Imaging in Anterior Glenohumeral Instability[1]

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In the shoulder, the advantages of range of motion are traded for the disadvantages of vulnerability to injury and the development of instability. Glenohumeral instability encompasses a broad spectrum of clinical complaints and presentations. The diagnosis can be obvious or entirely unsuspected. Imaging findings depend on numerous factors and range from gross osseous defects to equivocal labral abnormalities and undetectable capsular lesions. This review focuses on the imaging findings in three distinct clinical scenarios: acute first-time shoulder dislocation, chronic instability with repeated dislocation, and chronic instability without repeated dislocation. The biomechanics of dislocation and the pathophysiology of labral-ligamentous injury are discussed. The authors distinguish the findings that occur in the acutely traumatized shoulder from those that typify the chronic unstable joint. The roles of different imaging modalities are also distinguished, including magnetic resonance arthrography and the value of specialized imaging positions. The goal of imaging depends on the clinical scenario. Image interpretation and reporting may need to emphasize diagnosis and the identification of lesions that are associated with instability or the characterization of lesions for treatment planning.

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Learning Objectives:

After reading the article and taking the test, the reader will be able to:

- Describe the biomechanics of dislocation and the pathophysiology of labral-ligamentous injury
- Describe the roles of different imaging modalities in the diagnosis of anterior glenohumeral instability
- Evaluate the MR imaging findings that can help radiologists distinguish acute first-time shoulder dislocation from chronic instability with or without repeated dislocation

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The glenohumeral joint has the greatest range of motion of any major articulation in the human body. The trade-off for this mobility is vulnerability to injury and the development of shoulder instability. Dynamic stabilizers of the joint, including the cuff muscles, are insufficient to maintain normal glenohumeral location and function (1). Joint stability also depends on the passive constraints provided by intact static structures, especially the glenoid rim, glenoid labrum, and glenohumeral ligaments. The vast majority of unstable shoulders demonstrate characteristic abnormalities of the labral-ligamentous complex on magnetic resonance (MR) images (2).

Essentials

- Trauma accounts for more than 90% of first-time anterior shoulder dislocations.
- Reciprocating humeral and glenoid lesions are pathognomonic for articular malalignment (dislocation) at the time of trauma and are strongly associated with injuries to stabilizing soft-tissue structures.
- There are two major causes at either end of a continuum; at one end of the spectrum, a major traumatic event causes first-time dislocation followed by repeated dislocations requiring lesser degrees of force and provocation.
- At the other end of the spectrum, there is no antecedent trauma; glenohumeral hypermobility, which may be developmental, leads to multidirectional instability that worsens over months and years.
- In the latter scenario, instability causes symptoms due to recurrent glenohumeral subluxation, not dislocation; because the degree of subluxation may be minimal, this condition has also been described clinically as relative instability.

Glenohumeral instability can become the immediate sequelae of traumatic injury or evolve gradually owing to cumulative stresses related to occupational or sport-specific activities (2). Depending on the clinical scenario and the needs of the treating physician, imaging has different roles. In the acute setting following first-time shoulder dislocation, MR imaging is used to characterize the location and extent of structural damage and typically demonstrates an obvious, pathognomonic pattern of osseous edema, hemarthrosis, and labrocapsular injury. But as dramatic as these MR findings can be, they may not predict repeated dislocation or the future development of glenohumeral instability (3). In the setting of repeated dislocations, when the clinical diagnosis of instability becomes unambiguous, MR imaging may be able to show osseous and soft-tissue abnormalities that can guide surgical planning and the choice of stabilization procedure.

Shoulder instability also occurs in individuals who deny previous dislocations and major traumatic events (2). In one subset of these patients, functional disability and positive provocative testing lead to confident clinical conclusions. In another subset, equivocal history and physical examination lead to challenges in diagnosis (2). If imaging is requested, the history may state a question about the presence of rotator cuff tear or biceps lesion, possibly misleading the radiologist interpreting the imaging study (2).

In this review, we focus on imaging findings in three distinct clinical scenarios. The first scenario involves acute first-time shoulder dislocation. The second applies to chronic instability with repeated dislocation. In the third scenario, there is chronic instability without repeated dislocation. We distinguish imaging findings that characterize the acute setting from those that characterize the chronic unstable joint. We also distinguish the roles of different imaging modalities, including conventional MR imaging versus MR arthrography, and the value of specialized imaging positions such as abduction-external rotation (ABER).

Biomechanics

Dynamic and passive restraints together provide glenohumeral stability, maintaining nearly perfect rotation of the humeral head over the center of the glenoid fossa. Dynamic stabilizers include the rotator cuff and long head of biceps as well as pectoralis major, latissimus dorsi, and periscapular muscles (1). Passive stabilizers include the glenoid rim and concave glenoid fossa, labrum, and capsuloligamentous structures (1). The congruent articular surfaces provide stability through the principle of concavity compression, which is particularly important during the midrange of glenohumeral movement when the capsular ligaments are lax (4). The surrounding muscles create this dynamic joint compression and primary stabilization during the midrange (5). At the extremes of motion, the glenohumeral ligaments contribute most to stability, especially the inferior labral-ligamentous complex (5).

The glenohumeral ligaments (superior, middle, and inferior) are formed by discrete capsular bands that attach to the labrum and adjacent glenoid rim (5–9). The superior and middle glenohumeral ligaments arise anterosuperiorly, just anterior to the biceps tendon. The superior glenohumeral ligament merges with the coracohumeral ligament in the rotator interval and passes between the supraspinatus and the subscapularis to the lesser tuberosity at the bicipital groove. Together with the coracohumeral ligament, the superior glenohumeral ligament functions as a primary passive restraint to inferior

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Abbreviations:
ABER = abduction-external rotation
ALPSA = anterior labral-ligamentous periosteal sleeve avulsion
GLAD = glenolabral articular disruption
HAGL = humeral avulsion of the glenohumeral ligament
IGL = inferior glenohumeral ligament
3D = three-dimensional

Conflicts of interest are listed at the end of this article.

Figure 1

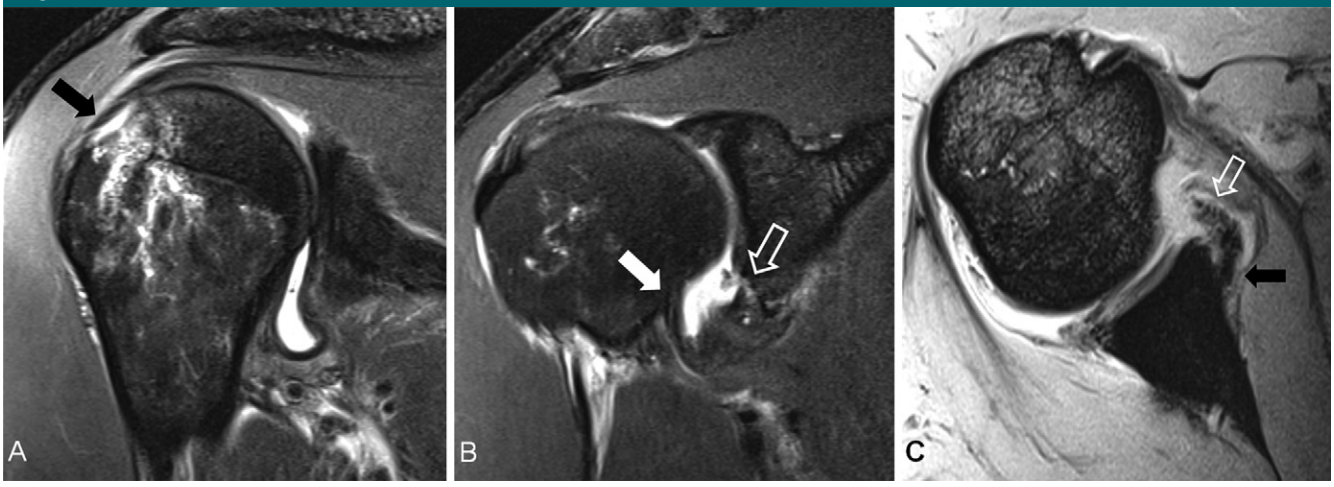

Figure 1: MR images in a 31-year-old male dancer obtained 4 days after acute first-time anterior shoulder dislocation. *A*, Oblique coronal fat-suppressed T2-weighted image shows a small Hill-Sachs fracture (arrow) with prominent bone marrow edema. Effusion is present. *B*, On more anterior oblique coronal fat-suppressed T2-weighted image, the inferior labral-ligamentous complex (open arrow) is detached and displaced from the inferior glenoid rim. The humeral attachment site (solid arrow) is unremarkable. The cuff is intact. *C*, On axial gradient-echo image, the anteroinferior labral-ligamentous complex (open arrow) is displaced from the glenoid rim, and the attached periosteum (solid arrow) is stripped medially along the glenoid neck. No glenoid rim fracture is present.

translation of the adducted shoulder (10). The middle glenohumeral ligament courses inferiorly toward the lesser tuberosity and blends into the capsular sheath of the subscapularis tendon. The middle glenohumeral ligament shows the greatest developmental variation, but differences in morphology do not seem to affect glenohumeral stability (11). The middle glenohumeral ligament limits anterior translation when the shoulder is externally rotated and moderately abducted (10). Both the superior and middle glenohumeral ligaments are commonly depicted on conventional and arthrographic MR images.

The inferior glenohumeral ligament (IGL) is composed of an anterior band, a posterior band, and the intervening axillary pouch. Compared with the superior and middle glenohumeral ligaments, the anterior band of the IGL is critical to passive joint stabilization (12). It typically courses from the anteroinferior labrum, where it forms a sleeve of continuous tissue with the glenoid rim, capsule, and periosteum, to the humeral metaphysis. The IGL becomes taut in the ABER position, resisting anteroinferior glenohumeral translation (10). When the IGL is attached to a torn labrum or is stripped medially along the glenoid neck with its periosteal sleeve, the IGL becomes incompetent and the shoulder becomes unstable (10,12). The IGL is best visualized on arthrographic MR images, with or without ABER positioning, and on conventional MR images when an effusion is present (13,14). Because the IGL is a constant anatomic structure, its labral attachment site can be presumed with confidence even though the lack of effusion prevents visualization. A labral tear located at this presumed attachment site is closely associated with glenohumeral instability (15).

Imaging Modalities and Techniques

In most circumstances, radiographs are first obtained following acute shoulder dislocation. Prior to reduction, radiographs demonstrate the direction of humeral translation. The usual trauma series can include anteroposterior internal rotation, anteroposterior external rotation, scapular "Y" and axillary views, assuming the patient can tolerate positioning. Additional Stryker notch and West Point views help to confirm the presence of Hill-Sachs lesion or Bankart fracture, respectively (2). Adduction-external rotation has been advocated for postreduction immobilization as a way to displace joint fluid posteriorly and approximate the detached labrum to the anterior glenoid rim and promote healing (16,17). Postreduction images are used to characterize residual alignment abnormalities and fractures. If reduction is not possible, cross-sectional imaging may be necessary in the acute setting to guide additional manipulation or surgery on the basis of the presence of osseous (engaging Hill-Sachs defect, obstructing fracture fragment from the glenoid or humeral head) or soft-tissue (intraarticular biceps dislocation blocking realignment) lesions (18).

Arthrographic MR imaging has little or no role in the acute setting when conventional MR images demonstrate periarticular edema or hemorrhage, depict the location and severity of bone marrow injuries, and elucidate the traumatic mechanism. Effusion, or hemarthrosis, provides an arthrographic effect in 97% of cases (19) by distending the joint capsule and outlining intraarticular structures, including the labrum, the glenohumeral ligaments, articular cartilage, and intraarticular loose bodies (Fig 1). MR imaging has the added benefit of

Figure 2

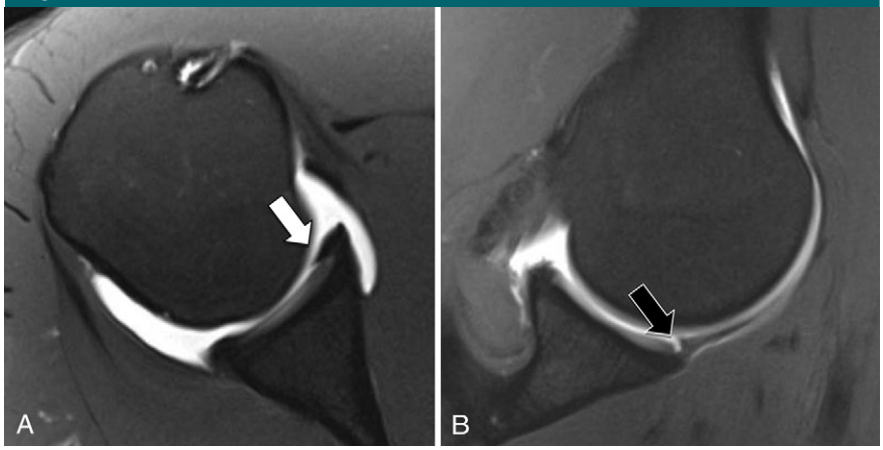

Figure 2: Acute first-time anterior shoulder dislocation: T1-weighted MR images in a 26-year-old man with shoulder pain after a single anterior dislocation. MR arthrography was performed 2 months after known dislocation owing to persistent pain and limitation. *A*, Axial fat-suppressed image demonstrates an intact anterior labrum (arrow). *B*, ABER image demonstrates an avulsion of the anterior labrum with an intact glenoid periosteum and capsule (arrow), highlighting the mechanical advantage of having the shoulder in this position during imaging.

demonstrating abnormalities of the rotator cuff and biceps tendon.

Once the acute situation has passed, MR arthrography becomes an option, assuming that fluoroscopy or sonography is available to guide needle placement for the intraarticular injection of contrast material. Some referring physicians are satisfied with conventional MR, whereas others prefer arthrographic MR and the diagnostic confidence provided by intraarticular contrast material. MR arthrography has the most compelling role in the assessment of younger individuals with suspected instability, when subtle labral-ligamentous abnormalities have profound influences on shoulder function, management, and prognosis (20).

Provocative positioning maneuvers, including imaging in both internal and external rotation, ABER, flexion-adduction and internal rotation (FADIR), and adduction-internal rotation (ADIR), have been proposed to improve visualization of the labral-ligamentous complex (14,21). Although ABER positioning was initially described as increasing sensitivity in the diagnosis of articular-sided rotator cuff lesions (22), it is now recognized as improving the detection of nondisplaced anteroinferior labral tears at the attachment site of the IGL (20,21) (Fig 2). In a series of 256 patients, ABER images improved the diagnostic performance of MR arthrography from 48% to 89% sensitivity (21). Used alone to evaluate the anteroinferior labral-ligamentous complex, ABER images demonstrated the same accuracy as the full complement of arthrographic MR sequences (23). FADIR may improve assessment of the posterior labrum and capsule (24), whereas ADIR may have advantages to ABER in discriminating between different Bankart subtypes (25).

In the setting of repeated dislocations and known instability, some referring physicians prefer computed tomography (CT) or CT arthrography for characterizing the sizes and locations of osseous defects and for guiding preoperative planning. CT reformations and three-dimensional (3D) reconstructions lend themselves to quantitative analysis (26–29). Although CT has a proved track record in the segmentation of cortical bone, 3D reconstruction, and quantification of osseous defects, MR imaging techniques can be modified to provide similar information. In patients with glenoid rim deficiency, CT and MR have been shown to generate comparable quantitative measurements (22). Three-dimensional MR reconstructions of the glenoid and humerus require nonstandard sequences to depict the osseous injuries of the glenoid and humeral head and can be incorporated into the conventional MR imaging protocol with minimal added imaging and postprocessing time (30).

Clinical Scenarios

Acute First-Time Shoulder Dislocation

The prevalence of anterior shoulder dislocation is reported to be as high as 2% in the general population (31–33). Although the location and severity of structural damage directly influence the likelihood of repeat dislocation, patient age at presentation is another important factor determining recurrence rate (34,35). The rate of recurrent dislocation is inversely related to age. Rowe (35) found that only 16% of patients older than 40 years at the time of first dislocation will have dislocation again, compared with 83% of patients younger than 20 years. In young athletes who return to sports activities and competition, the likelihood of repeat dislocation further increases to 92% (34). Even in athletes, the recurrence rate drops to 50%–75% between 20 and 25 years of age (36). Repeated dislocation has been reported to occur three times more frequently in young men compared with women (36). Noncompetitive activity level may also play a role, as shoulder dislocation has been reported more commonly in military personnel compared with the general population (37). Whereas nonsurgical management is the usual treatment in older patients, stabilization surgery may be necessary to avoid recurrent dislocation and instability in younger athletes unwilling to modify their activities or follow rehabilitation programs (34,37).

Trauma accounts for more than 90% of first-time anterior shoulder dislocations (31–36). A common traumatic mechanism is fall on an outstretched hand, or FOOSH. In contact sports, dislocation also results from forced ABER or a direct posterior blow to the shoulder (38). In individuals with developmental glenohumeral laxity, inherent

instability predisposes the shoulder to traumatic dislocation with lesser degrees of force (35). Owens et al (39) distinguished dislocation from subluxation depending on whether the shoulder required manual reduction maneuver at the time of injury. In their population of military cadets, nearly all of the subluxated shoulders, despite spontaneous reduction, demonstrated both Bankart and Hill-Sachs lesions on the basis of MR and arthroscopic images. The authors concluded that initial, traumatic subluxation produced the same pathologic lesions as dislocation and warranted the same cautionary prognosis and management. Therefore, patients with traumatic transient subluxation should be considered for early surgical stabilization to enable the anatomic repair of a detached labrum and reduce the risks of worsened intraarticular damage, chronic instability, and recurrent subluxation or dislocation (39).

Osseous injury.—At the time of dislocation or subluxation, as the humeral head slides over the glenoid rim and relocates in the glenoid cavity, shear forces and osteochondral compression can produce reciprocating osseous lesions of the posterosuperior humeral head and anteroinferior glenoid (1) (Fig 3). Compared with the hard, wedge-shaped cortex of the anterior glenoid rim, the flat contour and softer trabecular bone of the humeral head make it susceptible to Hill-Sachs fracture. Following first-time anterior dislocation, 25%–81% of shoulders show Hill-Sachs defects at arthroscopy (27,40,41).

On MR images of the shoulder, the appearance of an impaction lesion depends on the time interval between imaging and traumatic injury. In the acute and subacute settings, conventional MR imaging demonstrates bone marrow edema on both T1- and T2-weighted images, although fat-suppressed T2-weighted images are more sensitive for regions of subtle marrow edema (42). Proton-density images without fat suppression cannot reliably demonstrate marrow edema (42). In the absence of Hill-Sachs defect, the location and pattern of marrow injury can help to support the diagnosis of glenohumeral dislocation. As the osseous contusion heals over weeks or months, the edema dissipates and, along with it, valuable biomechanical information (42). Therefore, marrow contusion only provides biomechanical information if MR imaging is performed relatively soon after traumatic injury.

Whereas Hill-Sachs defect is the more common imaging finding following dislocation, glenoid rim fracture has the greater prognostic significance. The risk for recurrent dislocation and chronic instability increases with the size of the glenoid bone defect (43–45). In the acute setting, displaced glenoid fragments may prohibit relocation, requiring surgical reduction and internal fixation (46,47). A nondisplaced fracture can heal with conservative treatment if repeated injury is avoided through activity modification. Unfortunately, nondisplaced fractures can be difficult to identify on MR images. Oblique sagittal images, when prescribed in the plane of the glenoid fossa, best demonstrate the glenoid contour and the presence of an anteroinferior fracture line. In the acute setting, when bone marrow edema is present, the fracture line is better characterized on T2-weighted compared with T1-weighted MR images (42). CT reformations in the oblique sagittal plane show nondisplaced fracture lines and small cortically based fragments. Three-dimensional CT reconstructions depict the location of a displaced fragment and the size of its donor site.

Less common osseous injuries involve the site of tendon attachments. The coracoid process is infrequently fractured, typically as a result of direct impaction by the dislocated humeral head (3%–13%) (48). Tuberosity fractures occur in 15%–35% of dislocated shoulders, especially in older patients who are also susceptible to rotator cuff tears (49). Greater tuberosity fractures in the setting of anterior shoulder dislocation may involve the attachment sites of supraspinatus and infraspinatus tendons (50–52). Displaced fractures of the greater tuberosity may require surgical intervention if the cuff tendon becomes interposed between the fragment and its donor site on the humeral head (18,53–55).

Figure 3

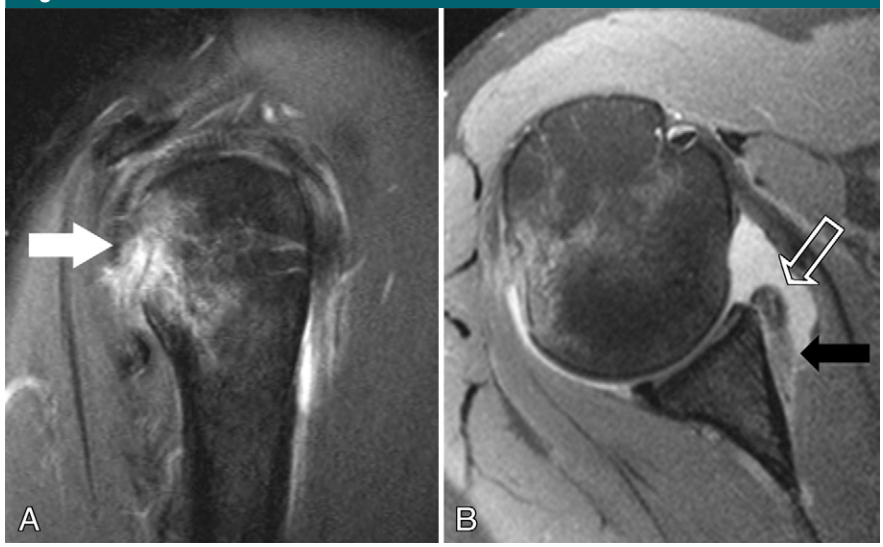

Figure 3: MR images of acute ALPSA in a 24-year-old man who sustained a first episode of anterior shoulder dislocation 3 weeks prior to imaging. *A*, Fat-suppressed T2-weighted and, *B*, axial fat-suppressed intermediate-weighted images demonstrate a Hill-Sachs lesion (white arrow) with marrow edema and associated anteroinferior labral tear with anteromedially displaced fragment (open arrow). Note stripping and edema of the scapular periosteum with developing granulation tissue (black arrow).

Figure 4

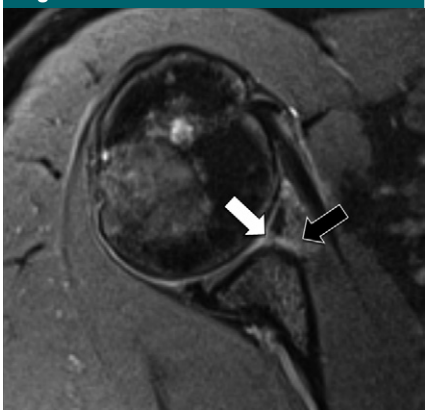

Figure 4: Axial fat-suppressed intermediate-weighted MR image of subacute first-time dislocation in a 32-year-old man with a prior anterior shoulder dislocation 6 weeks prior to imaging. There is a minimally displaced, detached tear of the anterior labrum (white arrow) with tearing of the adjacent capsule (black arrow), consistent with a Bankart lesion, and flattening of the anterior glenoid margin, consistent with a mildly impacted fracture.

Although lesser tuberosity fractures are exceedingly uncommon in anterior shoulder dislocation, the subscapularis is more susceptible to myotendinous strain or contusion compared with supraspinatus and infraspinatus (56,57). Subscapularis injuries may have prognostic implications in older patients owing to its role in static and dynamic stabilization of the glenohumeral joint (58). In high-grade subscapularis tear, chronic instability is more likely to develop and a Bankart repair is less likely to succeed.

Soft-tissue injury.—During the days and weeks following shoulder dislocation, MR imaging findings typically include effusion and periarticular edema or hemorrhage. When hemarthrosis is present, the joint fluid is increased in signal intensity compared with muscle on T1-weighted images (19). Intraarticular fluid creates an arthrographic effect, outlining structures, filling defects, and improving diagnostic confidence. Localized extraarticular edema or hemorrhage can also provide diagnostic advantages by calling attention to a region of capsular or myotendinous injury.

The inferior labral-ligamentous complex, the primary passive stabilizer of the joint, can demonstrate an abnormality anywhere along its course from the glenoid rim to the humerus. After first-time dislocation, four injuries of the inferior labral-ligamentous complex have characteristic appearances on MR images. These abnormalities include the Bankart lesion, the Perthes lesion, the glenolabral articular disruption (GLAD) lesion, and the humeral avulsion of the glenohumeral ligament (HAGL) lesion (20).

At the time of glenohumeral dislocation or subluxation, traction on the IGL is transmitted to the labrum, which can be torn partially or detached completely from the glenoid rim. Avulsion of the labrum, which requires disruption of its periosteal sleeve, is called a Bankart lesion, the classic pathoanatomic hallmark of anterior instability that Bankart described as the "essential" lesion in recurrent shoulder dislocation (Fig 4) (15). This injury occurs at the anteroinferior IGL attachment site and represents the most common labral injury following first-time traumatic dislocation (59). In the acute setting when effusion is present, conventional MR images show fluid separating the labrum from the glenoid rim.

The Perthes lesion, first described in 1905, is similar to the Bankart lesion in that the labrum is separated from underlying articular cartilage but differs in that the labrum remains partially attached to the glenoid rim by an intact periosteal sleeve (60,61). Because the periosteum is weak, the IGL loses function and the shoulder becomes unstable. Whereas the Bankart lesion is most closely associated with traumatic dislocation, the Perthes lesion can result from either dislocation or cumulative stresses on the IGL owing to physical overuse and repetitive microtrauma (62,63). The Perthes lesion is more difficult to diagnose on MR images since the torn labrum can be normal or nearly normal in anatomic position. Therefore, it can be overlooked not only on MR images but also at surgery unless the arthroscopist probes the labrum carefully (64). While the Perthes lesion may be subtle or not apparent on axial images, it may become more apparent on ABER images owing to the traction provided by the inferior glenohumeral ligament on the labrum (65).

During dislocation, shear forces and osseous impaction can damage the articular cartilage over the anteroinferior glenoid fossa. The GLAD lesion combines a labral tear with an adjacent cartilage defect (66). When the labral tear is minimal, patients complain of pain from the osteochondral defect rather than instability (67). Whereas conventional and arthrographic MR images both show focal cartilage loss and full-thickness defects, neither imaging examination may be successful in demonstrating small, delaminated chondral flaps (68). Effusion and intraarticular contrast material administration improve the likelihood of visualizing chondral flaps (68).

When failure occurs at the humeral attachment site, it is called a HAGL lesion (66,69,70) (Figs 5, 6). Bigliani et al (71), in a cadaver study investigating the tensile properties of the inferior labral-ligamentous complex and its sites of mechanical weakness, reported that the labrum failed 40% of the time, the IGL substance failed 35% of the time, and the humeral insertion failed 25% of the time. In the clinical literature, the incidence of HAGL is substantially lower than the 25% predicted by the biomechanical research of Bigliani et al (71). The discrepancy reflects, in part, the differences between cadavers in the laboratory versus athletes on the playing field but also is demonstrative of the difficulty in diagnosing HAGL lesions. At arthroscopy or open surgery, the HAGL lesion can be missed unless a targeted search is triggered by the knowledge gained from preoperative imaging (12,63,69,70,72,73).

In the acute setting, IGL injury is associated with periarticular edema and hemorrhage that localize to the axillary pouch, quadrilateral space, and proximal humerus at the base of the lesser tuberosity on MR images (74). Although the degree of IGL injury can be overestimated if hemorrhage is extensive (75), it should trigger a directed

Figure 5

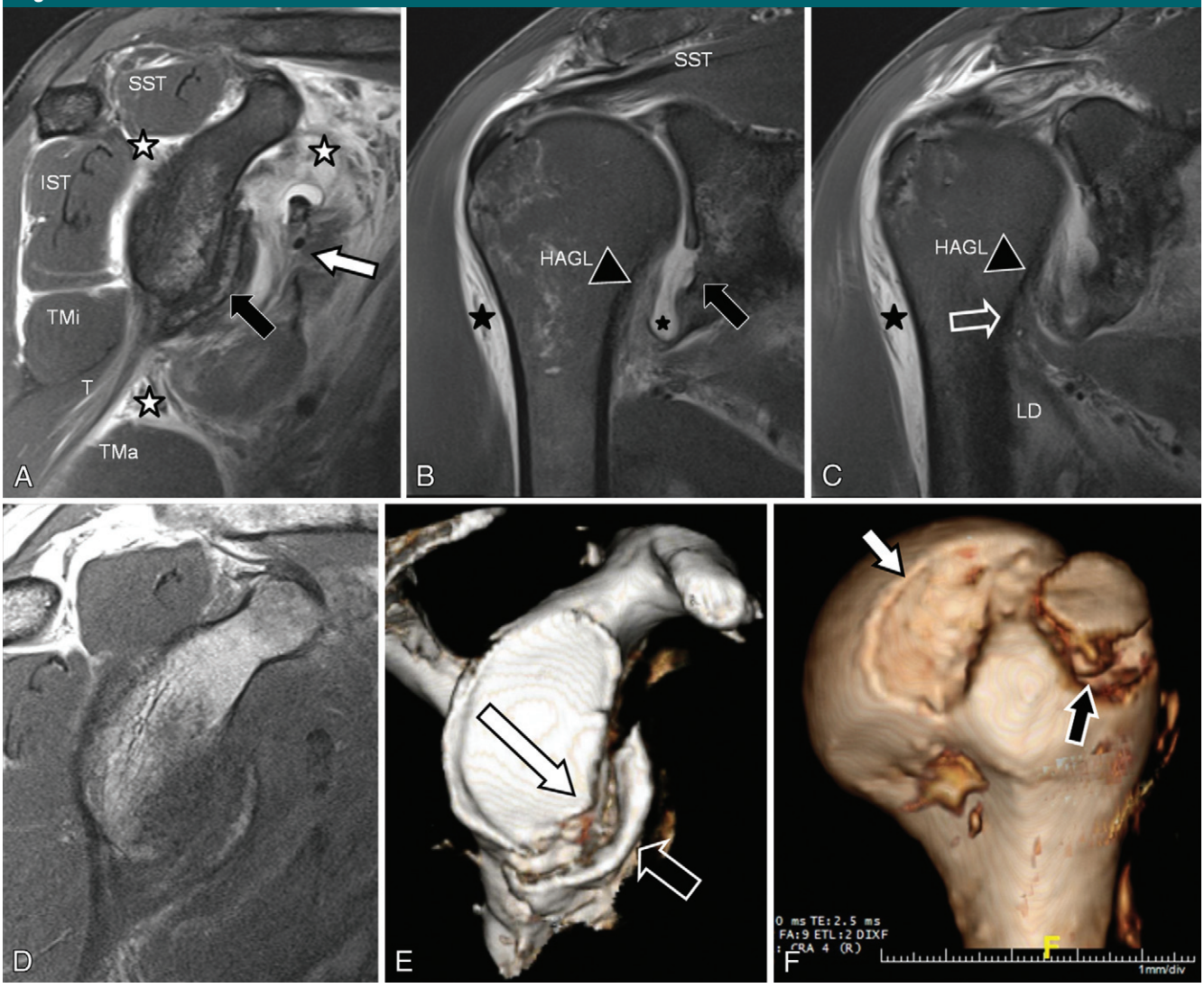

Figure 5: MR images of acute recurrent anterior shoulder dislocation with Bankart fracture in a 64-year-old man. *A*, Sagittal and, *B*, *C*, coronal fat-suppressed T2-weighted images demonstrate extensive periarticular posttraumatic swelling (☆) with high-grade partial thickness tears of the subscapularis myotendinous junction (white arrow) and humeral muscular insertion (open arrow) as well as strains of the supraspinatus *(SST)*, infraspinatus *(IST)*, teres minor *(Tmi)*, latissimus dorsi *(LD)*, triceps *(T)*, and teres major *(Tma)* muscles. Glenohumeral and subacromial subdeltoid effusions (★), as well as humeral avulsion of the inferior glenohumeral ligament (HAGL ▲) and a detached and mildly displaced Bankart fracture (black arrow). *D*, Sagittal T1-weighted image demonstrates a displaced Bankart fracture fragment. The defect in the anteroinferior glenoid rim is somewhat ill defined. *E*, Three-dimensional MR reconstruction demonstrates the amount of glenoid bone loss, glenoid fracture margin (white arrow), and adjacent fracture fragment (open arrow) to a better extent than the conventional MR images. *F*, Three-dimensional MR reconstruction of the humeral head demonstrates a mildly displaced avulsion fracture of the greater tuberosity at the insertion of the supraspinatus tendon (black arrow), as well as an impacted Hill-Sachs lesion (white arrow).

search for additional findings that support a more specific diagnosis of HAGL lesion. The "J" sign (in a right shoulder; reversed "J" in a left shoulder) is fairly specific for IGL rupture and occurs on oblique coronal images when edema and hemorrhage outline the torn, retracted stump of the anterior band of the IGL (76,77). Following primary traumatic shoulder dislocation, IGL abnormalities were identified in 52% of shoulders at baseline MR imaging performed within 7 days of trauma compared with 12% of the same shoulders at arthrographic MR imaging performed 21–54 days later (74). Findings specific for HAGL lesions were identified in 21% of these shoulders at baseline but only 7.1% at follow-up. These results reinforce the difficulty in diagnosing HAGL lesions on MR images, especially months

Figure 6

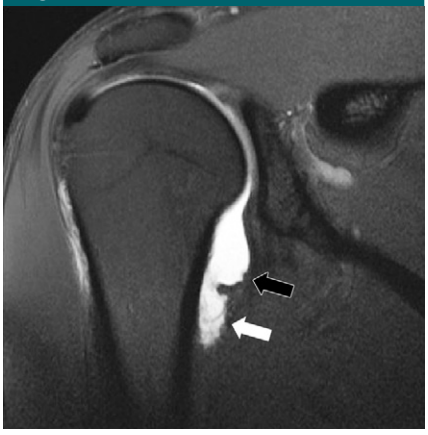

Figure 6: Chronic multidirectional instability in a 41-year-old woman with apprehension, disability, and provocative testing positive for instability and without previous dislocation or major traumatic event. At conventional MR imaging of the shoulder 5 weeks earlier at an outside institution, findings were interpreted as negative for labral-ligamentous injury. On this oblique coronal fat-suppressed T1-weighted arthrographic MR image, contrast material outlines the anterior band of the inferior glenohumeral ligament (black arrow), which is ruptured and retracted from its humeral attachment site. Contrast material leaks from the joint into the quadrilateral space (white arrow). Capsulorrhaphy was performed arthroscopically.

after injury when the damaged tissue has healed and remodeled but the IGL remains incompetent (74). The prevalence of bony HAGL lesion (avulsion of humeral cortex along with the IGL) ranges from 0% to 20% in the literature (74,77).

Chronic Instability with Repeated Dislocation

Traumatic shoulder dislocation can lead to chronic instability and repeated dislocations requiring successively lesser degrees of force and provocation (78). This topic will be addressed in the following paragraphs. Chronic instability and repeated dislocations may also develop without antecedent trauma (3). This disorder, which is beyond the scope of our review, results from excessive laxity and stretching of the glenohumeral joint capsule. As hypermobility worsens over time, the shoulder develops multidirectional instability. In contrast to the osseous lesions and labral-ligamentous tears that characterize trauma-related instability, lax shoulders may show minimal MR imaging findings (79). Despite repeated dislocations, the labrum can be normal in appearance or blunted and mildly deficient (59,63). On the basis of MR findings alone, therefore, the evidence for instability may be minimal or equivocal.

Glenohumeral instability is the obvious clinical diagnosis when recurrent dislocations follow initial traumatic dislocation. In this setting, the role of imaging shifts from lesion detection to lesion characterization for treatment planning. MR or CT imaging is typically requested to guide the surgical approach and the choice of stabilization procedure. Depending on the presence and severity of osseous versus soft tissue lesions, the surgeon may decide on open surgical versus arthroscopic visualization (2). The primary goal of the stabilization procedure may be the reconstruction of humeral and glenoid osseous defects or the repair of anteroinferior capsulolabral soft tissues (2).

Many of the same osseous and labral-ligamentous lesions are seen in first-time and recurrent dislocators (80,81). In arthroscopic studies comparing these groups, hemarthrosis, HAGL lesion, and other capsular injuries were more common in acute primary dislocation, whereas Bankart lesion, anterior labral-ligamentous periosteal sleeve avulsion (ALPSA) lesion, capsular laxity, Hill-Sachs defect, glenoid rim deficiency, and greater tuberosity fracture were more common in chronic repeated dislocation (80,81). Glenoid rim fracture and rotator cuff tear occurred in both groups but were more frequent in repeat dislocators (80,81).

Osseous injury.—The prevalence of Hills-Sachs defects increases from 25% in first-time dislocators to 40%–90% in repeat dislocators (81,82). The defects enlarge in size with increasing numbers of dislocations, eventually taking the signature hatchet morphology (83). The location and orientation of the impaction fracture depend on the position of the humeral head during dislocation and the magnitude of compressive force (83).

Hill-Sachs defects rarely require surgical treatment unless they are large enough to cause mechanical symptoms or engage the glenoid rim (83–89). Larger lesions of the humeral head may be associated with catching, clicking, or popping (90). The engaging Hill-Sachs defect extends into the articulating surface of the humeral head. In the abducted and externally rotated position, the glenoid rim drops into this defect causing apprehension and subluxation in mild cases or locking and dislocation in severe cases (91). When conservative treatment fails, surgical options include remplissage, bone grafting, osteoarticular allograft reconstruction, resurfacing arthroplasty, and hemiarthroplasty (92). Functional assessment based on clinical criteria, not imaging criteria, differentiates the engaging lesion from the nonengaging lesion (91). MR and CT imaging aid in preoperative planning by showing the location and size of the Hill-Sachs defect, as well as the percentage of involved articular surface (90,93).

The glenoid rim can become flattened and deficient due to fracture, bony remodeling, or a combination of both (94) (Fig 5). If a fracture fragment resorbs over time, its donor site is exposed on the glenoid rim. Smaller glenoid defects cause instability due to incompetence of the inferior labral-ligamentous complex. With increasing loss of bone stock, articular incongruency creates a mechanical mismatch that predisposes to recurrent dislocation and severe functional disability (83).

Image interpretation should take into account the size of the glenoid lesion and the contour of the glenoid rim. Recurrent dislocation increases in likelihood when the anteroposterior width of the glenoid defect measures 21% or more of the total glenoid length, or the glenoid articular surface area decreases by 20%–30% (44,45). Once the glenoid defect reaches approximately 25% of glenoid width, the glenoid changes in appearance from a pear shape to an inverted pear shape (44). The amount of glenoid bone loss guides management.

When the loss of bone stock is mild (less than 15% of glenoid width and/or 10% of glenoid surface area), open or arthroscopic soft-tissue repairs are usually sufficient (43,95). When moderate (15%–30% of glenoid width and/or 10%–25% of glenoid surface area), bone augmentation may be appropriate in patients who are physically active (47). When severe (more than 30% of glenoid width and/or 25% of glenoid surface area), surgical treatments prevail, including glenoid reconstruction with bone graft from the iliac crest or transfer of the coracoid process (Bristow and Latarjet procedures) (47,71,72,95) (Fig 7). The Latarjet procedure, in particular, can be done either as an open procedure or arthroscopically (96,97). Classically, it has been used as a secondary method of repair for patients who have failed initial surgical stabilization treatment, but it is becoming increasing more popular as a first-line treatment option for the patient with recurrent shoulder instability (96,97).

Conventional CT enables two-dimensional reformations and the segmentation of cortical bone for 3D reconstructions. Reformations in the plane of the glenoid fossa allow standard quantification techniques, such as measurement of defect width and calculation of glenoid surface area (95,98,99). CT arthrography improves soft-tissue evaluation, but iodinated contrast material may limit quantitative osseous assessment because the injected contrast solution can have the same attenuation as cortical bone and fracture fragments. Although CT has been in use for a longer period of time for the quantification of glenoid deficiency, MR imaging techniques can be modified to provide similar information (29,30). The MR protocol is more time-intensive due to specialized imaging sequences and postprocessing, but after further validation MR should be able to duplicate the results of 3D CT

Figure 7

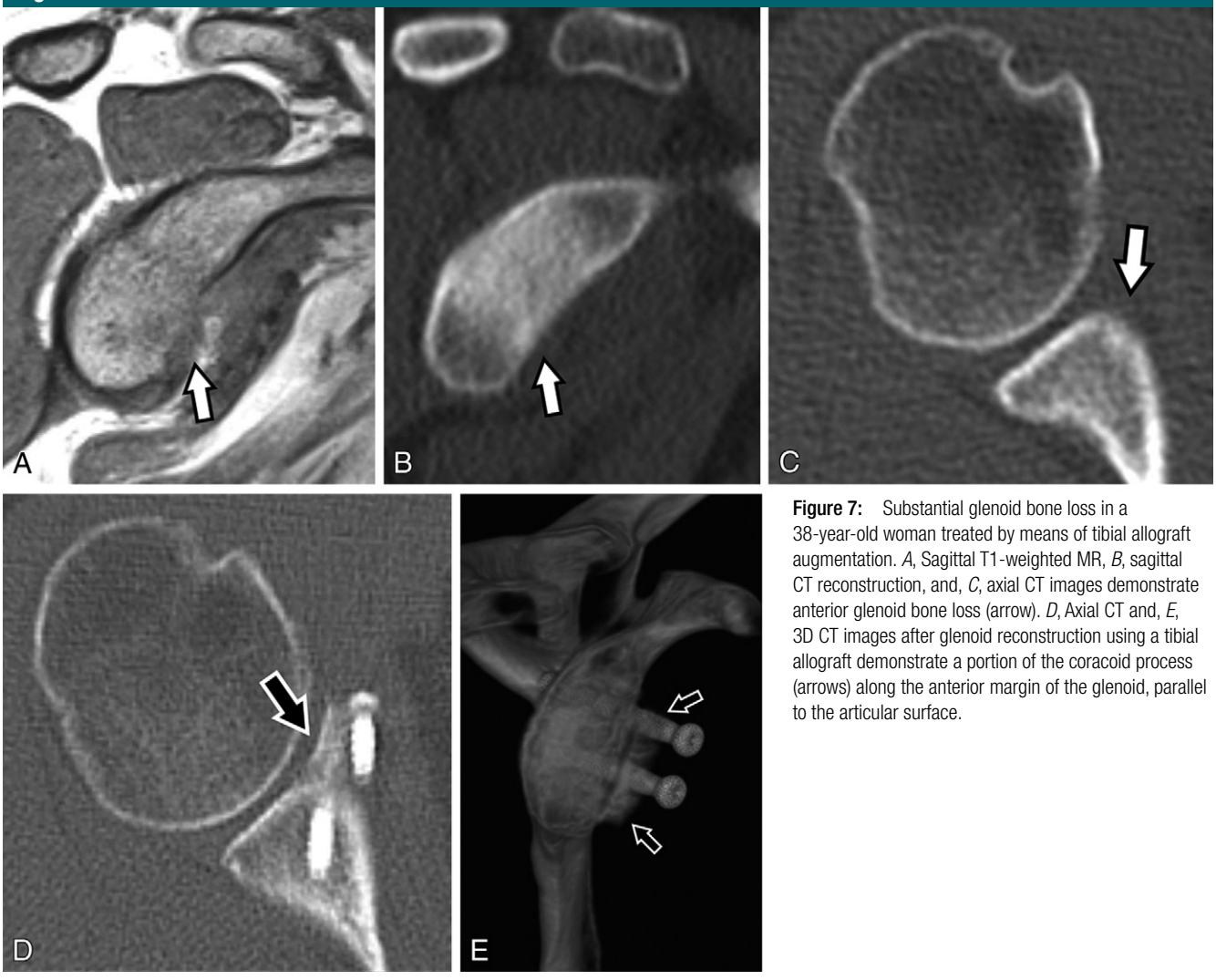

Figure 7: Substantial glenoid bone loss in a 38-year-old woman treated by means of tibial allograft augmentation. *A*, Sagittal T1-weighted MR, *B*, sagittal CT reconstruction, and, *C*, axial CT images demonstrate anterior glenoid bone loss (arrow). *D*, Axial CT and, *E*, 3D CT images after glenoid reconstruction using a tibial allograft demonstrate a portion of the coracoid process (arrows) along the anterior margin of the glenoid, parallel to the articular surface.

Figure 8

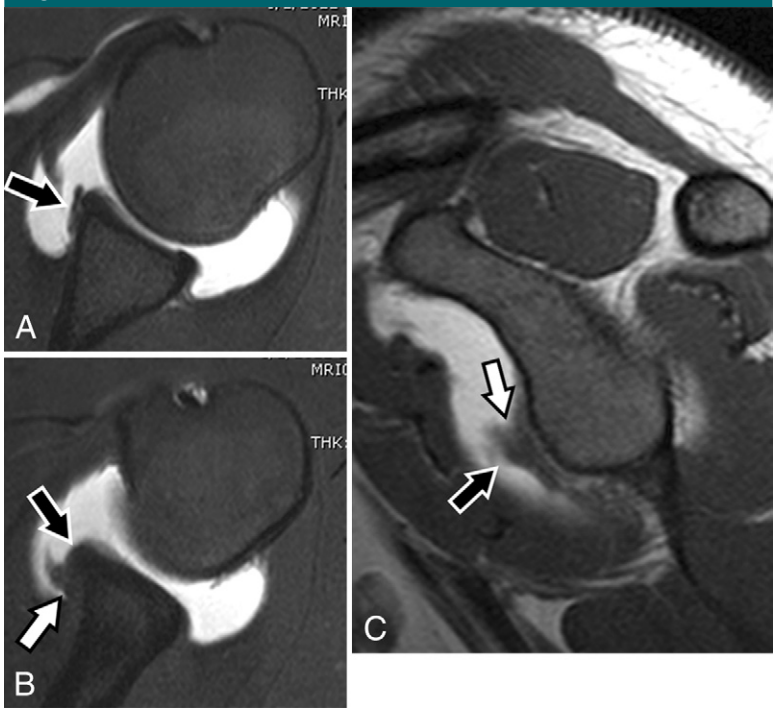

Figure 8: Chronic ALPSA in a young baseball player with clinical findings of anteroinferior instability and no history of prior dislocation. *A*, *B*, Axial and, *C*, sagittal fat-suppressed T1-weighted MR arthrograms demonstrate a medially displaced anteroinferior labral tear (black arrows) and stripped off scapular periosteum with scar tissue annealing the fragment and periosteum to the inferomedial scapular neck (white arrows). (Image courtesy of Jason Mayo, MD.)

and obviate the exposure to ionizing radiation (29,30).

Soft-tissue injury.—During the months and years following acute first-time dislocation, labral-ligamentous lesions evolve depending on numerous factors, such as the location of initial tissue damage, the activity level of the individual, and the numbers of interval dislocations (80,81). Some lesions, such as the Bankart lesion, can be present following first-time dislocation or repeated dislocations. Over time, nondisplaced Bankart lesions become displaced from the glenoid rim (72). As the degree of displacement increases, the anteroinferior glenoid rim appears bare, or deficient, of labrum (Fig 8). GLAD lesions also evolve. With repeated dislocations, chondral flaps increase in size and detach from the glenoid fossa, leading to focal cartilage loss and intraarticular loose bodies (100) (Fig 9). With progressive cartilage loss, degenerative changes such as subchondral sclerosis and bony remodeling develop at the anteroinferior glenoid fossa (100). When acute dislocation is superimposed on chronic instability, hemarthrosis and bone marrow contusion may coexist with the sequelae of repeated dislocation.

The ALPSA lesion is common in chronic instability and can arise from the Perthes lesion (65,101). If the periosteum remains attached to the labrum, traction is transmitted along the IGL, stripping the periosteum medially along the glenoid neck. With repeated dislocation or subluxation, the labral-ligamentous complex retracts medially with the periosteum, rolling up like a shirtsleeve. It can scar down to the glenoid neck and become immobile (101). Although the healing process creates a smooth, synovialized surface that can obscure the ALPSA lesion at arthroscopy, both conventional and arthrographic MR images show a characteristic pattern of findings (65,66) (Fig 8). On axial and oblique coronal images, the anteroinferior glenoid rim is deficient, or bare. This absence of labrum is a clue that the labral-ligamentous complex may be displaced and should lead to a targeted search for focal soft-tissue thickening along the glenoid neck 5–15 mm medial to the glenoid rim (65,66). Because the diagnosis of ALPSA lesion is most certain when the IGL can be identified and followed to the thickened periosteal sleeve, arthrographic MR, with or without ABER positioning, has diagnostic advantages over conventional MR (65,66).

HAGL lesions, and capsular injuries in general, are more likely to be identified when MR imaging immediately follows the traumatic event (74). Because of the healing process and the remodeling of capsular tissue in the axillary pouch, the IGL can scar down to the humerus and appear normal in contour at arthroscopy despite repeated dislocations (69). If the IGL does not scar down to the humerus, mechanical insufficiency can cause chronic instability (70). The capsular defect at the humeral attachment site enables the development of a pseudo-pouch adjacent to the normal axillary pouch (74). This pseudo-pouch may be difficult to identify on conventional MR images unless it becomes distended by effusion (74). At MR or CT arthrography, contrast material fills the pseudo-pouch and flows distally along the humerus into the quadrilateral space near the axillary nerve and inferior circumflex neurovascular bundle, giving the appearance of two axillary pouches (Fig 6). In the months and years following trauma, this pseudo-pouch suggests the diagnosis of HAGL lesion (74). CT may better demonstrate the bony HAGL, which occurs when IGL avulsion is associated with cortical fracture or periosteal detachment.

Chronic Instability without Repeated Dislocation

This scenario poses the greatest diagnostic challenges. In acute first-time dislocation and chronic instability with

repeated dislocation, the clinical history, signs and symptoms, and imaging findings are usually straightforward. In chronic instability without repeated dislocation, disability results from glenohumeral subluxation or micro-motion, not dislocation (62,102–105). Because the degree of subluxation may be minimal, this condition has also been described as occult recurrent subluxation, relative instability, functional instability, or microinstability (62,102,103,106,107). The clinical presentation can be confusing due to nonspecific symptoms and equivocal signs at physical examination. Imaging findings may also be subtle or equivocal (63). When imaging is requested, referring physicians may question the presence of rotator cuff tear, impingement, or superior labral tear rather than implicate instability (103). Because the clinical indications can be misleading, radiologists should consider the prospect of glenohumeral instability during the interpretation of most shoulder MR imaging studies performed in young adults.

Three major etiologic subsets may be associated with chronic instability in the absence of repeated dislocation: remote trauma (including previous first-time dislocation), overuse activity, and generalized capsular laxity (62,63,78,102,103,107). Remote trauma may be forgotten by patients or dismissed as unimportant if immediate, urgent treatment was unnecessary (103). Subluxation or even dislocation may have occurred without recognition because the shoulder reduced spontaneously (103). Expecting full recovery, patients may decide against medical evaluation until persistent symptoms and disability necessitates treatment. Therefore, MR imaging can be delayed for weeks or months following the traumatic event. Unrecognized dislocation puts the shoulder at risk for the same labral-ligamentous injuries as recognized first-time dislocation (102,103). The delay in imaging enables the resolution of bone marrow edema and effusion, the healing and remodeling of capsular defects, and the scarring of nondisplaced labral fragments to the glenoid rim (65,80,81).

Figure 9

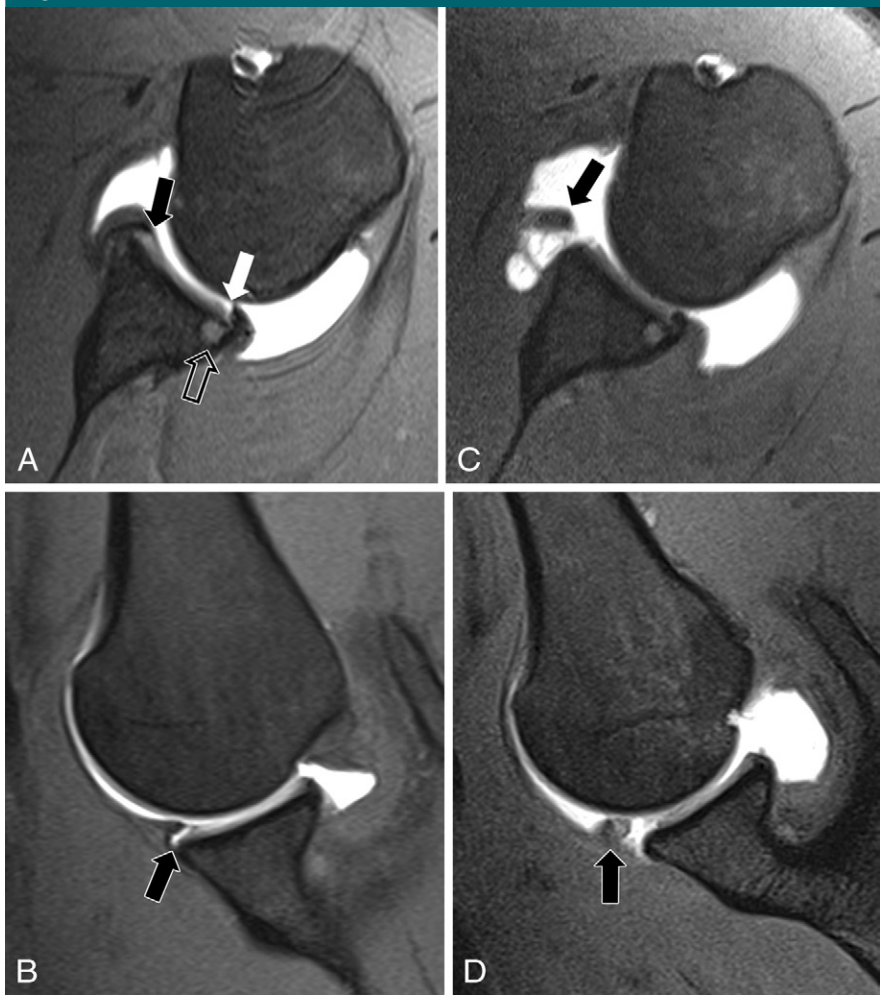

Figure 9: MR images in a 24-year-old extreme athlete who denied previous dislocation or major shoulder trauma. A, Axial, fat-suppressed T1-weighted MR arthrogram shows increased signal intensity at the labral chondral junction (black arrow), as well as a nondisplaced posteroinferior labral tear and a contrast agent–filled chondral defect with a GLAD lesion (white arrow). A small subchondral cyst is found adjacent to the posterior tear (open arrow). *B*, On ABER fat-suppressed T1-weighted MR arthrogram, tear of the anteroinferior labral chondral is made conspicuous (arrow). At the time of initial MR imaging, the patient elected to continue competition, forgoing surgical intervention. Repeat MR imaging 2.5 years later was performed due to disability preventing further competition. *C*, Axial fat-suppressed T1-weighted MR arthrogram shows a Bankart lesion and marked displacement of the anteroinferior labral-ligamentous complex (arrow) from the glenoid rim. *D*, ABER fat-suppressed T1-weighted MR arthrogram demonstrates progression to a GLAD injury in the anteroinferior labrum with detachment and displacement of a large labral chondral fragment (arrow). Arthroscopic stabilization procedure was performed. The posteroinferior labral chondral GLAD lesion appears to have healed in the interval.

Labral-ligamentous injuries, including Bankart lesion, Perthes lesion, and HAGL lesion, can be overlooked in the absence of Hill-Sachs defect or other osseous evidence for previous dislocation (20,21,64,74). Compared with conventional MR imaging, MR arthrography improves diagnostic accuracy because contrast material can fill the labral defects in nondisplaced lesions and outline abnormal capsular contours (64). ABER positioning may further increase diagnostic confidence by transmitting tension from the IGL to the labrum,

thereby displacing an occult lesion from the glenoid rim or revealing an intact periosteal sleeve (101) (Fig 2).

Overuse represents another important etiologic category in the development of occult recurrent glenohumeral subluxation. Although athletes and physicians are less likely to consider the diagnosis of instability in the absence of contact trauma, pitchers, swimmers, tennis players, and weight lifters all may present with functional instability manifested only as pain (62,103–108). Repetitive noncontact activities create cumulative stresses on both the passive and dynamic structures that stabilize the shoulder (103–106,108). These stabilizing structures gradually fail, leading to the development of chronic instability. Structural failure may result from stretching of the inferior glenohumeral ligament and joint capsule, tearing of the anteroinferior glenoid labrum, and/or delamination of the articular cartilage over the anteroinferior glenoid fossa (62,102–104,106,107). Whereas some overuse lesions may be subtle and easily overlooked on MR images, others may be obvious and lead to the overestimation of their physiologic significance (104–106,108). In the symptomatic overhead athlete, for example, stretching of the joint capsule may not be detectable on MR images, but causes an acquired laxity, or microinstability, that can lead to other disorders such as cuff tears, labral tears, and biceps lesions (105,106). In the asymptomatic overhead athlete, anteroinferior labral tear may be present on MR images despite the lack of compromise on competitive performance (109,110). In pitchers, anteroinferior labral tear may actually represent an adaptive change that improves performance by allowing a greater degree of ABER during the late cocking and early acceleration phases of throwing (104,105,108).

The third etiologic subset includes patients with the atraumatic onset of multidirectional, bilateral laxity or instability (AMBRI) for which rehabilitation has a primary role in treatment rather than surgery (59,111,112). In AMBRI, developmental capsular laxity and glenohumeral hypermobility are exacerbated by numerous factors, including repetitive overstretching, proprioceptive imbalance, and connective tissue deficiency (24,63). Patients typically complain of functional limitations due to involuntary glenohumeral instability (111). There is no history of instigating trauma. At physical examination, the shoulder is unstable in more than one direction (multidirectional), which helps to differentiate AMBRI from traumatic unidirectional instability (63,73,111). The opposite shoulder may also feel loose. Surgery, which typically involves capsulorrhaphy or inferior capsular shift, is considered if conservative treatment and rehabilitation fail (111). At arthroscopy, the most common finding is capsular redundancy in the axillary pouch (113). Therefore, the imaging diagnosis of AMBRI is challenging because labral tears and osseous defects are typically absent (111,112). MR arthrography, and in some instances CT arthrography, may suggest the presence of capsular redundancy, but reliable criteria are lacking (114).

Conclusion

Glenohumeral instability encompasses a broad spectrum of clinical complaints and presentations. The diagnosis can be obvious to the referring physician, but often it is unsuspected. Most unstable shoulders have never dislocated (3). Radiologists should also keep in mind that the history of one-time dislocation is not the equivalent of shoulder instability (3). Hill-Sachs fracture is an imaging marker of dislocation but should not be interpreted as a sign of instability without other supporting abnormalities (110). Imaging findings depend on the clinical scenario: acute first-time shoulder dislocation, chronic instability with repeated dislocation, or chronic instability without repeated dislocation. Imaging abnormalities that occur in the acutely traumatized shoulder are substantially different from those that typify the chronic unstable joint. The goal of imaging depends on the clinical scenario. Image interpretation and reporting may need to emphasize the identification of lesions for diagnosis, or the characterization of lesions for treatment planning. Therefore, the decision to use CT, CT arthrography, MR, or MR arthrography also depends on the clinical scenario and goal of imaging.

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