Glenohumeral Ligament Pathology: A Practical Imaging Approach

Introduction: Small ligaments with a major role in shoulder stability

The glenohumeral ligaments are important static stabilizers of the shoulder, but their imaging assessment remains challenging. They are thin, anatomically variable, position-dependent structures that are intimately related to the joint capsule and labrum. Furthermore, ligament abnormalities rarely occur in isolation: they are usually part of a broader capsulolabral injury pattern associated with glenohumeral instability.

The superior (SGHL), middle (MGHL) and inferior glenohumeral ligaments (IGHL) should therefore not simply be regarded as three structures to be checked individually. Their function depends on arm position, and each of them becomes important in different ranges of motion.

For the radiologist, the relevant questions are: **Which ligament is abnormal? Where is the lesion—glenoid, midsubstance or humeral attachment? Is the abnormality traumatic, degenerative or related to capsular disease? And which associated labral, osseous or tendinous abnormalities reveal the mechanism of injury?**

Think functionally: Ligament tension changes with arm position

The glenohumeral joint achieves its remarkable mobility at the cost of intrinsic osseous stability. The capsule, labrum and glenohumeral ligaments therefore provide essential passive stabilization.

The **SGHL** originates near the superior glenoid and labrum and courses toward the lesser tuberosity. Together with the coracohumeral ligament, it contributes to the rotator interval and plays an important role in limiting inferior translation and external rotation with the arm adducted.

The **MGHL** is the most anatomically variable of the three. It generally originates from the anterior glenoid or labrum and inserts on the anterior humeral neck. It becomes an important anterior stabilizer in intermediate degrees of abduction.

The **IGHL complex** is the major stabilizing structure when the arm is abducted. It consists of anterior and posterior bands connected by an axillary pouch. The anterior band becomes particularly important in abduction and external rotation—the position in which anterior shoulder dislocation commonly occurs.

Thus, glenohumeral ligament interpretation should always be linked to **mechanism and position**, rather than morphology alone.

MRI or MR arthrography?

Routine MRI can demonstrate major capsuloligamentous abnormalities, particularly in acute post-traumatic patients, when joint effusion provides natural distension. But the thin glenohumeral ligaments can be difficult to evaluate in a nondistended joint.

Direct MR arthrography remains particularly valuable in patients with suspected instability because intra-articular contrast distends the capsule, separates the ligaments from adjacent structures and outlines small tears.

High-resolution axial and coronal oblique images are fundamental. Sagittal oblique images are particularly useful for evaluating the rotator interval and overall capsular anatomy.

Imaging in the **ABER position—abduction and external rotation—** can provide additional information in selected cases. ABER places tension on the anterior band of the IGHL and anterior-inferior labrum and can improve visualization of subtle abnormalities that may be inconspicuous in the neutral position.

However, MR arthrography should not be interpreted simply by searching for contrast leakage. The morphology, insertion and tension of each ligament are equally important.

1) The IGHL complex: The key ligaments in traumatic instability

Among the glenohumeral ligaments, the IGHL complex is the most clinically important in anterior instability.

During anterior dislocation, the humeral head is displaced anteriorly and inferiorly, placing considerable tension on the anterior-inferior capsulolabral structures. Injury may occur at several sites.

The classic **Bankart lesion** represents detachment of the anterior-inferior labrum from the glenoid, usually with associated injury to the adjacent capsuloperiosteal structures. However, the same traumatic force can **disrupt the IGHL** itself.

The radiologist should therefore follow the IGHL from its glenoid origin through the axillary pouch to its humeral insertion rather than stopping after identifying a labral lesion.

A useful conceptual approach is:

glenoid attachment → ligament substance → humeral attachment.

Failure can occur at any of these levels.

HAGL: Do not forget the humeral attachment

A humeral avulsion of the glenohumeral ligament (HAGL) is an important and frequently overlooked cause of persistent instability.

In a HAGL lesion, the IGHL is disrupted at its humeral attachment rather than at the glenoid side. The lesion may occur with or without an associated Bankart lesion.

On coronal MR arthrography, the normal axillary pouch has a relatively smooth U-shaped configuration. Disruption of the humeral attachment can produce the classic **J-sign**, with contrast extending inferiorly along the humeral neck.

However, the J-sign should not be used in isolation. Capsular distension and patient positioning can modify the appearance of the axillary pouch.

The most convincing finding is direct discontinuity of the ligament at its humeral insertion, particularly when associated with contrast extravasation and surrounding soft-tissue abnormalities.

HAGL lesions are important because they may be missed if attention is focused exclusively on the glenoid labrum.

GAGL and midsubstance lesions

The IGHL can also fail at its glenoid attachment independently of a classic labral avulsion, sometimes described as a glenoid avulsion of the glenohumeral ligament (GAGL).

Midsubstance capsular or ligamentous tears may also occur.

These lesions are less common and may be difficult to diagnose because the distinction between ligament, capsule and labral tissue is not always obvious.

The radiologist should therefore avoid forcing every instability lesion into a single named category when the anatomy is uncertain. A precise description of the location and extent of capsuloligamentous disruption may be more useful to the surgeon than an uncertain acronym.

2) The MGHL: beware of normal variants

The MGHL is probably the glenohumeral ligament most likely to generate interpretative errors because of its marked anatomical variability.

It may be thin, thick, duplicated or completely absent. A prominent cord-like MGHL may occur as part of a **Buford complex**, in which the anterosuperior labrum is absent and a thickened MGHL is present.

This normal variant must not be mistaken for an anterosuperior labral tear.

The MGHL can itself be injured, particularly during anterior instability, but isolated MGHL lesions are considerably less common than combined capsulolabral injuries.

As always, abnormality should be diagnosed on the basis of convincing discontinuity, irregularity, abnormal contrast extension or associated traumatic findings—not simply unusual morphology.

A useful rule is: **Variation is common; isolated pathology is less common.**

3) The SGHL and rotator interval

The SGHL should be interpreted together with the coracohumeral ligament, long head of the bicipital tendon and rotator interval capsule.

Abnormalities of this region may occur in instability, biceps pulley lesions and adhesive capsulitis.

The SGHL and coracohumeral ligament contribute to stabilization of the long head of the biceps tendon at the entrance to the bicipital groove. Injuries involving the pulley may therefore be associated with medial subluxation of the biceps tendon and tears of the upper subscapularis.

When an abnormal SGHL is suspected, the radiologist should systematically evaluate the **biceps–subscapularis–rotator interval complex** rather than considering the ligament in isolation.

Ligaments in multidirectional instability

Not all of the instability patterns result from an acute traumatic tear.

Patients with multidirectional instability may demonstrate capsular redundancy and increased joint volume rather than a discrete ligament rupture. The axillary recess may appear capacious, and capsular structures may be elongated.

However, imaging findings substantially overlap with normal anatomical variation.

A large capsule on MRI does not independently establish multidirectional instability. The diagnosis remains predominantly clinical.

The radiologist's role is to identify capsular morphology, exclude focal traumatic lesions and report associated structural abnormalities.

This is another situation in which imaging should support, rather than replace clinical assessment.

Adhesive capsulitis: when the capsule becomes the disease

Glenohumeral capsular pathology is not limited to instability.

In adhesive capsulitis, inflammatory and fibrotic changes involve the capsule and rotator interval. MRI may demonstrate thickening and increased signal of the inferior glenohumeral capsule, particularly within the axillary recess, together with abnormalities of the coracohumeral ligament and rotator interval.

The inferior capsule can therefore provide valuable diagnostic information.

This produces an interesting contrast:

In instability, the capsule may be torn or excessively lax; in adhesive capsulitis, it becomes thickened and contracted.

The same anatomical structures can therefore participate in almost opposite biomechanical disorders.

Look for associated lesions: they tell the story of the trauma

Glenohumeral ligament abnormalities should never be interpreted without examining the remainder of the shoulder.

In anterior instability, look for a Hill-Sachs lesion, Bankart or Bankart-variant lesion, glenoid bone loss and rotator cuff injury.

In posterior instability, search for a reverse Hill-Sachs lesion and posterior labral or capsular abnormalities.

Bone lesions are particularly useful because they provide evidence of the direction and severity of a previous dislocation.

The imaging interpretation should therefore reconstruct the mechanism:

Which direction did the humeral head move, which structures were placed under tension, and where did the stabilizing complex fail?

This biomechanical reasoning is often more reliable than searching independently for a collection of named lesions.

A practical reading strategy

A systematic approach (in 6 steps) is useful in patients with suspected instability:

1. determine whether there is evidence of previous anterior or posterior dislocation by examining the humeral head and glenoid.
2. evaluate the labrum circumferentially.

3. follow the IGHL complex from the anterior-inferior glenoid through the axillary pouch to the humeral neck. Do not stop after finding a Bankart lesion.
4. evaluate the MGHL while remembering its numerous normal variants.
5. inspect the SGHL, coracohumeral ligament, biceps pulley and upper subscapularis as a functional unit.
6. integrate ligamentous findings with capsular morphology, bone loss and rotator cuff abnormalities.

Conclusion

Glenohumeral ligament pathology is best understood as part of the broader biomechanics of shoulder stability.

The radiologist should move beyond simply stating that the glenohumeral ligaments are "intact" or "torn." The clinically relevant information concerns **which component is injured, where it has failed, and how the lesion fits with the mechanism of instability**.

Three principles are particularly useful:

.The IGHL should be followed from glenoid to humerus—do not stop at the labrum

.The MGHL is highly variable—do not mistake normal anatomy for disease

.Glenohumeral ligament lesions rarely exist in isolation—always reconstruct the complete instability pattern.

Ultimately, the best interpretation does not merely identify a ligament abnormality. It explains **how the shoulder lost—or altered—its stability**.

Lesions of the 3 GHL ligaments will be illustrated in the oral presentation.

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