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Humeral Avulsion of the Inferior Glenohumeral Ligament in College Female Volleyball Players Caused by Repetitive Microtrauma

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Background: Humeral avulsion of the inferior glenohumeral ligament is a rare injury resulting from hyperabduction and external rotation, and it is most commonly seen with sports-related injuries, including those from volleyball. The anterior band of the inferior glenohumeral ligament is most commonly injured (93%), whereas the posterior band is infrequently injured. The axillary pouch humeral avulsion of the inferior glenohumeral ligament as a result of repetitive microtrauma has not been yet described in the English literature.

Hypothesis: Humeral avulsions of the inferior glenohumeral ligaments are identifiable in volleyball players without acute injuries, and they have a unique pathologic pattern in these athletes.

Study Design: Case series; Level of evidence, 4.

Methods: Four female college volleyball players with pain in their dominant shoulder and with inferior capsular laxity and/or instability—without a known history of trauma or dislocation of the same shoulder—were referred by an experienced sports medicine orthopaedic surgeon for the magnetic resonance arthrogram procedure of the same shoulder. The imaging findings were retrospectively correlated with the initial interpretation and arthroscopic findings.

Results: All 4 patients had an axillary pouch humeral avulsion of the inferior glenohumeral ligament. Three had articular surface partial-thickness rotator cuff tear, and 3 had a labral tear. All were outside hitters or middle blockers who consequently performed multiple hitting maneuvers in practice and games.

Conclusion: Repetitive microtrauma from overhead hitting in volleyball generates forces on the inferior capsule of the shoulder joint that may cause inferior capsular laxity and subsequent failure of the humeral side of the axillary pouch portion of the inferior glenohumeral ligament.

Keywords: humeral avulsion of the inferior glenohumeral ligament lesion; female college volleyball; volleyball-related shoulder injury; axillary pouch humeral avulsion of the inferior glenohumeral ligament

Shoulder pain syndromes are the third-most common injury and the second-most common overuse injury among volleyball athletes, accounting for 8% to 20% of all volleyball injuries.22 These injuries include the more commonly reported rotator cuff tendinitis,16,30 suprascapular nerve entrapment,11 and isolated infraspinatus muscle atrophy,16 all of which can be related to “sick scapula” syndrome.6,7 Volleyball-related shoulder overuse injuries result in an average of 6.5 weeks of lost training and/or competition time.29 With the exception of suprascapular neuropathy, relatively little is known about the epidemiology of shoulder pain among volleyball players.22

In this article, we describe a rare humeral avulsion of the inferior glenohumeral ligament (IGHL) involving the axillary pouch (or APHAGL lesion), which we consider to be the result of volleyball-related kinetics and repetitive microtrauma. We discuss the normal anatomy of the IGHL and the volleyball kinetics that may result in development of an APHAGL lesion. To our knowledge, neither volleyball kinetics (hitting/spiking cycle) nor the APHAGL lesion has been described in the English
We also discuss the transosseous tunnel technique of APHAGL lesion repair, which has also not been formally described.

**INFERIOR GLENOHUMERAL LIGAMENT**

The IGHL is the primary stabilizer of the shoulder with the arm at 90° of abduction and external rotation. It is composed of an anterior band, a posterior band, and an interposed axillary pouch (Figure 1). When viewed en face, the anterior band of the IGHL originates between the 2-o’clock and 4-o’clock positions on the glenoid, whereas the posterior band originates from the 7-o’clock to 9-o’clock positions. The IGHL attaches to the humerus just below the articular margin of the humeral head in 2 distinct configurations: a collarlike attachment or a V-shaped attachment. In normal-shoulder magnetic resonance arthrography (MRA), the axillary pouch and IGHL are seen as a fluid-distended U-shaped structure sitting below the inferior margin of the glenoid and adjacent articular edge of the humeral head.

**Humeral Avulsion of the Inferior Glenohumeral Ligament**

The humeral avulsion of the inferior glenohumeral ligament (HAGL) lesion represents avulsion of the IGHL from its humeral attachment. This rare lesion is a result of hyperabduction and external rotation. It is most commonly seen with sports-related injuries, with a reported incidence of 1% to 9% of shoulders requiring treatment for instability symptoms. With a tear of the IGHL labral complex, the U-shaped axillary pouch is converted to a J shape as the IGHL draws away from the humeral neck, with concurrent extravasation of joint fluid or contrast (in MRA). The majority of HAGL lesions occur in association with an anterior inferior shoulder dislocation; 93% involve the anterior band; and they are frequently associated with concurrent injury to the glenoid labrum or rotator cuff. Eighty percent of HAGL lesions are associated with a concurrent Bankart lesion. The involvement of the posterior band of the IGHL (posterior HAGL lesion) is less common. Stoller stated that identification of the HAGL lesion requires the presence of a joint effusion or the use of MRA.

**Summary of Literature of HAGL Lesion Repair**

There is no single physical examination finding that will assist the surgeon to diagnose a HAGL lesion versus the more commonly found Bankart lesion and capsular laxity. Rather, the lesion is diagnosed on preoperative MRA examinations or diagnostic arthroscopy.

The traditional repair of the HAGL lesion has been accomplished with open surgery. The humeral capsular attachment of the middle and inferior glenohumeral ligaments is divided from the humeral neck to gain literature.
access to the glenoid labrum and articular surfaces. In effect, this creates a large HAGL, which must be repaired to the humeral neck upon closure of the shoulder joint. This is usually accomplished with multiple sutures passed from the ligamentous cut margin to the lesser tuberosity stump of the subscapularis, shifting the capsule superiorly for tensioning. The capsular margin is thus drawn into a shallow bone trough created at the capsule’s original attachment site on the surgical neck of the humerus. Alternatively, one or more suture anchors can be positioned in the humeral neck trough to obtain secure attachment.

The surgical diagnosis of HAGL lesion as a cause for anterior shoulder instability can be difficult because of the open anterior approach to the unstable shoulder. In attempting to define the plane between the subscapularis tendon and the capsule, the inadvertent entry into the shoulder joint may mimic an actual HAGL lesion. Furthermore, leaving the deeper portion of the subscapularis tendon over the capsule may mask a HAGL lesion.

Previous descriptions of arthroscopic repair of the HAGL lesion include that of Wolf et al., in which 4 polydioxanone bioabsorbable sutures from the capsular margin are passed through the subscapularis near the lesser tuberosity attachment and tied over the deltopectoral fascia but beneath the skin of the anterior shoulder. To avoid the neurovascular structures, the capsule margin is shifted anteriorly and superiorly with these sutures while being tensioned to lie in the bone trough on the humeral neck. Although the repair technique is performed arthroscopically, this technique lacks the security of the previously described open suture anchor techniques because the ligaments are not repaired directly to bone.

In 2005, Arciero and Mazzocca described mini-open repair of the HAGL lesion with sparing of the superior 50% of the subscapularis tendon. An L-type incision is made in the lower third of the subscapularis tendon approximately 1 cm medial to the lesser tuberosity. The transverse limb of this incision is made just superior to the anterior circumflex vessels. Beginning inferiorly, the subscapularis tendon is lifted up, exposing the IGHL stump of the subscapularis, shifting the capsule superiorly and anteriorly with these sutures while being tensioned to lie in the bone trough on the humeral neck. The anterior and posterior bands of the IGHL lesion are then repaired with 2 or 3 suture anchors anatomically. The advantages of this technique are preservation of the important superior tendinous portion of the subscapularis tendon, thereby preserving strength, easier rehabilitation, and return to full activity.

In 2004 and 2006, Richards and Burkhart and Huberty and Burkhart, respectively, described suture anchor placement in the humeral neck for an arthroscopic all-inside repair of the HAGL lesion, done with effort to return the capsule to its anatomical site without shifting superiorly. An all-arthroscopic technique using suture anchors is a difficult and demanding technique. Perpendicular access for drilling and insertion of suture anchors would leave the instruments passing directly through the axillary neurovascular structures. Successful arthroscopic technique for repair of the HAGL lesion using a suture anchor technique was reported in 2005 by Spang and Karas.

The transosseous tunnel technique, which we are introducing, accomplishes the same goal of anatomical attachment of the capsular margin to a shallow bone trough in the humeral neck but by use of humeral transosseous tunnels.

**MATERIALS AND METHODS**

This study, which was Health Insurance Portability and Accountability Act compliant and approved by the institutional review board of our institution, waived the requirement for written informed consent for retrospective data analysis. The arthograms, subsequent MRA studies, and subsequent arthroscopic surgery procedures were performed in a clinical setting. Patients gave separate written informed consents for the arthograms and arthroscopic surgery procedures.

**Patients**

Our goal was to evaluate the MRA findings of the dominant shoulder in female college volleyball players who had chronic repetitive activity-related pain and who gave no history of prior trauma or dislocation to the same shoulder. We were specifically interested in evaluating for the presence of HAGL lesions. In a 5-year period (2001-2006), we found, in our hospital database, 5 female college volleyball players who were referred by an experienced sports medicine orthopaedic surgeon for the MRA procedure of their symptomatic dominant shoulders. All 5 had diagnosed APHAGL lesions, 4 of which were arthroscopically repaired. The anterior and posterior bands of the IGHL were intact in all. One of the 5 with a repaired APHAGL lesion was excluded from this review owing to a history of motor vehicle trauma of the same shoulder. None of the remaining 4 patients (average age, 20 years; range, 19-21 years) had a known history of shoulder trauma, subluxation, or dislocation, and none participated in sports activities other than volleyball. The data were retrieved from the clinical and surgical records of our sports orthopaedic surgeon who takes care of the athletic team at our university.

**Clinical Presentation and Examination**

All athletes were Division I volleyball players (all outside hitters) who had posterior shoulder pain or anterior and posterior shoulder pain for at least 6 months (range, 6 months to 4 years) despite a concerted therapy and shoulder-strengthening program. All reported pain produced with overhead hitting and serving and variably recounted a sensation of progressive shoulder weakness with loss of velocity in hitting. On clinical examination, all had findings consistent with dominant shoulder anterior inferior and/or posterior inferior capsular laxity and internal impingement. All had their pain reproduced by 90° to 120° of abduction with maximal external rotation position, which was relieved by relocation maneuver. In this position, all had asymmetric weakness on the internal
All exhibited increased range of external rotation motion in the abducted position (from 120° to 145°) and load and shift testing demonstrated laxity grades of 2 to 3 anterior without apprehension. One athlete demonstrated posterior load and shift grade 2 laxity with apprehension. Weakness in rotator cuff testing positions, atrophy, and scapular dyskinesia were not apparent, and no one reached the diagnostic criteria for glenohumeral internal rotational deficit.

Imaging Procedures

The athletes underwent arthrography and a subsequent MRA of the symptomatic dominant shoulder. For arthrography, all glenohumeral joints were injected in a standard fashion on the fluoroscopy table using the anterior approach, through the region of the anterior rotator cuff interval, after local anesthesia with 1% lidocaine. A 20-gauge spinal needle was used to inject 12 to 14 mL of a mixture of 0.1 mL of gadopentetate dimeglumine (Magnevist, Berlex Imaging, Montville, New Jersey), 10 mL of iohexol (Omnipaque 300, Amersham Health, Oslo, Norway; a non-ionic low-osmolar iodinated contrast agent), and 10 mL of sterile 0.9% sodium chloride (Hospira Inc, Lake Forest, Illinois). After the contrast injection, frontal radiographs were obtained of the injected shoulder in external and internal rotation.

All MRA studies were performed on one of two 1.5-T Signa MRI machines (GE Healthcare) with a dedicated receive-only phase array shoulder coil (MRI Devices, Gainesville, Florida). Our shoulder MRA imaging protocol comprises T1-weighted images with fat suppression (repetition time, 500 milliseconds; echo time, 20 milliseconds) in 3 planes (coronal oblique, axial, and sagittal oblique), coronal oblique T1-weighted images without fat suppression (repetition time, 600 milliseconds; echo time, 20 milliseconds), axial spoiled gradient-echo images with fat suppression (repetition time, 18 milliseconds; echo time, minimum full [3.5-13]; flip angle, 5°), and sagittal oblique T2-weighted images with fat suppression (repetition time, 3800 milliseconds; echo time, 80 milliseconds). Imaging in abduction external rotation is not routinely performed in our institution.

Surgical Technique of APHGL Lesion Repair

All athletes underwent thorough shoulder examination under anesthesia, diagnostic shoulder arthroscopy, and subacromial bursoscopy, which were performed by the same orthopaedic surgeon.

Of the 4 APHAGL lesions, 3 were repaired with a transosseous tunnel anatomical repair technique (Figure 2), tying the avulsed axillary pouch of the IGHL to the anatomical neck of the humerus. Initial arthroscopy accomplished labral repairs and partial-thickness rotator cuff debridement. In addition, the subacromial bursa was entered for visualization of the greater tuberosity before passage of the transosseous drill bits.

The shoulder was then positioned in 30° of abduction to relax the axillary pouch and position the greater tuberosity lateral to the acromial margin.

While the arthroscope was positioned in the anterior superior portal, the avulsed inferior capsule was debrided to create a fresh margin. A motorized bur was inserted through the posterior-inferior and anterior-inferior portals to create a shallow trough of roughened bone for reattachment of the capsule on the humeral neck.

The margin of the axillary pouch was then captured with horizontal mattress suture (or sutures) using a Linvatec spectrum (ConMed Linvatec, Largo, Florida) while under direct viewing. The original monofilament suture was exchanged for a braided nonabsorbable suture, such as No. 2 Ticron. Depending on the size of the humeral avulsion, 1 to 3 horizontal mattress sutures were used.

A modified tibial ACL drill guide (Smith & Nephew per Illuziturri, originally for creation of the anterior portal in hip arthroscopy) was positioned through the posterior inferior portal, with the aiming tip placed in the anterior aspect of the humeral neck trough (Figure 2). The wire guide end was then positioned just adjacent to the lateral acromial border, where a stab incision portal was created to allow the drill guide barrel to be applied to the posterior greater tuberosity and locked into place. A drill-tipped
guide wire was advanced until its tip was seen in the trough while in view with the 70° arthroscope in the anterior superior portal.

The first drill-tipped guide wire was left in place, and a second spot in the humeral neck trough was targeted to allow a second guide wire to be positioned, slightly more posterior on the humeral tuberosity. Further drill holes were created as needed in the same fashion.

Sequentially, the guide wires were removed and replaced with a Smith & Nephew Suture Retriever to enable passage of the axillary pouch sutures up through the transosseous tunnels to the tuberosity (Figure 3C). The sutures then were tensioned and tied over the tuberosity bone bridge (or bridges) under direct observation in the subacromial bursa. The result was a tensioned inferior axillary pouch (Figure 3D) applied to a bleeding bone trough at the site of the native capsular attachment.

Postoperative Management

After the operation, the patient was placed in a gunslinger position sling with a small abduction pillow. During the first 4 postoperative weeks, she was instructed to perform active elbow motion, wrist, and hand exercises. From 4 to 6 weeks postoperatively, she was instructed on passive forward flexion and rotational stretches, with external rotation limited to 30° at the side and abducted external rotation limited to 80° until postoperative week 8. Thereafter, range of motion was progressed to 10° to 15° short of normal side motion. We anticipated that at 4 months postoperatively, a slight restriction to abducted external rotation would remain (10°) and be a permanent but asymptomatic restriction. There was no expected restriction in scapular plane flexion. Progressive resistance training with elastic bands began at 6 weeks postoperatively. During months 3 and 4, the patient was instructed in neuromuscular facilitation and rhythmic stabilization using the Body Blade (Mad Dogg Athletics, Venice, California). At month 4, she began a throwing program and progressed to serving and volleyball hitting at 5 to 6 months.

Images: Findings, Evaluation, and Correlation

The MRA images were retrospectively reevaluated by 2 experienced musculoskeletal radiologists, by consensus, and after that correlated with the initial reading. The images were evaluated for rotator cuff, glenohumeral ligament, bone, and surrounding soft tissue abnormalities, as well as labral, capsular, and acromioclavicular joint abnormalities. The MRA findings were then correlated with the arthroscopic results.

RESULTS

One of the arthroscopically proven APHAGL lesions was not described on the initial MRA reading but was seen on the retrospective evaluation of the images by both readers. There were no other significant discrepancies in the initial and retrospective radiology reading or between the radiology reports and the arthroscopic findings. Table 1 presents the results.

IGHL Abnormalities

All 4 patients had arthroscopically proven tears of the humeral attachment of the IGHL (ie, HAGL lesion) involving the region of the axillary pouch, which we are classifying as an APHAGL lesion (ie, an axillary pouch HAGL lesion). Three of these APHAGL lesions were arthroscopically repaired (Figures 3 and 4). The remaining lesion was small, and the surgeon opted not to repair it. In the initial MRA reports, 3 of 4 APHAGL lesions were described as tears of the inferior joint capsule/IGHL. In the third patient’s MRA, an APHAGL lesion was retrospectively identified (Table 1). All patients had arthroscopically proven intact anterior and posterior bands of the IGHL, although 2 glenoid labral avulsion tears (1 anterior and 1 posterior) were encountered and repaired.

Rotator Cuff Abnormalities

None of the patients had a full-thickness or bursal-side partial-thickness rotator cuff tear. Three had articular-surface partial-thickness (tension) tears, which were debrided. One had a low-grade tear involving the supraspinatus and infraspinatus tendons; one had a moderate-grade tear of the supraspinatus tendon; and the other had a low-grade tear of the supraspinatus tendon (Figure 4B and 4E).

Glenoid Labral Abnormalities

Three of four athletes had labral tears: 1 SLAP (superior labral anterior posterior) type IIC tear (Figure 3A), 1 midline posterior labral avulsion tear (Figure 4C and 4F), and 1 old-appearing anterior inferior labral avulsion tear. All labral lesions were repaired using suture anchors to the glenoid rim.

Treatment Outcome

All 4 patients returned to full volleyball competition after completion of the rehabilitation program and within 6 to 8 months of surgery. Requirements for return to play were equivalent overhead shoulder strength compared with nondominant arm and negative instability and labral signs (internal rotation resistance strength test, apprehension, relocation, and O’Brien test). All athletes thereafter continued to compete in collegiate or professional volleyball for at least 2 additional years (range, 2-5 years) without shoulder symptoms. Patient No. 2, who did not undergo repair of a small APHGL lesion along with the Bankart repair, returned to play at 6.5 months and played 1 further collegiate season, followed by 3 years professionally.
DISCUSSION

Humeral capsular avulsion of the IGHL was first described by Nicola in patients with anterior inferior shoulder dislocation, in the setting of combined hyperabduction and external rotation. In 2007 Bui-Mansfield et al performed an extensive review of literature and found 71 reported cases of HAGL (including their 6) as a result from various sports-related injuries, including volleyball in a single patient, with the majority of lesions related to rugby (42%), located anteriorly, and found in male patients. In their retrospective study of 17 patients, Chung et al

Figure 3. A 20-year-old female college volleyball player with arthroscopy-confirmed right shoulder axillary pouch humeral avulsion of the inferior glenohumeral ligament (APHAGL) lesion and SLAP (superior labral anterior posterior) type IIC tear, both of which were repaired. A, T1-weighted fat-suppressed coronal magnetic resonance arthrography image shows extravasation of the glenohumeral joint contrast through the axillary pouch of the inferior glenohumeral ligament consistent with APHAGL lesion (arrow) and a SLAP type II superior labral tear (solid head arrow). B-D, Posterior inferior arthroscopic views show (B) a large APHAGL lesion (arrows), with (C) transosseous sutures tying this lesion to the anatomical neck of the humerus (arrows) before and (D) after tensioning of the repaired APHAGL lesion (arrows). HH, humeral head.
described humeral avulsion of the posterior band of the IGHL without history of acute trauma in 7 athletically active patients with multidirectional shoulder instability, of which 1 was a competitive high school volleyball player.

The HAGL lesion may occur in isolation, but it more commonly occurs in association with other abnormalities, including rotator cuff tear, Bankart lesion, Hill-Sachs deformity, or labral tear. Failure to treat a concurrent HAGL lesion may result in subsequent postoperative recurrence of the symptoms. All 4 of our patients had concurrent rotator cuff or labral tears, which were debrided or repaired.

Humeral avulsions of the IGHL are uncommon in female patients and are infrequently reported as volleyball-related injuries. To our knowledge, the APHAGL lesion has not been reported in the English literature. We believe that the APHAGL lesion in our patients occurred as a result of repetitive microtrauma related to volleyball spiking—related kinetics that can be explained by tension created in the inferior joint capsule with hitting.

In our study, none of the 4 volleyball players had a history of previous trauma or shoulder dislocation. They all suffered from chronic dominant shoulder pain associated with their sports-related activities and repetitive microtrauma. All 4 had arthroscopically proven HAGL lesion, with the humeral avulsion of the IGHL involving the axillary pouch and with intact anterior and posterior bands, which we classified as a seventh-type/variant of a HAGL lesion—namely, the axillary pouch avulsion of the IGHL (APHAGL lesion) (Figure 5). All 4 athletes with APHAGL lesions returned to competitive college volleyball after surgical treatment. The fifth with a repaired APHAGL lesion was excluded from this review owing to a history of motor vehicle trauma of the same shoulder, thereby confounding the traumatic origin of the APHAGL lesion.

One of our patients had a small APHAGL lesion that the operating surgeon considered insignificant and opted not to repair. This patient had a small Bankart lesion repaired (inferior to the anterior band), and the surgeon was concerned about excessive restriction of the IGHL (limiting abduction) if both ends were repaired. Additionally, the APHAGL lesion in this patient was quite small such that the majority of the IGHL remained attached to the humerus. This athlete returned to competitive volleyball 6.5 months after surgery, played 1 further collegiate season, and continued to play for additional 3 years professionally.

The other athletes had larger lesions that were considered to be at least partially responsible for the patients' symptoms. These lesions were repaired; however, it is not possible to establish which of the pathologic entities was the primary source of symptoms.

The elite volleyball athlete performs an estimated 40,000 spikes in a season. There has been extensive research into the kinetics and kinematics of the overhead throwing motion, with most of the published studies focused on baseball, but a few studies have focused on volleyball-related shoulder disorders. There are similarities between the biomechanical aspects of these 2 overhead sports, with majority of the force imparted to the volleyball during the spike as originating from the torso. However, the contact point in the spiking motion is higher than the release point of the pitcher such that the glenohumeral joint is in greater (maximal) abduction/elevation when these forces are imparted. This hitting position explains the more inferior axillary position of the APHAGL lesion that we see in volleyball hitters, as compared with the anterior-band HAGL seen more with throwing athletes.

Kugler et al reported that volleyball attackers demonstrate different muscular and capsular qualities in the playing shoulder as compared with the opposite shoulder. The playing shoulder is depressed, the scapula laterализed, and the dorsal muscles and posterior and inferior part of the shoulder capsule shortened. Similar findings have
been reported to occur in other overhead athletes and described as sick scapula (scapular malposition, inferior medial border prominence, coracoid pain and malposition, pectoralis minor contracture, and scapular dyskinesis).6,7 Sick scapula is associated with shoulder pain attributed to the spectrum of rotator cuff injury and functional instability, and it may contribute to the development of suprascapular neuropathy. Reeser et al22 reported that suprascapular neuropathy occurred in 45% of their elite volleyball players. However, none of our patients had clinical findings consistent with sick scapula syndrome or suprascapular neuropathy.

We believe that the humeral avulsion of the axillary pouch portion of the IGHL in our patients was a result of repetitive microtrauma and may thus be explained by the kinetics of a volleyball spiking cycle, which may be divided into the cocking phase, acceleration phase, contact phase at full elevation, and follow-through phase (Figure 6). Cocking phase positions the arm in abducted external rotation and coils the trunk in torsion. The acceleration phase uncoils the trunk, maximally elevating and externally rotating the shoulder joint and generating high tension in the inferior joint capsule. Contact occurs at maximal shoulder elevation, which focuses

Figure 4. A 21-year-old female college volleyball player with arthroscopically confirmed right shoulder axillary pouch humeral avulsion of the inferior glenohumeral ligament (APHAGL) lesion (repaired), a moderate-grade partial-thickness articular surface distal supraspinatus tendon tear (debrided), and a posterior midline labral avulsion tear (repaired). A, T1-weighted fat-suppressed coronal magnetic resonance arthrography image shows extravasation of the glenohumeral joint contrast through the axillary pouch of the inferior glenohumeral ligament consistent with APHAGL lesion (arrow). B, T1-weighted fat-suppressed coronal magnetic resonance arthrography image shows extension of the contrast into the undersurface of the distal supraspinatus tendon consistent with a moderate-grade partial-thickness tear (arrow). C, axial gradient-echo fat-suppressed magnetic resonance arthrography image shows contrast undercutting the posterior labrum at approximately the 9-o’clock position, consistent with a posterior midline labral avulsion tear (arrow). D, posterior inferior arthroscopic view shows an APHAGL lesion (arrows). E, posterior arthroscopic view shows a partial-thickness supraspinatus tendon tear (arrows). F, anterior superior arthroscopic view shows a midline posterior labral avulsion tear (arrows). HH, humeral head; BT, biceps tendon (long head); G, glenoid.
the tension on the most inferior joint capsule (axillary pouch). Repetitive overuse can cause stretching and injury and thereby result in capsular laxity and subsequent development of the APHAGL lesion. The short follow-through phase moves the tension toward the posterior inferior joint capsule while high eccentric contraction forces are developed in the posterior rotator cuff to decelerate the arm. The association in our HAGL lesion patients of internal impingement sequelae (tension-sided cuff tears and SLAP tear) along with instability lesions (traction-type labral tears) suggest that subclinical instability is present and resultant from this repetitive spiking activity.

Figure 5. A, artist’s schematic of the axillary pouch (AP) humeral avulsion of the inferior glenohumeral ligament (APHAGL) lesion with intact anterior and posterior bands (AB and PB) in the coronal plane. HH, humeral head; G, glenoid. B + C, artist’s drawing of the appearance of the APHAGL lesion as viewed from outside the capsule but beneath the rotator cuff.

Figure 6. The hitting cycle of volleyball spiking may be divided into (A) the cocking phase, with the playing shoulder in abduction and external rotation and the trunk coiled in torsion; (B) the acceleration phase, with the trunk uncoiling, the shoulder reaching maximal external rotation, and the resultant tension translating to the anterior and inferior joint capsule; (C) the contact phase, with the shoulder at maximal elevation and with tension primarily on the inferior joint capsule; and (D) the short follow-through phase, with an abrupt deceleration of the arm, resulting in tension translating to the posterior inferior joint capsule and significant posterior rotator cuff eccentric contraction forces.

The major limitation to our study is the small number of patients with rare APHGL lesions.

One possible criticism is that the APHGL lesions may represent defects of the axillary pouch distant from humeral attachment. Previous radiologic studies have shown false-positive HAGL lesions with defect in the axillary pouch and no arthroscopically proven humeral avulsion of any of the 3 parts of the IGHL. In 2008 Melvin et al. reported 4 false-positive MRA cases of HAGL lesion in the region of the axillary pouch, which on arthroscopy had an appearance of a defect in the IGHL without humeral avulsion of this structure. The authors proposed caution in diagnosing HAGL lesion on MRI and suggested more broad description of the HAGL lesions as defects of the IGHL complex. In 2009 Murphy et al. reported 3 additional false-positive HAGL lesions in the region of the axillary pouch that subsequently healed per follow-up MRA studies. However, in our study, the existence and position of the APHGL lesion was arthroscopically proven in each patient. We also believe that arthrographic overdistension of the glenohumeral joint may result in joint decompression through the regions where the joint capsule is weak, such as the axillary pouch. These iatrogenic defects are of no clinical significance and may heal spontaneously, similar to arthroscopic portals. In our patients, the glenohumeral joints were not overdistended on arthrographic injections, and all APHGL lesions were arthroscopically assessed, with 3 of them considered large enough to require treatment.

An additional limitation to our study is that it is not possible to establish which of the pathologic abnormalities was the primary source of symptoms. However, we believe that
the large capsular defects (APHGL) lesions should be viewed as being pathologic and requiring treatment. Another limitation is that a standardized outcome score was not used in follow-up.

Additional studies with more surgically proven HAGL lesions related to repetitive microtrauma in volleyball players are needed to contribute further understanding of volleyball-related kinetics shoulder joint injuries, including HAGL lesion variants. We welcome further studies that would contribute to better understanding of this lesion and its treatment.

CONCLUSION

Humeral ligamentous avulsions occur in the region of the axillary pouch (between the anterior and posterior bands of the IGHL) in the dominant hitting shoulders of volleyball-playing females. These lesions occur with partial-thickness undersurface rotator cuff tears and labral tears in the volleyball hitter. Clinical presentation is that of internal impingement pain and hyperlaxity without true instability. These lesions are assumed to result from the repetitive microtrauma of overhead hitting, creating cumulative tension injury to this area of the capsule. The biomechanics of the overhead hitting athlete need further study to elucidate this mechanism.

REFERENCES