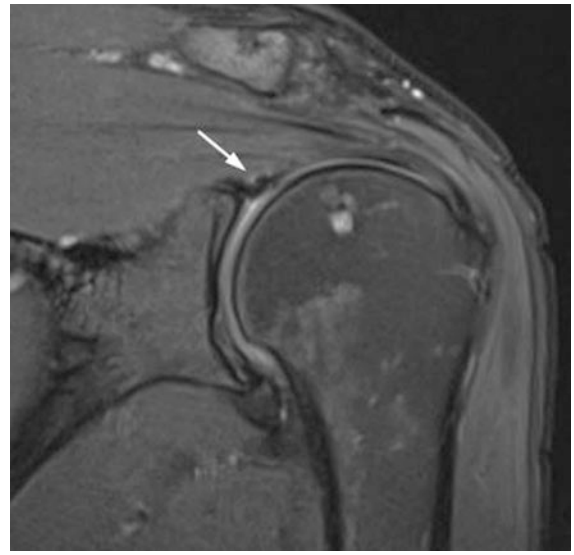

Fig. 8.9 SLAP I lesion. Coronal fat-suppressed T2-weighted image demonstrates degenerative fraying of the superior labrum (arrow), consistent with a SLAP I lesion

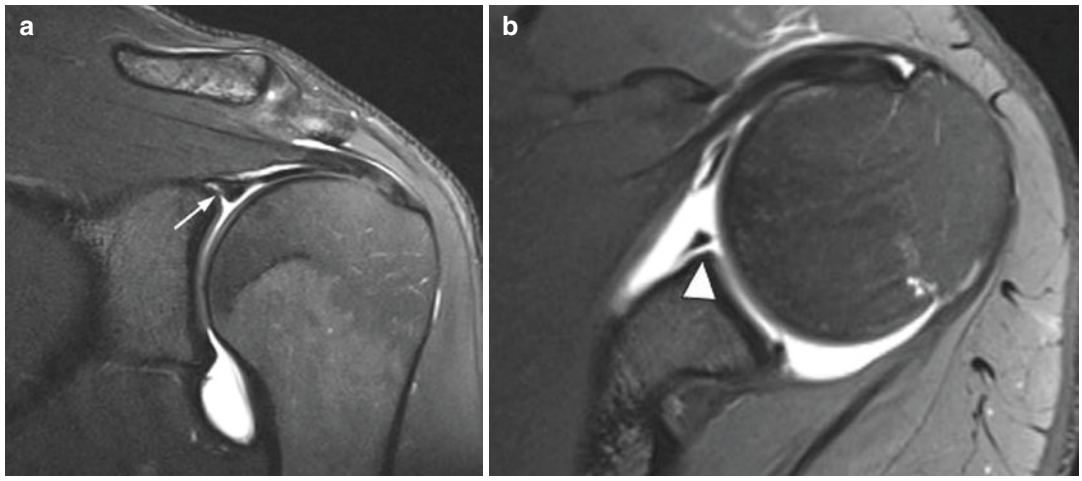

Fig. 8.10 SLAP II lesion. (**a**) Coronal fat-suppressed T2-weighted MR arthrographic image demonstrates detachment of the superior labrum and biceps anchor from the underlying glenoid with slightly irregular margins (arrow), consistent with a SLAP II lesion. (**b**) Axial fat-suppressed proton density MR arthrographic image at the level of the anterosuperior labrum demonstrates that the tear propagates to a sublabral foramen (arrowhead). Note that, in contrast to the SLAP tear, the sublabral foramen has smooth margins.

different types of SLAP lesions are treated via different surgical techniques. Furthermore, the different SLAP types have different implications for the stability of the biceps anchor. The four original types of SLAP tears include:

- Type I—Degenerative fraying of the superior labrum with an intact biceps anchor (Fig. 8.9).
- Type II—The superior labrum and biceps anchor are detached from the underlying glenoid (Fig. 8.10). This results in an unstable biceps anchor.
- Type III—Bucket-handle tear of the superior labrum without extension to the biceps tendon (Fig. 8.11). The central portion of the tear may or may not be displaced inferiorly into the joint. The biceps anchor remains attached to the glenoid.
- Type IV—Bucket-handle tear of the superior labrum with extension of the tear to the biceps

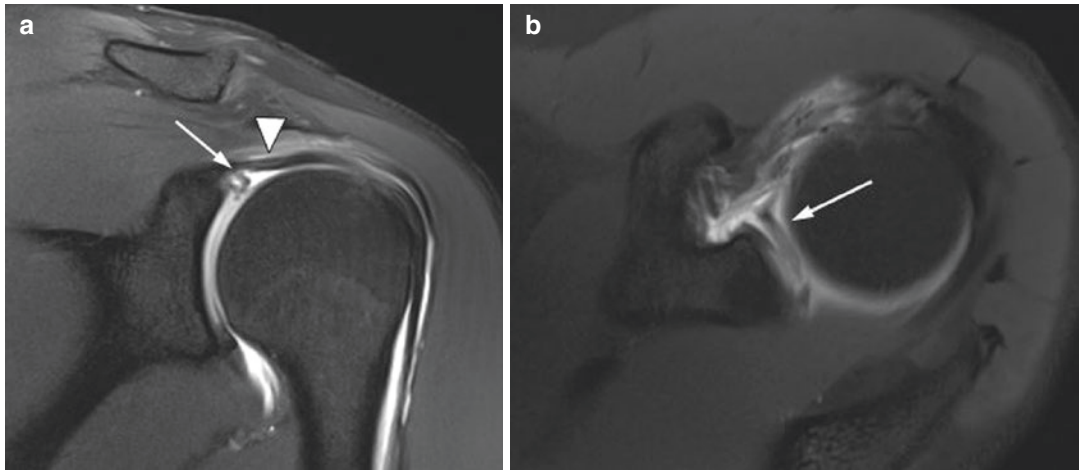

Fig. 8.11 SLAP III lesion. Coronal (**a**) and axial (**b**) fat-suppressed T1-weighted MR arthrographic images demonstrate a bucket-handle tear of the superior labrum, with the detached labrum surrounded by intra-articular contrast (arrow). Note a normal biceps tendon (arrowhead)

tendon. The biceps tendon and the attached labral flap may displace into the joint. This type of tear renders the biceps anchor unstable.

The passage of synovial fluid through the cleft created by a labral tear may result in the formation of a paralabral cyst (Fig. 8.12).

There is discrepancy in the frequency of the different SLAP lesions reported in the literature. In part, this is related to differences in patient demographics across studies, particularly since there is increasing prevalence of degenerative labral fraying with age [64]. However, considerable variability also exists in the threshold used by arthroscopists in classifying the different SLAP lesions. The original study by Snyder and colleagues reported the type II SLAP lesion as the most common (41%), followed by type III (33%), with only 11% of patients having a type I SLAP lesion [63]. However, this study looked at a relatively young patient population, with an average age of 37.5 years, and had stringent criteria for classifying type I SLAP lesions. In a relatively older patient population, with an average age of 44.2 years, Kim and colleagues found that the type I SLAP lesion was most common, accounting for 74% of SLAP lesions, followed by type II (21%) [64].

In the years following the classification of the original four SLAP lesions, six additional SLAP categories have been described [65–67]. Also known as extended SLAP, this group of SLAP lesions encompasses superior labral tears that also propagate to other labral quadrants or capsuloligamentous structures (Table 8.1), including a superior labral tear propagating to a Bankart lesion of the anteroinferior labrum (SLAP V) (Fig. 8.13), SLAP tear extending to the posterior labrum (SLAP VIII), circumferential tear of the labrum (SLAP IX), and a SLAP lesion that extends into the rotator interval, including the SGHL, coracohumeral ligament, or rotator interval capsule (SLAP X).

8.3.2 Pathophysiology

The pathophysiology of SLAP lesions can be divided into those that are caused by repetitive overhead activity and those that are not related to overhead activity, although there is overlap between the two. One of the more common mechanisms for the development of a SLAP lesion in the non-overhead athlete is a fall on an outstretched hand. This mechanism causes compression of the biceps-labral complex between the humeral head and glenoid [63]. A biomechanical study by Clavert and colleagues demonstrated that in the setting of a fall on an

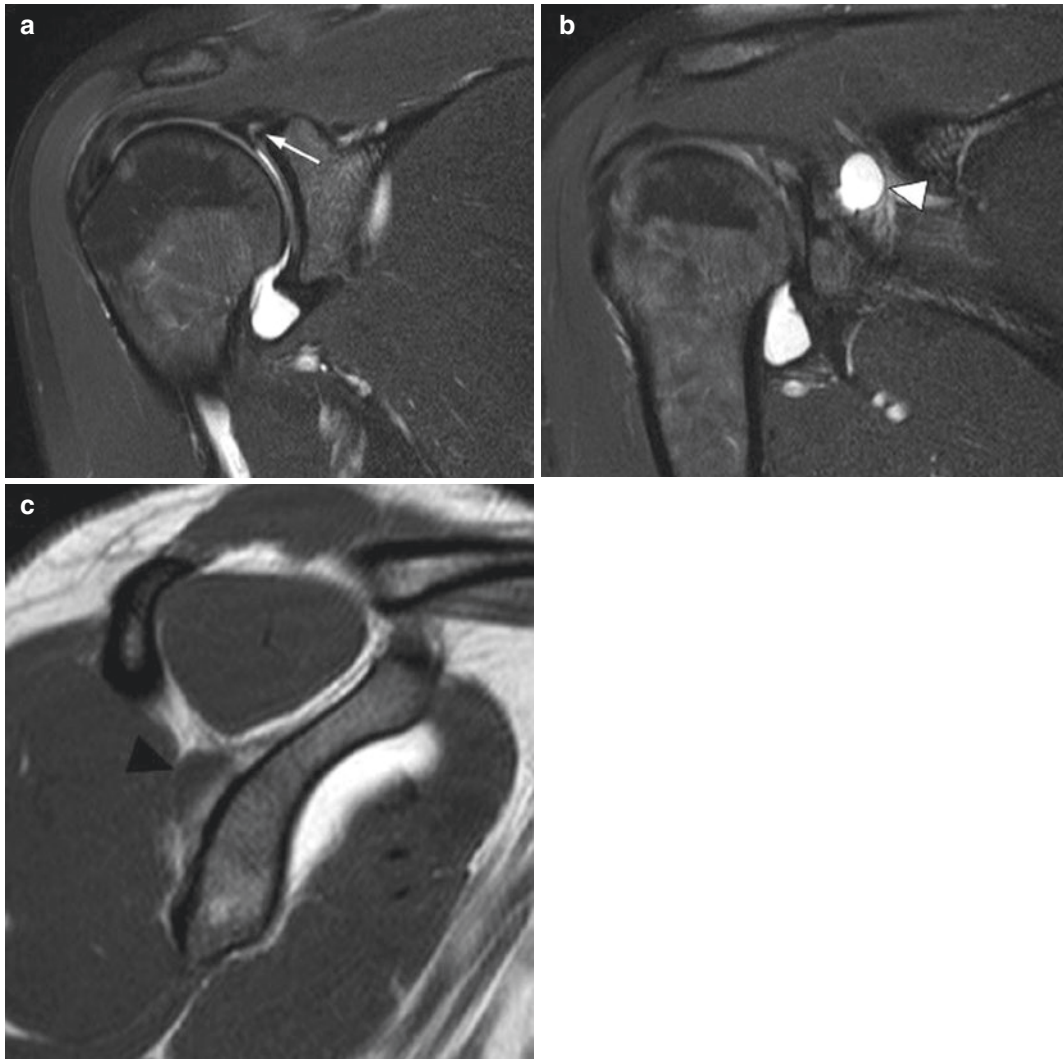

Fig. 8.12 SLAP II lesion with paralabral cyst. (**a**) Coronal fat-suppressed T2-weighted MR arthrographic image demonstrates detachment of the superior labrum and biceps anchor from the underlying glenoid (arrow), consistent with a SLAP II lesion. (**b**) Coronal fat-suppressed T2-weighted and (**c**) sagittal T1-weighted MR arthrographic images demonstrate an associated paralabral cyst in the spinoglenoid notch (arrowhead). Note that the cyst is hypointense on the T1-weighted sequence since it does not fill with intra-articular contrast

outstretched hand, SLAP tears are more likely with a forward fall (shoulder flexed) compared to a backward fall (shoulder extended) [68].

Another important mechanism responsible for the pathogenesis of some SLAP lesions is traction on the biceps-labral complex by forceful contraction of the biceps tendon, such as when lifting a heavy object. A biomechanical study by Bey and colleagues found that the generation of SLAP lesions by traction from the biceps tendon is facilitated by inferior subluxation of the humeral head [69]. In some circumstances, a combination of mechanisms may be responsible for the development of SLAP lesions in the setting of a single traumatic event. For example, a forceful contraction of the biceps tendon during a fall on an outstretched hand can combine both compressive and tensile forces on the superior labrum and biceps anchor.

In the overhead-throwing athlete, several factors contribute to the development of SLAP

lesions. As previously discussed, internal impingement in the shoulder is caused by excessive external rotation, which leads to abnormal contact between the greater tuberosity, posterosuperior labrum, and rotator cuff. This mechanism produces posterosuperior SLAP lesions. Posterior capsular contracture/GIRD also contributes to posterosuperior labral lesions since the associated posterosuperior shift of the humeral head relative to the glenoid places increased stress on the posterosuperior labrum. Finally, another important component of the cascade of biomechanical factors resulting in SLAP lesions in the throwing athlete is known as the peel-back mechanism [41, 70]. In the position of maximal shoulder abduction and external rotation experienced during the late cocking and early acceleration phases of throwing, the biceps tendon exerts significant tensile and torsional forces on the biceps-labral anchor. These forces result in stripping and tearing of the biceps-labral anchor which may propagate posteriorly, or both posteriorly and anteriorly. The combination of the above mechanisms results in SLAP lesions in throwing athletes that almost always extend to the posterosuperior

Table 8.1 Classification of SLAP lesions

SLAP lesion	Description
Type I	Degenerative fraying of the superior labrum
Type II	Detachment of the superior labrum and biceps anchor from the glenoid
Type III	Bucket-handle tear of the superior labrum without extension to the biceps tendon
Type IV	Bucket-handle tear of the superior labrum with extension to the biceps tendon
Type V	Superior labral tear continuous with a Bankart lesion of the anteroinferior labrum
Type VI	Unstable flap tear of the superior labrum
Type VII	Superior labral tear that extends anteriorly to involve the middle glenohumeral ligament
Type VIII	Superior labral tear extending to the posterior labrum (to at least 9 o'clock)
Type IX	Circumferential tear of the labrum
Type X	Superior labral tear extending into the rotator interval

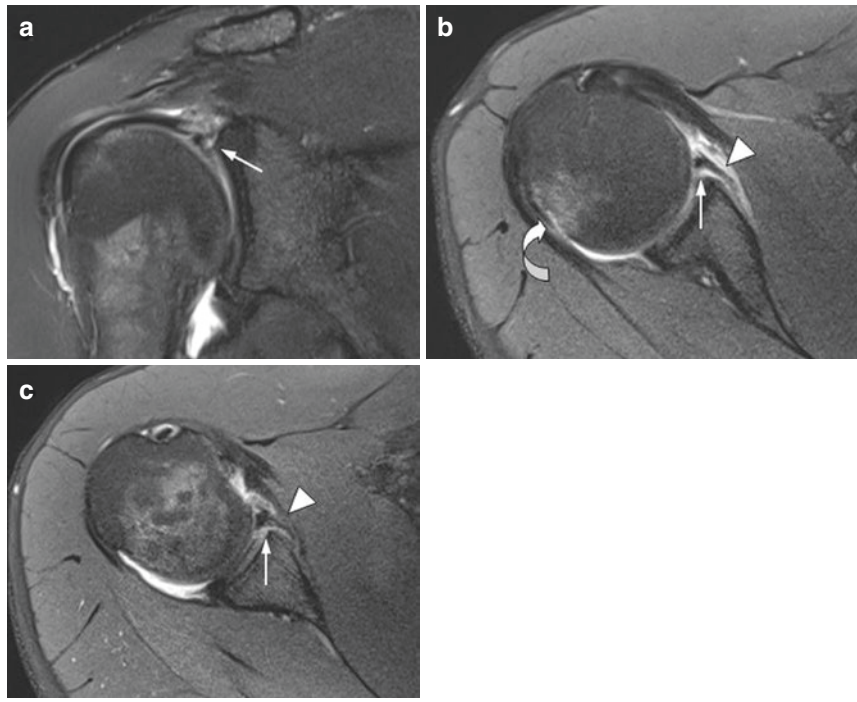

Fig. 8.13 SLAP V lesion. (**a**) Coronal fat-suppressed T2-weighted image demonstrates superior labral tear (arrow). Axial fat-suppressed proton density images at the level of the equator (**b**) and anteroinferior labrum (**c**) demonstrate propagation of the tear to the anterior and anteroinferior labrum (arrow) with stripping of the periosteal sleeve (arrowhead), consistent with anterior labral periosteal sleeve avulsion (ALPSA), a labral Bankart variant. There is also a Hill-Sachs impaction fracture of the posterolateral humeral head (curved arrow)

quadrant. Indeed, SLAP lesions of the anterosuperior labrum without posterior extension are rare in throwing athletes [41].

SLAP lesions also contribute to microinstability of the shoulder, particularly the lesions that cause instability of the biceps anchor. A study by Pagnani and colleagues found that SLAP lesions that destabilized the biceps anchor resulted in increased anteroposterior and superoinferior glenohumeral translation compared to SLAP lesions that did not [71]. Hantes and colleagues found a higher rate of preoperative shoulder dislocations in patients with combined Bankart and SLAP lesions compared to those with Bankart lesions alone [72]. Indeed, SLAP lesions can be found in both acute and recurrent glenohumeral dislocations [73–75]. In this setting, SLAP lesions are believed to be contributors to instability rather than the primary lesions responsible for instability.

8.3.3 Technical Considerations: Conventional MRI Versus MR Arthrography

MR arthrography is a technique whereby contrast material is introduced into a joint to help visualize both normal anatomy and pathology. There are two methods to perform MR arthrography—direct and indirect. In direct MR arthrography, dilute contrast material is injected directly into a joint via an 18–22 gauge needle [76]. For the shoulder, approximately 10–15 mL of a gadolinium solution is diluted to a concentration of 1–2 mmol/L with normal saline, lidocaine, and iodinated contrast if fluoroscopic guidance is used [76, 77]. Although injection can be done without direct visualization, sonographic or fluoroscopic guidance is preferred to help insure instillation of the contrast mixture into the joint. Multiple approaches for needle placement can be used, including anterior, posterior, and rotator interval approaches [78–80]. The approach is generally chosen to avoid crossing structures that are suspected of having pathology. MR imaging should be performed within 1 h of intra-articular injection to maintain adequate contrast-to-noise ratio, as the intra-articular gadolinium diffuses out of the joint with time [81].

Indirect MR arthrography involves the intravenous injection of gadolinium-based contrast media in a concentration of 0.1 mmol/kg [77]. The intravenous contrast diffuses into the joint space over time. The rate of diffusion depends on the permeability of the joint which is increased in infectious and inflammatory conditions, the pressure differential between the intravascular and joint spaces, and the viscosity of joint fluid [82, 83]. Exercising the joint prior to imaging increases both vascular permeability and vascular pressure, thereby increasing the amount of contrast diffusing into the joint. For the shoulder, imaging is generally performed with a delay of 15 min after contrast injection [83].

For both direct and indirect MR arthrography, the imaging protocol consists of fat-suppressed T1-weighted sequences in multiple planes to visualize the contrast material and intra-articular structures. At least one fluid-sensitive sequence, such as a STIR, fat-suppressed T2, or fat-suppressed proton density, is also acquired to evaluate for extra-articular fluid collections, T2 hyperintense periarticular mass lesions, or bone marrow edema.

One of the main advantages of direct MR arthrography compared to indirect arthrography or conventional MRI consists of superb joint distention. This helps separate intra-articular structures, which delineates anatomy and allows contrast to outline defects and tears. The disadvantages of direct MR arthrography include its relatively invasive nature, which may make some patients hesitant to undergo the procedure, and the additional amount of time necessary to perform the intra-articular injection. Although indirect MR arthrography is not invasive and also allows contrast to outline intra-articular structures, it lacks the capability to create the joint distention that may be necessary in some cases to fully evaluate the joint. Furthermore, since the contrast is not introduced selectively into the joint of interest with indirect arthrography, other extra-articular structures can enhance as well, including blood vessels and synovial lined spaces, such as bursae and tendon sheaths.

A number of studies have looked at the diagnostic performance of MR arthrography compared to conventional MRI and arthroscopic/surgical findings in diagnosing SLAP lesions. In a

study evaluating direct MR arthrography with surgical findings as the reference standard, Bencardino and colleagues found a high sensitivity (89%), specificity (91%), and accuracy (90%) for MR arthrography in diagnosing SLAP lesions [84]. MR arthrography correctly classified 76% of SLAP lesions that it identified. A study by Waldt and colleagues found that MR arthrography had sensitivity of 82%, specificity of 98%, and accuracy of 94% in diagnosing SLAP lesions [85]. A study by Chandnani and colleagues comparing MR arthrography to conventional MR imaging in the evaluation of labral tears found similar sensitivities, 96% and 93%, respectively [86]. However, direct MR arthrography performed better at detecting detached labral fragments—96% compared to 46% for conventional MRI. Amin and Youssef found that in 34 patients who had normal conventional MRIs, MR arthrography was able to detect 18 SLAP lesions that were confirmed by arthroscopy [87]. In a study of 20 athletes by Magee and colleagues, MR arthrography detected 9 labral tears that were not seen on conventional MRI, 6 of which were SLAP lesions [88].

Comparing indirect MR arthrography to conventional MRI in detecting SLAP lesions, Herold and colleagues found a higher sensitivity (91% vs. 73%), the same specificity (85% vs. 85%), and higher accuracy (89% vs. 77%) for indirect MR arthrography [89]. Dinaeur and colleagues found that indirect arthrography had higher sensitivity (84–91% vs. 66–85%), a slightly higher accuracy (78–86% vs. 70–83%), but lower specificity (58–71% vs. 75–83%) compared to conventional MRI in detecting SLAP lesions [90]. In a head-to-head comparison of indirect and direct MR arthrography, Jung and colleagues found no statistically significant difference in the sensitivity and specificity of both methods in diagnosing labral tears [91].

8.3.4 The Role of Field Strength: 1.5 T Versus 3 T

With the continued evolution of MR magnets and coils, 3 T MR imaging is becoming widely available. High-field-strength 3 T MR imaging offers unique benefits and challenges compared to lower field strength systems. The main advantage of 3 T imaging lies in the higher signal-to-noise ratio (SNR) afforded by higher field strength systems. SNR increases linearly with field strength for frequencies less than 250 MHz [92, 93]. This means that, with all other parameters held constant, the SNR at 3 T is twice that compared to 1.5 T. The extra SNR allows imaging at smaller voxel sizes (larger matrix), thus improving spatial resolution, and has the potential to decrease imaging time, since the same amount of signal can be acquired in a shorter imaging period. As a result, 3 T imaging has the potential to afford improved evaluation of small, signal-poor structures, such as the shoulder labrum, that require both high spatial resolution and SNR to accurately diagnose tears.

3 T imaging also allows the implementation of a wide array of novel imaging techniques and pulse sequences. Parallel imaging, a technique that uses spatial information from individual radiofrequency coil elements to decrease imaging time, can only be performed on high-field-strength systems, since there is inherent loss of signal associated with this technique [94]. High-field-strength imaging is also necessary to perform functional imaging, such as T2 mapping, a technique that has been studied extensively in the evaluation of articular cartilage and is gaining new applications [95, 96].

The multiple advantages of 3 T imaging do not come without a cost. The hardware and radiofrequency coils from a 1.5 T system cannot be simply transposed to a 3 T system; 3 T systems require their own dedicated hardware and coils. 3 T imaging accentuates MRI artifacts, alters image contrast, and presents unique safety challenges compared to lower field strength systems. Susceptibility artifact, which causes signal loss and geometric distortion around paramagnetic materials, such as metal, air, and blood products, is much more pronounced at 3 T. This artifact is particularly problematic when imaging orthopedic hardware. Chemical shift artifact is also greater at 3 T due to the doubling of the frequency separation between fat and water [97]. This produces spatial misregistration at fat-water interfaces that is proportional to the frequency shift between fat and water.

Apart from the exaggeration of artifacts, another important effect of high-field-strength imaging is the alteration of T1 contrast due to the increase in the T1 relaxation time of tissues [97]. This leads to loss of signal unless there is compensatory increase in the repetition time (TR). Finally, increased field strength also causes increased energy deposition in the patient, which can cause tissue heating [94]. This issue can be a particular challenge at 3 T that has required substantial technical advances to overcome and must be addressed with each exam by the careful selection of sequence parameters. Ultimately, the adjustment of sequence parameters necessary to overcome significant artifacts, alteration of image contrast, and problem of increased energy deposition may partially offset the increased SNR afforded by 3 T imaging.

As far as the evaluation of the diagnostic performance of 3 T MRI in the detection of SLAP lesions, Magee and Williams found a sensitivity of 90% and specificity of 100% for conventional 3 T MRI compared to arthroscopy [98]. This is as good or better than the sensitivity (41–98%) and specificity (75–100%) reported for 1.5 T imaging [89, 90, 99–102]. To address whether the advantages of 3 T imaging may obviate the need for MR arthrography, Magee looked at the diagnostic performance of conventional MRI compared to MR arthrography at 3 T [103]. MR arthrography had a statistically significantly higher sensitivity (98%) than conventional MRI (83%), with the same specificity (99%), for the detection of SLAP lesions. On the other hand, Major and colleagues showed the same sensitivity (75%) and specificity (100%) for MR arthrography and conventional MRI at 3 T in the diagnosis of SLAP lesions, although MR arthrography performed better than conventional MRI in the other labral quadrants [104].

8.3.5 Imaging Diagnosis of SLAP Tears, and Differentiating Variants from Tears

The distinction between normal labral variants and tears is particularly challenging in the case of SLAP lesions, since the majority of labral variants occur in the superior and anterosuperior labral quadrants. The distinction is important clinically since operating on a normal labral variant will not address the source of a patient's pain and may lead to adverse consequences. A number of distinguishing features between variants and tears have been proposed, although they are not always absolute, and correlation with the clinical scenario is essential, especially in cases that are ambiguous on imaging.

Increased signal can often be seen in the posterosuperior labrum in the absence of labral pathology (Fig. 8.14). This is attributed to magic angle phenomenon, with the orientation of the labral collagen fibers in this position relative to the main magnetic field generating spurious increased signal on short TE (echo time) sequences, such as T1 and proton density [105, 106]. Adjusting the TE and positioning can help overcome this artifact (Fig. 8.14) [106]. Knowledge of this artifact is especially important in the context of the overhead-throwing athlete, given the posterosuperior location of labral tears in posterosuperior internal impingement.

The two most common normal variants of the superior labrum include cartilage undercutting and sublabral recess. Cartilage undercutting can be distinguished from a SLAP tear by its intermediate linear signal, similar to the rest of the hyaline articular cartilage, that is medially oriented, paralleling the contour of the glenoid rim (Fig. 8.1). A SLAP tear, on the other hand, often curves laterally away from the glenoid and demonstrates irregular margins. Cartilage undercutting also demonstrates smooth margins, width less than 2 mm, and normal adjacent labral signal [107]. Similarly, a smooth contour fluid signal cleft that parallels the curvature of the glenoid and is less than 2 mm in width is highly suggestive of a sublabral recess rather than a SLAP tear (Fig. 8.2b, c) [108]. The sublabral recess was initially thought to never extend posterior to the LHBT insertion [9]. However, given the variability in the superior labral attachment of the LHBT, studies have shown that a sublabral recess can indeed extend posterior to the LHBT insertion in the absence of a SLAP tear [17, 20, 108].

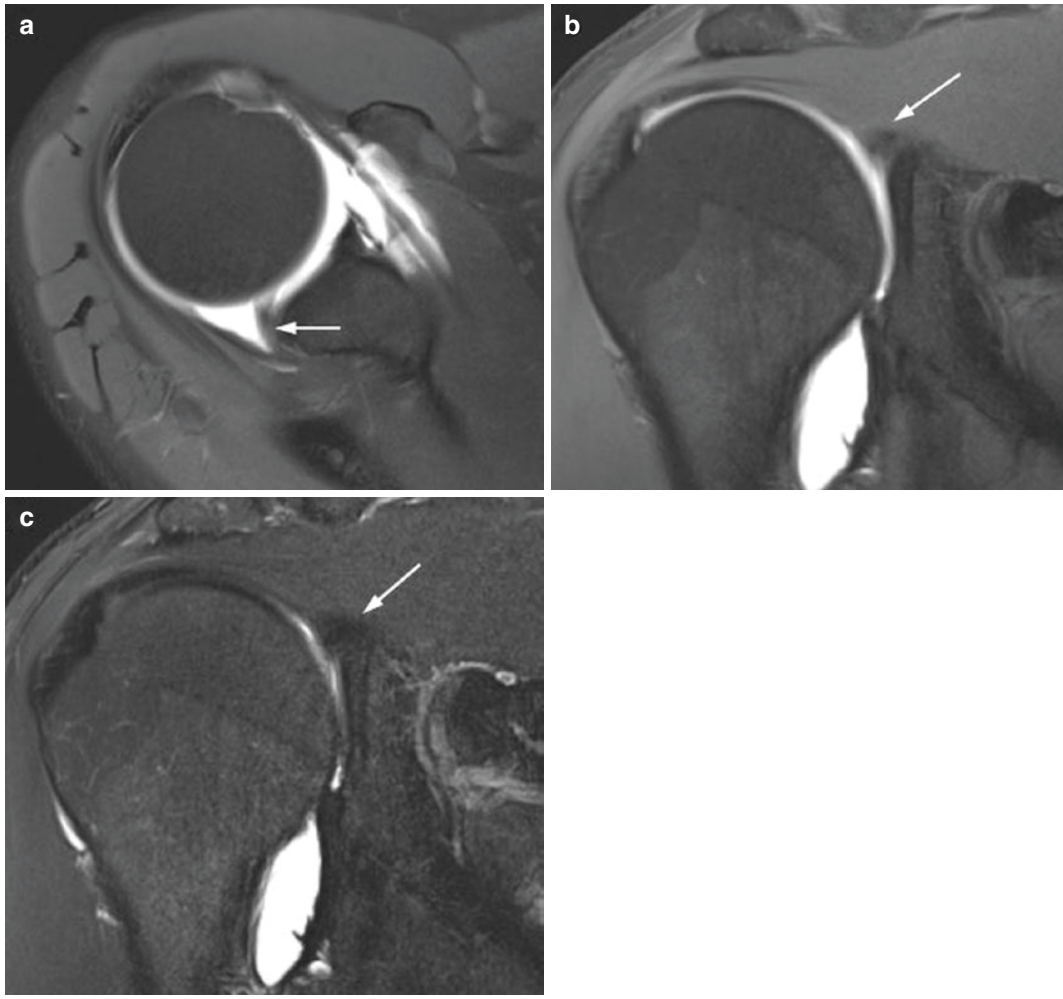

Fig. 8.14 Magic angle phenomenon in the posterosuperior labrum. (**a**) Axial and (**b**) coronal fat-suppressed T1-weighted MR arthrographic images demonstrate intermediate signal in the posterosuperior labrum (arrow). (**c**) Coronal T2-weighted image, which has a longer TE (echo time), shows a hypointense posterosuperior labrum (arrow), confirming that the increased signal on the T1-weighted sequences is an artifact. No posterosuperior labral injury was found on arthroscopy

The variants in the anterosuperior labral quadrant that can be confused for a SLAP tear include the sublabral foramen and Buford complex. The features distinguishing a sublabral foramen from a SLAP tear include focal detachment of the anterosuperior labrum without involvement of the biceps origin, labral displacement less than 1–2 mm, and a smooth labral contour (Fig. 8.5) [15]. The sublabral foramen is often associated with a sublabral recess [109–111]. The Buford complex represents an absent or a hypoplastic anterosuperior labrum accompanied by a thickened cordlike MGHL (Fig. 8.4). The Buford complex can sometimes be mistaken for a displaced labral tear. This pitfall can be avoided by following the thickened MGHL to its insertion on the humeral neck or as it blends with the anterior joint capsule. Correlating with the sagittal images is also important since the thickened MGHL can be well appreciated in the sagittal plane (Fig. 8.4b). Although it was previously thought that the anterosuperior labral variants cannot extend below the 3 o'clock position, studies have shown that both the sublabral foramen and

Buford complex can extend into the anteroinferior labrum to the level of the anterior band of the IGHL [24, 112].

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