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REVIEW ARTICLE

Spectrum of shoulder injuries in the baseball pitcher

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Abstract This review describes a range of shoulder injuries experienced by baseball pitchers. It is estimated that more than 57% of pitchers suffer some form of shoulder injury during a playing season. Knowledge of the overhead throwing cycle is crucial for our understanding of these shoulder injuries. Baseball pitchers are prone to rotator cuff tears from tensile overload and impingement. Glenoid labrum degeneration or tears are also common, due to overuse syndrome (micro-instability), internal impingement and microtrauma. An understanding of the lesions involved in overhead throwing is crucial in baseball pitchers, as long-term disability can result from these injuries, sometimes with severe financial consequences to the player.

Keywords Baseball · Pitcher · MR imaging · Shoulder

Introduction

This review describes a range of shoulder injuries experienced by baseball pitchers. It is estimated that more than 57% of pitchers suffer some form of shoulder injury during a playing season [1]. Knowledge of the overhead throwing cycle is crucial in understanding these shoulder injuries. Baseball pitchers are prone to rotator cuff tears from tensile overload and impingement. Glenoid labrum degeneration or tears are also common, due to overuse syndrome (micro-instability), internal impingement and microtrauma. An understanding of the lesions involved in overhead throwing is crucial in baseball pitchers, as long-term disability can result from these injuries, sometimes with severe financial consequences to the player.

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Biomechanics

The throwing cycle develops potential energy, which is then converted efficiently into kinetic energy transferred to the baseball. The overhead throwing cycle has six sequential phases [2, 3]:

1. "The wind-up". The body's overall center of gravity is first raised. The shoulder is in slight internal rotation. There is minimal stress at the shoulder in this phase. There is also minimal shoulder abduction.
2. "Early cocking". Activation of the deltoid initiates the cocking motion. The shoulder becomes abducted at 90°, with the humerus aligned at approximately 15° from the horizontal plane. Increased activity of the supraspinatus, infraspinatus and teres minor muscles marks the end of this phase [4]. The elbow is located in a plane posterior to the torso.
3. "Late cocking". Late cocking begins with the planting of the striding leg. The scapula retracts, forming a stable base for the humeral head. Abduction and external rotation result in posterior translation of the humeral head on the glenoid [5]. The torso rotates forward, generating a 400 N shear force across the anterior shoulder. Supraspinatus, infraspinatus and teres minor muscle activity increases. Rotator cuff activity produces a compressive force of

650 N. At the end of this phase, the shoulder is in maximal external rotation, reaching up to 180°.

4. "Acceleration". In this phase, there is progressive increased activity of the pectoralis major, latissimus dorsi and serratus anterior muscles. The shoulder rotates at speeds greater then 7,000° per second. There is early activity of the triceps and progressive activity of the pectoralis major, latissimus dorsi and serratus anterior. There is minimal posterior shear stress at the shoulder (50 N) in this phase. The capsule unwinds, and the humeral head re-centers as the shoulder rotates to the object release point of 90° [2–4].
5. "Deceleration". The deceleration phase begins when the object is released. Kinetic energy not imparted to the thrown object is dissipated within the peri-articular soft tissues. Deceleration is the most violent phase of the throwing cycle, with violent eccentric contraction of all muscle groups to decelerate the arm. The load on the joints are maximal, with posterior shear forces of 400 N, inferior shear forces of 300 N and a compressive force of 1000 N [2–4].
6. "Follow through". The body moves forward with the arm until motion stops. The muscles then revert to resting levels of activity.

Adaptive anatomic changes

Adaptive anatomic changes refer to non-pathological acquired variations, seen more frequently in baseball pitchers than in the general population:

- Generalized adaptive muscular hypertrophy of the dominant shoulder [6].
- Increased range of motion in external rotation by up to 16°.
- Increased anterior capsular laxity [7]. This is associated with increased anterior glenohumeral translation in external rotation and abduction.

Rotator cuff injuries

Pathophysiologic classification of rotator cuff injuries:

1. External Impingement
   I. Primary
   ii. Secondary
2. Internal Impingement (posterosuperior impingement)
   I. Primary
   ii. Secondary
3. Tensile overload (overuse syndrome)

Primary external impingement

Primary external impingement causes pain due to contact between the coracoacromial arch and the rotator cuff without perceptible glenohumeral joint instability. The severe inferior torque in the deceleration phase of the throwing cycle may lead to subacromial impingement [4]. Post-traumatic deformity, down-sloping acromion (Fig. 1), acromioclavicular degenerative change, and congenital thickening of the coracoacromial arch and os acromiale [8] increase the risk of impingement [9].

On MRI, rotator cuff tendinosis and tears associated with primary external impingement are more frequently seen in older athletes and are typically at the anterior half of the supraspinatus tendon within 1 cm from its insertion [10] (Fig. 1). Tendinosis manifests as thickening of the rotator cuff tendon, with increased signal intensity of the rotator cuff tendon on T2-weighted sequences. Tears are diagnosed when visualization of fluid intensity signal on T2-weighted imaging or gadolinium (on fat-saturated T1-weighted imaging) extends into a rotator cuff tendon. Treatment for primary external impingement includes subacromial decompression.

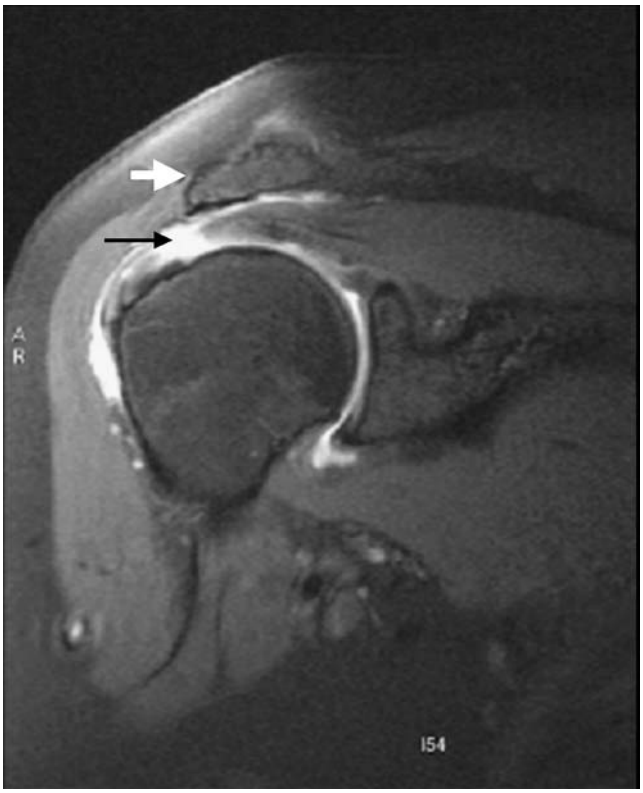

Fig. 1 Primary external impingement. Coronal T1-weighted fat-saturated image of a shoulder arthrogram demonstrates a down-sloping acromion (*white arrow*). There is an underlying full-thickness tear of the supraspinatus, approximately 1 cm from the distal insertion, visualized as hyperintense gadolinium extension into the subdeltoid subacromial bursa (*black arrow*)

Coracohumeral impingement syndrome can also cause pain in the baseball pitcher. In these cases, the distance between the coracoid and the lesser tuberosity of the humerus is decreased. Gerber et al. found that the normal coracohumeral distance measures 8.6 mm [11]. A coracohumeral distance of fewer then 6 mm on MRI is suggestive of impingement. There may be associated cystic changes at the lesser tuberosity of the humerus [12] (Fig. 2). The presence of coracohumeral impingement increases the likelihood of subscapularis tear [13].

Secondary external impingement

Secondary external impingement results from glenohumeral joint micro-instability [9]. It is the most frequent cause of pain in athletes [14]. There is significant repetitive shear force applied across the shoulder during the late cocking and early acceleration phases of throwing. The anterior capsule eventually fails, giving rise to anterior glenohumeral joint laxity and increased anterior translation during throwing [2]. Baseball pitchers with superior labrum anterior to posterior (SLAP) tears (Fig. 3) or injury to the long head of the biceps are also susceptible to secondary impingement. On MRI, supraspinatus and infraspinatus abnormalities can be at any location within the tendon substance [9]. While the imaging consequences of primary and secondary external impingement are similar, making a distinction between both types of external impingement is important, as surgical treatment is directed at different structures. Athletes with secondary impingement undergo correction of capsuloligamentous laxity and labral abnormalities (to address instability), while primary impingement is treated with coracoacromial decompression (to reduce bony impingement).

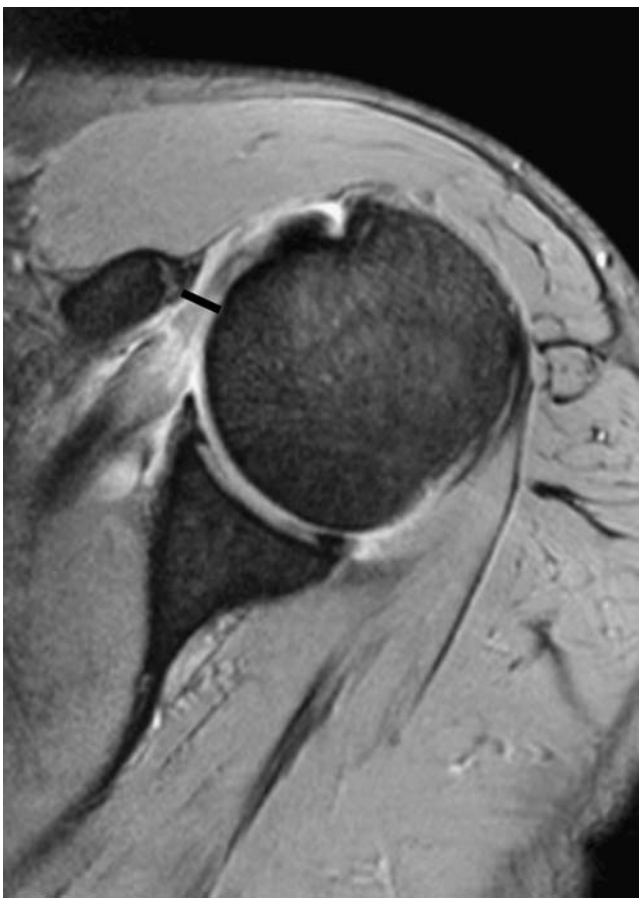

Fig. 2 Coracohumeral impingement. Axial gradient echo image shows 6 mm distance between the coracoid process and the humeral head, indicated by the *black line*. There is thickening and partial tear of the subscapularis tendon

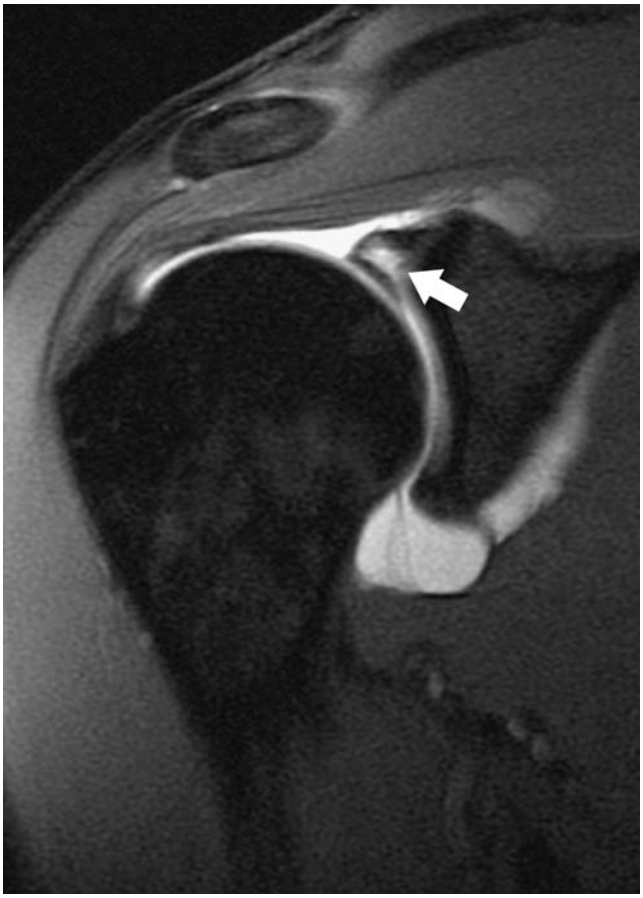

Fig. 3 Superior labral tear. Coronal T1-weighted fat-saturated image of a shoulder MR arthrogram shows a linear hyperintense extension of gadolinium at the superior labrum (*arrow*)

Primary and secondary internal impingement (posterosuperior glenohumeral impingement)

In the late cocking phase of the throwing cycle, during extreme abduction and external rotation, repetitive impaction of the posterior greater tuberosity on the supraspinatus/infraspinatus junction and posterosuperior glenoid results in internal or posterosuperior impingement. Internal impingement may result in rotator cuff tears and posterosuperior

labral tears [15]. Although internal impingement may occur in patients without evidence of glenohumeral instability (primary internal impingement) [16], it is usually secondary to increased anterior shoulder laxity and instability (secondary internal impingement) [15]. Arthroscopically, 81–100% of patients with a clinical diagnosis of pathological internal impingement have fraying or tears of the posterosuperior labrum and/or the cuff [17].

On MRI, posterior supraspinatus or supraspinatus-infraspinatus junction (posterior interval) tears are seen, associated with demonstrable impingement when the shoulder is abducted and externally rotated surgically. It is worth noting that this form of impingement can be seen in those that are not athletes, as well as in baseball pitchers [18]. Tears are usually small and involve the articular surface of the rotator cuff. Internal impingement tears are better seen on MR arthrography as a small undersurface linear contrast extension in the tendon (Fig. 4). Abduction and external rotation (ABER) positioning may be useful for tear detection in these patients, as relaxation of the posterior superior rotator cuff may allow gadolinium to seep into an occult or subtle tear [9, 19]. In addition, these athletes often have associated posterosuperior labral abnormalities. Cysts and impaction deformity are also seen at the posterior greater tuberosity and can increase diagnostic confidence in the diagnosis of internal impingement [19].

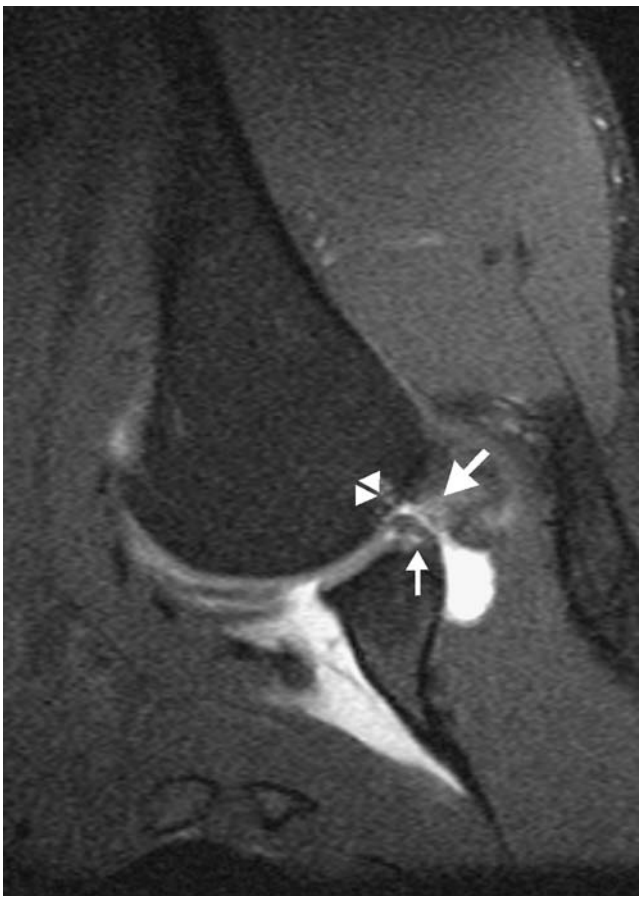

Fig. 4 Posterior superior glenohumeral impingement. T1-weighted fat-saturated ABER image of shoulder MR arthrogram shows an articular surface tear at the posterior supraspinatus (*large arrow*). There is also a superior labral tear (*thin arrow*) and a small cysts at the greater tuberosity (*arrowheads*)

Tensile overload (overuse syndrome)

Rotator cuff injuries from tensile overload are secondary to repetitive traction. This type of rotator cuff injury is typically seen in athletes following a sudden increase in pitching duration or intensity [20]. Axial tendon strength is greater than that of bone [4, 21], but violent stress at the rotator cuff during the deceleration phase of the throwing cycle is eccentric (not axial) and is likely responsible for tensile overload tears in normal tendons [20]. Repetitive stress encountered in pitchers also speeds up the normal aging process of the rotator cuff, leading to early tendinosis [9].

Rim–rent tears from tensile overload can be seen at the articular surface at the humeral tendinous insertion. These tears show high signal intensity extending between the greater tuberosity tendon insertion (supraspinatus foot print) and the tendon (Fig. 5) on MR fluid-sensitive sequences. They are mostly located in the anterior half of the supraspinatus and can be mistaken at times for intratendinous signal on MRI [10]. Rim–rent tears are treated with arthroscopic debridement [9]. Other tensile overload injuries are usually tiny tears at the articular surface of the supraspinatus and infraspinatus tendon (Fig. 6). These tears are often small and best seen on MR arthrography with the patient in the ABER position as fluid intensity or extension of gadolinium-based intra-articular contrast medium into the low intensity line on the articular surface of rotator cuff tendons [9].

Neuropathic cuff injury

Infraspinatus atrophy [4] is thought to be secondary to functional denervation from excessive tension on the suprascapular nerve (Fig. 7) [22, 23]. It has been reported to occur in up to 4.4% of major league pitchers [23]. Selective entrapment of the motor branches of the infraspinatus muscle has been postulated to occur when the nerve undergoes compression as it traverses and courses around the edge of the lateral border of the spine of the scapula [22]. Other causes of entrapment of the suprascapular nerve, including calcified or hypertrophic spinoglenoid ligament, arteriovenous malformations and ganglion cysts, have been described and can be detected on MRI. These other causes of suprascapular entrapment occur at the level of the spinoglenoid notch and are associated with supraspinatus muscle atrophy.

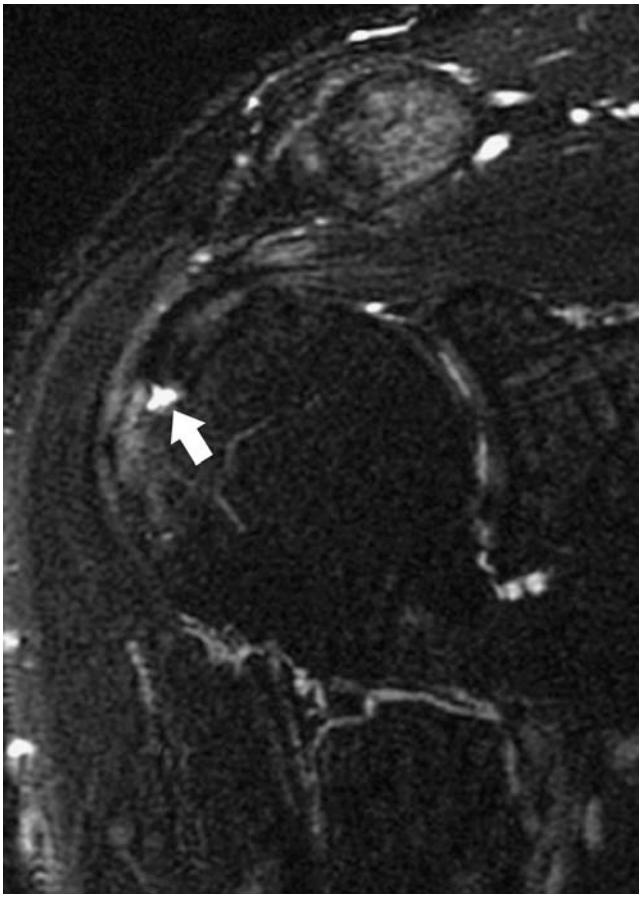

Fig. 5 Supraspinatus rim–rent tear. Coronal short-tau inversion recovery (STIR) image of shoulder MR arthrogram shows hyperintense fluid signal on the articular surface of the distal supraspinatus tendon footprint (*arrow*)

Labral injuries

Pathophysiologic classification of labral injury in pitchers [24].

Classification:

- Micro-instability and overuse syndrome
- Primary instability
  1. Microtrauma
  2. Generalized ligamentous laxity
- Acute traumatic instability

Micro-instability and overuse syndrome

Micro-instability occurs in baseball pitchers that have no perceptible instability of the glenohumeral joint. In this category, injuries are secondary to normal overuse of the shoulder.

The glenoid labrum is often torn, without perceptible underlying shoulder instability in pitchers and throwing athletes in general. The humeral head mildly translates back and forth during late cocking and deceleration phases of throwing under compressive forces against the labrum. This micro-instability and grinding often causes injuries of the anterosuperior labrum [4, 25, 26].

A tight postero-inferior capsule and inferior glenohumeral ligament (IGHL) result from repetitive follow-through motions after throwing. In long-term situations, the center of rotation is shifted posterosuperiorly on the glenoid and alters the interplay of torsional forces on the superior labrum and related biceps tendon and anchor. This condition is termed glenohumeral internal rotation deficit (GIRD). It allows for further external rotation by increasing the clearance for the greater tuberosity and also avoids direct internal impingement of the posterosuperior labrum/rotator cuff. In the long run, anterior capsular laxity and loss of glenohumeral internal rotation in abduction occur, resulting in a thrower's "dead-arm". In addition, there is posterior shift of the biceps vector, with twisting of the biceps attachment in the late cocking phase of throwing. The increased torsional forces are transmitted back to the biceps anchor and the posterosuperior labrum, resulting in a

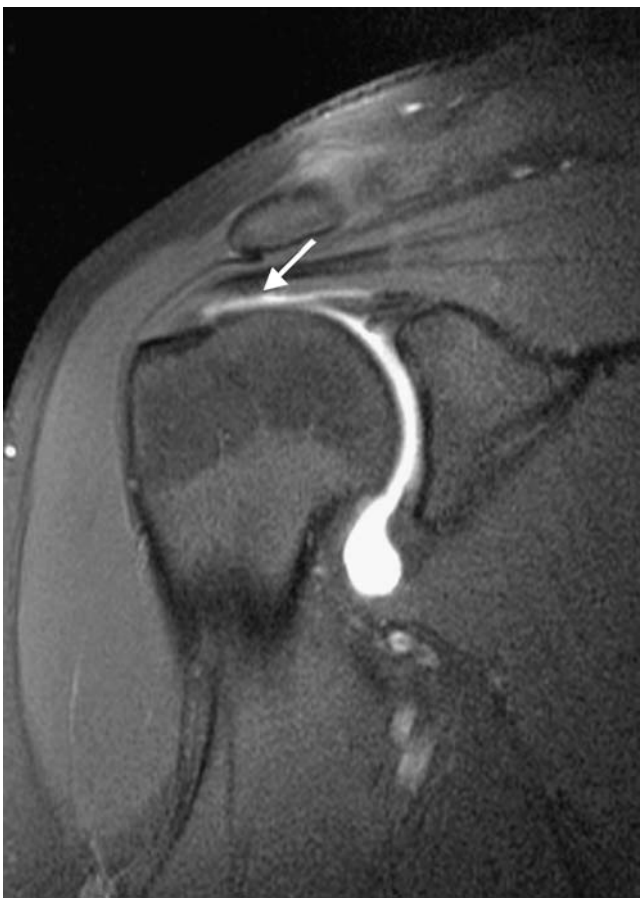

Fig. 6 Supraspinatus tendon articular surface tear. Coronal T1-weighted fat-suppressed image of shoulder MR arthrogram shows subtle irregularity at the undersurface of the supraspinatus tendon (*arrow*)

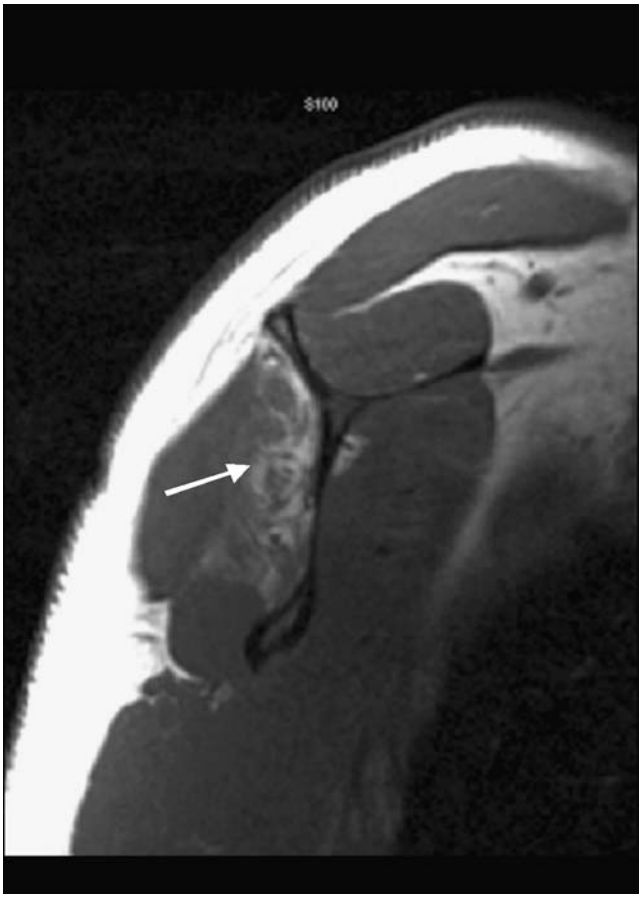

Fig. 7 Infraspinatus atrophy. Sagittal oblique T1-weighted sequence shows hyperintense signal (*arrow*) in the infraspinatus muscle, representing muscular atrophy and fatty infiltration

SLAP II lesion [4]. SLAP tears with significant posterior extension can be disabling for pitchers, because of posterior superior laxity [27]. The appearance of SLAP tears on MRI includes intermediate hyperintense labral degeneration and linear fluid or gadolinium undercutting the superior labrum extending posterior to the biceps origin [28].

A Bennett lesion is a bony excrescence at the posterior capsular insertion of the posterior inferior glenoid [29, 30] (Fig. 8). Bennett lesions have been documented in approximately one-quarter of professional pitchers [31]. The exostosis is likely due to traction of the posterior band of the inferior glenohumeral ligament that occurs during the deceleration phase of throwing. Further growth of the lesion is likely due to posterior humeral impingement in the late cocking phase [30]. Bennett lesions are frequently associated with tears of the posterior labrum, teres minor and infraspinatus tendons in symptomatic shoulders [30]. Large Bennett lesions may be a source of posterior shoulder pain. Lesions greater then 100 mm^2 contribute to posterior impingement and are usually debrided [29].

In an article discussing the close association between internal impingement and GIRD, Tuite et al. found that overhead throwing athletes tended to have increased labral length and a shallow posterior capsular joint recess near the attachment of the posterior band of the inferior glenohumeral ligament [32]. Specifically, they found a statistically significant difference between the mean thickened capsule–labral length in GIRD/internal impingement in athletes (2.9 mm) versus controls (2.1 mm). They also confirmed thickening of the postero-inferior capsule and suggested that these are important radiological signs in the diagnosis of internal impingement/GIRD [32].

Primary instability

Repetitive microtrauma

There is significant shear force across the anterior shoulder in the late cocking and early acceleration phases of throwing. Eventually, there is an increased load at the anterior capsule and subsequent load failure. This causes anterior glenohumeral joint laxity [2].

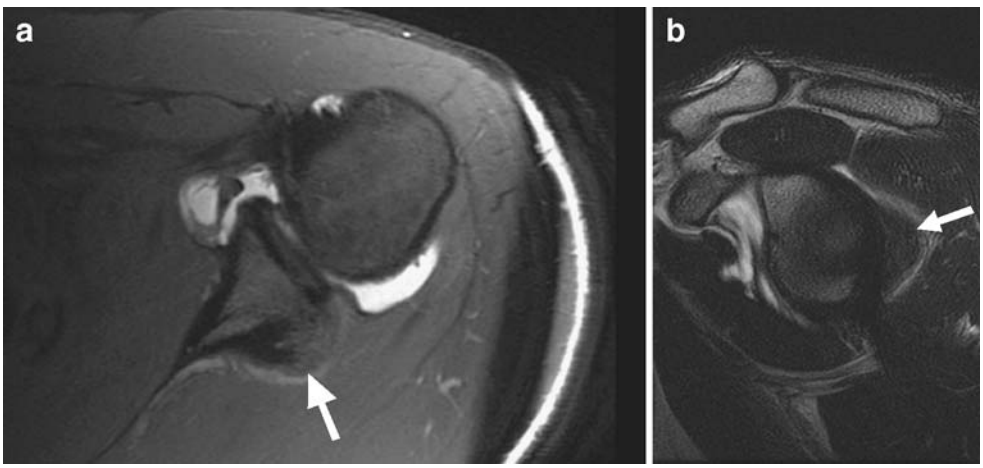

Fig. 8 **a**, **b**. Bennett lesion. MR arthrographic images of the shoulder. Axial T1-weighted fat-saturated (**a**) and sagittal T2-weighted (**b**) images show hypointense area at posterior inferior aspect of glenoid, representing mineralization (*arrow*)

Anterior shoulder joint laxity allows increased anterior translation during throwing. This, in turn, results in increased likelihood of anterior labral lesions and SLAP tears. Capsular laxity must be addressed during arthroscopic repair of other intra-articular abnormalities in athletes with primary instability in order that optimal return to competitive throwing be achieved. Repair of intra-articular abnormality will inevitably fail if the instability is not corrected [33].

Posterosuperior glenohumeral impingement

In baseball pitchers there is repetitive impaction of the posterosuperior glenoid and posterior humeral greater tuberosity during extreme abduction and external rotation. Although posterosuperior impingement is sometimes seen in the stable shoulder, it often occurs in athletes with anterior capsular laxity and glenohumeral instability [4, 15]. Posterosuperior impingement may result in rotator cuff tear and changes at the posterior humeral head, as described in the rotator cuff section. Associated tears of the posterosuperior labrum may be subtle but can be confirmed on MR arthrogram, with the patient in the ABER position [9, 34].

Generalized ligamentous laxity

Generalized ligamentous laxity is a subtype of primary instability. Athletes with such instability have bilateral, symmetric, increased, shoulder laxity. They often also have hyperextension at other joints, such as the knees and elbows. The labrum may be hypoplastic. Baseball pitchers with generalized ligamentous laxity are capable of the highest velocity throws but are at a significantly increased risk of microtrauma and long-term injury secondary to throwing [4].

Acute traumatic instability

Acute traumatic instability is uncommon in the baseball pitcher. An acute traumatic event, as seen in contact sports, typically leads to a unidirectional pattern of instability of the glenohumeral joint [35].

Conclusion

The biomechanics of the overhead throwing cycle is crucial to our understanding of the pathophysiology and imaging appearance of injury in baseball pitchers. The shoulder is commonly injured in overhead throwing activities. As a result of initial adaptive changes to repetitive forceful throwing, alterations in shoulder anatomy, which include shifting of the glenohumeral rotation center posterosuperiorly and increased ligamentous laxity, can eventually lead to tears of the rotator cuff and labrum. Classification of these injuries into impingement and tensile overload syndromes (for rotator cuff injuries) and instability syndromes (for labral injuries) enables a structured examination to be made for specific injury patterns. Knowledge of injury pathophysiology is crucial if we are to understand treatment rationale.

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